EJMI

DOI: 10.14744/ejmi.269425
EJMI 2026;10(3):425–432

Research Article

Survival Outcomes and Inflammatory-Nutritional Prognostic Markers in Adult Ewing Sarcoma: A Single-Center Retrospective Cohort

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Abstract

Objectives: Adult ewing sarcoma is uncommon, and real-world data on survival outcomes and accessible prognostic markers remain limited. This study evaluated survival outcomes and exploratory prognostic factors, with emphasis on lactate dehydrogenase (LDH) and inflammatory-nutritional markers, in an adult single-center Ewing sarcoma cohort.

Methods: Adult patients with histologically confirmed Ewing sarcoma who were diagnosed between May 2010 and January 2026 and treated and/or followed at a single medical oncology department were retrospectively reviewed. Follow-up data were updated through May 2026. Overall survival (OS) was calculated from diagnosis to death or last follow-up, and progression-free survival (PFS) was calculated from diagnosis to first progression or death. Survival was estimated using the Kaplan–Meier method. Prognostic associations were assessed using univariable Cox regression and exploratory two-variable Cox models adjusted for metastatic disease.

Results: Twenty-three adult patients were included. The median age was 36.4 years, and 9 patients had metastatic disease at diagnosis. During follow-up, 12 deaths and 13 PFS events occurred. Median OS was 78.3 months, with 1-, 2-, and 5-year OS rates of 86.7%, 67.9%, and 52.4%, respectively. Median PFS was 15.9 months, with 1-, 3-, and 5-year PFS rates of 56.5%, 47.8%, and 47.8%, respectively. Metastatic disease was associated with worse OS (HR 6.14, 95% CI 1.74–21.7; p=0.005). Median PFS was not reached in localized disease and was 6.2 months in metastatic disease (log-rank p<0.001). LDH >259 U/L was associated with worse OS in univariable analysis (HR 6.63, 95% CI 1.77–24.9; p=0.005) and remained associated with worse OS after adjustment for metastatic disease (adjusted HR 6.13, 95% CI 1.55–24.2; p=0.010). Platelet-to-lymphocyte ratio (PLR) >169 and systemic inflammation response index (SIRI) >1.74 were associated with worse OS in univariable analysis, whereas a higher prognostic nutritional index (PNI) showed a favorable univariable association and a borderline adjusted association.

Conclusion: In this adult Ewing sarcoma cohort, metastatic disease and elevated LDH were associated with inferior survival. LDH was the most consistent laboratory-based prognostic signal after adjustment for metastatic disease. PLR, SIRI, and PNI should be interpreted as exploratory markers requiring validation in larger cohorts.

Keywords: Ewing sarcoma, Adult, Survival analysis, Lactate dehydrogenase, Platelet-to-lymphocyte ratio, Prognostic nutritional index

Cite This Article: *Doğan T, Demiray AG, Yapar Taşköylü B, Yaren A, Gököz Doğu G. Survival Outcomes and Inflammatory-Nutritional Prognostic Markers in Adult Ewing Sarcoma: A Single-Center Retrospective Cohort. EJMI 2026;10(3):425–432.*

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Submitted Date: July 9, 2026 **Revised Date:** August 18, 2026 **Accepted Date:** August 19, 2026

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Ewing sarcoma is a rare malignant tumor that can originate in bone or soft tissue and is usually related to EWSR1-ETS fusion biology, most often EWSR1-FLI1.[1,2] Standard treatment is built around chemotherapy, with surgery and/or radiotherapy used for local control when feasible.[3,4] Although most literature discusses the disease in children, adolescents, or young adults, patients diagnosed after 18 years of age form a distinct, clinically relevant subgroup. Adult datasets are small, treatment delivery may differ from pediatric protocols, and real-world outcome data remain relatively sparse.[5,6] In almost all published series, metastatic disease at presentation is among the most consistent adverse prognostic factors.[7]

For diagnosis, morphology and immunohistochemistry must be interpreted together, and molecular confirmation is used when it is available. The current classification of small round cell sarcomas separates conventional Ewing sarcoma from other genetically defined tumors.[8,9] CIC-rearranged and BCOR-altered round cell sarcomas are no longer treated as variants of classic Ewing sarcoma.[8–10] This distinction is important in retrospective hospital series because molecular testing may not have been performed in every historical case. For that reason, the diagnostic basis of such cohorts should be described with appropriate caution.

In daily practice, baseline laboratory tests may add context to clinical risk assessment. Lactate dehydrogenase (LDH) has previously been associated with outcome in Ewing sarcoma and may act as a simple marker of tumor burden or aggressive biology.[11] Blood-count-derived inflammatory scores, including the neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, and related indices, have also been examined, but these markers are affected by tumor load, intercurrent inflammation, and treatment selection.[12,13] The prognostic nutritional index has mainly been studied in broader sarcoma populations, and data specific to Ewing sarcoma are limited.[14] We therefore evaluated survival outcomes and exploratory prognostic markers, particularly LDH and inflammatory-nutritional indices, in an adult single-center cohort.

Methods

Study Design and Patients

We performed a retrospective review at the medical oncology department of a tertiary university hospital (institution blinded for peer review). The hospital archive was screened for patients aged 18 years or older with histologically confirmed Ewing sarcoma diagnosed from May 2010 to January 2026 and treated and/or followed at the center. Follow-up was updated in May 2026. Of the 23 patients, 18 (78.3%) received their entire treatment at our center, whereas 5 (21.7%) had been diagnosed and/or initially treated elsewhere before referral**.** Patients were included when the records contained the diagnosis date, vital status, baseline clinicopathological information, treatment variables, and follow-up data required for survival analysis. The study used routine clinical records only; no extra tests were requested for research purposes.

From the records, we extracted age, sex, Eastern Cooperative Oncology Group (ECOG) performance status, tumor site, stage, histological grade, body mass index, treatment information, and pretreatment laboratory values. Disease extent was grouped as localized or metastatic according to the presentation at diagnosis, and the documented clinical stage was retained. Primary surgery and adjuvant therapy were recorded descriptively and were not interpreted as causal treatment effects. For laboratory-derived indices, analyses used patients with available values for the relevant marker; missing laboratory data for one marker did not remove the patient from the entire cohort.

Pathological and Molecular Assessment

Diagnostic information was taken from institutional pathology reports. Morphological features and immunohistochemical staining results were reviewed from the written records, including CD99, FLI1, and NKX2.2 when reported. In tested cases, EWSR1 rearrangement assessed by fluorescence in situ hybridization and/or reverse transcription polymerase chain reaction was used as supportive evidence, and available next-generation sequencing results were also recorded. When molecular testing was not available, the recorded diagnosis was accepted if it was supported by the combined morphological and immunohistochemical profile. Cases documented as CIC-rearranged or BCOR-altered round cell sarcoma were excluded. EWSR1 rearrangement was molecularly confirmed in 15 of 23 patients (65.2%), whereas the remaining 8 patients (34.8%) were diagnosed on the basis of compatible morphological and immunohistochemical findings without molecular confirmation.

Inflammatory and Nutritional Indices

Pretreatment complete blood count and biochemical results recorded at diagnosis were used for the inflammatory and nutritional indices. The calculated markers were the neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, lymphocyte-to-monocyte ratio, systemic immune-inflammation index, systemic inflammation response index, and C-reactive protein-to-albumin ratio.

The systemic immune-inflammation index was defined as platelet count multiplied by neutrophil count, divided by

lymphocyte count. The systemic inflammation response index was defined as neutrophil count multiplied by monocyte count, divided by lymphocyte count. The prognostic nutritional index was calculated using the formula 10 × albumin (g/dL) + 0.005 × total lymphocyte count (per mm^3); albumin values entered as g/L were converted to g/dL before calculation.

For survival comparisons, laboratory markers were divided at the median of this cohort. Values above the median were compared with values at or below the median. The thresholds used were LDH 259 U/L, PLR 169, PNI 49.7, SIRI 1.74, and SII 1085. The local laboratory upper limit of normal for LDH was 214 U/L. The cohort-median threshold of 259 U/L was therefore above the local upper limit of normal, indicating that the median-based dichotomization did not correspond to a conventional normal-versus-elevated LDH classification.

Endpoints and Follow-up

Overall survival was calculated from the diagnosis date to death from any cause; patients still alive were censored at the last follow-up. Progression-free survival was calculated from diagnosis to the first documented progression or death, whichever came first. Patients alive without progression were censored on the date of last contact. Follow-up was summarized with the reverse Kaplan–Meier method.[15]

Statistical Analysis

Numerical variables were described by median and interquartile range; categorical variables were given as counts and percentages. Kaplan–Meier estimates were used for survival curves, and the log-rank test was used for group comparisons. Univariable Cox proportional-hazards models were fitted for candidate predictors of overall survival, and results are shown as hazard ratios with 95% confidence intervals. When both tests were used, Cox and log-rank p values are reported separately.

To avoid overfitting in a small dataset, adjusted analyses were limited to two-variable exploratory Cox models. Each model included metastatic disease at diagnosis and one selected laboratory marker. Harrell's concordance index was used to summarize discrimination, and Schoenfeld residuals were checked for the proportional-hazards assumption. All tests were two-sided, with $p<0.05$ accepted as statistically significant. Because the cohort and event count were small, prognostic findings were interpreted as exploratory. Analyses were run in Python version 3 using the lifelines package.

Ethics

The Pamukkale University Non-Interventional Clinical Research Ethics Committee Non-Interventional Clinical Research Ethics Committee approved the study (committee name, approval number, and date blinded for peer review; full details are given on the title page). The study followed the principles of the Declaration of Helsinki. Written informed consent was waived because the analysis was retrospective.

Use of Artificial Intelligence–Assisted Technologies

OpenAI ChatGPT (GPT-5.5 Thinking; OpenAI; accessed July 2026) was used only for sentence-level English language and grammar editing. The prompts were limited to language-editing requests and instructed the tool not to change scientific content, study data, statistical analyses, numerical results, tables, figures, references, or interpretation. No AI tool was used to create or modify figures. The authors reviewed the final manuscript and take responsibility for its content.

Results

Patient Characteristics

Twenty-three adults with histologically confirmed Ewing sarcoma met the eligibility criteria. All were at least 18 years old at diagnosis. The median age was 36.4 years (IQR 20.0–46.1; range 18.0–71.8); 14 patients were younger than 40 years, and 9 were aged 40 years or older. The cohort included 12 male and 11 female patients. ECOG performance status (ECOG PS) was 0 in 12 patients, 1 in 9 patients, and 2 in 2 patients.

The primary site was extraosseous in 13 patients and skeletal in 10. Stage distribution was stage II in 7 patients, stage III in 7, and stage IV in 9. Nine patients had metastatic disease at diagnosis. Histological grade was grade 3 in 21 patients and grade 2 in 2. The median BMI was 22.7 kg/m^2 (IQR 21.7–25.0). Sixteen patients underwent primary surgery, and 15 received adjuvant treatment. The median baseline LDH was 259 U/L (IQR 196–400; n=21), and the median albumin was 43.0 g/L (IQR 38.0–44.2; n=21). Median NLR, PLR, and PNI were 4.0 (IQR 2.4–6.2), 169 (IQR 146–266), and 49.7 (IQR 45.9–52.9), respectively. EWSR1 rearrangement was molecularly confirmed in 15 patients (65.2%). Table 1 summarizes the baseline characteristics.

Survival Outcomes

The median observation time was 35.9 months (IQR 17.5–82.3). The reverse Kaplan–Meier median follow-up was 98.7 months. Twelve of the 23 patients died during follow-up. The median overall survival was 78.3 months. Estimated OS was 86.7% at 1 year, 67.9% at 2 years, and 52.4% at 5 years (Fig. 1).

Table 1. Baseline characteristics of the cohort (n=23)

Characteristic	Value
Age, years — median (IQR) [range]	36.4 (20.0–46.1) [18.0–71.8]
<40 / ≥40 years	14 (61%) / 9 (39%)
Sex — male / female	12 (52%) / 11 (48%)
ECOG performance status — 0 / 1 / 2	12 (52%) / 9 (39%) / 2 (9%)
Primary site — extraosseous / skeletal	13 (57%) / 10 (43%)
Stage — II / III / IV	7 (30%) / 7 (30%) / 9 (39%)
Metastatic disease at diagnosis	9 (39%)
Grade — 3 / 2	21 (91%) / 2 (9%)
BMI, kg/m^2 — median (IQR)	22.7 (21.7–25.0)
Primary surgery	16 (70%)
Adjuvant treatment	15 (65%)
LDH, U/L — median (IQR), n=21	259 (196–400)
Albumin, g/L — median (IQR), n=21	43.0 (38.0–44.2)
NLR — median (IQR), n=23	4.0 (2.4–6.2)
PLR — median (IQR), n=23	169 (146–266)
PNI — median (IQR), n=21	49.7 (45.9–52.9)
Deaths	12 (52%)
Median observation time, months (IQR)	35.9 (17.5–82.3)

Data are median (IQR) [range] or n (%). BMI: Body mass index; ECOG: Eastern Cooperative Oncology Group; IQR: Interquartile range; LDH: Lactate dehydrogenase; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; PNI: Prognostic nutritional index.

Thirteen progression-free survival events occurred. The median PFS was 15.9 months. Estimated PFS was 56.5% at 1 year and 47.8% at both 3 and 5 years (Fig. 2).

Factors Associated With Overall Survival

In univariable Cox analysis, metastatic disease at diagnosis was associated with a higher risk of death than localized disease (HR 6.14, 95% CI 1.74–21.7; Cox p=0.005; log-rank p=0.002). The Kaplan–Meier comparison is presented in Figure 3.

Patients who did not undergo primary surgery had worse OS than those who underwent surgery (HR 5.91, 95% CI 1.76–19.8; Cox p=0.004). The absence of adjuvant treatment likewise predicted shorter OS (HR 3.22, 95% CI 1.03–10.1; Cox p=0.045). Both the surgery and adjuvant-treatment variables were treated as observational and were not interpreted as treatment effects. Poorer performance status (ECOG PS ≥1 vs 0) conferred inferior OS (HR 4.33, 95% CI 1.27–14.8; Cox p=0.019). Age ≥40 years, however, showed no significant relationship with OS (HR 2.80, 95% CI 0.89–8.81; Cox p=0.078).

Among laboratory-based variables, an LDH >259 U/L predicted shorter OS (HR 6.63, 95% CI 1.77–24.9; Cox p=0.005; log-rank p=0.001). A PLR >169 likewise identified patients with poorer OS (HR 7.08, 95% CI 1.48–34.0; Cox p=0.014). Conversely, a higher PNI (>49.7) tracked with better OS (HR 0.25, 95% CI 0.07–0.94; Cox p=0.041; log-rank p=0.028). SIRI >1.74 showed a borderline univariable association with worse OS (HR 3.76, 95% CI 1.01–14.0; Cox p=0.048), whereas SII >1085 did not reach statistical significance. NLR, LMR, and CAR were not retained as main findings. Univariable OS analyses are summarized in Table 2.

Progression-Free Survival

PFS differed according to metastatic status at diagnosis. Among patients with localized disease (n=14), 5 PFS events

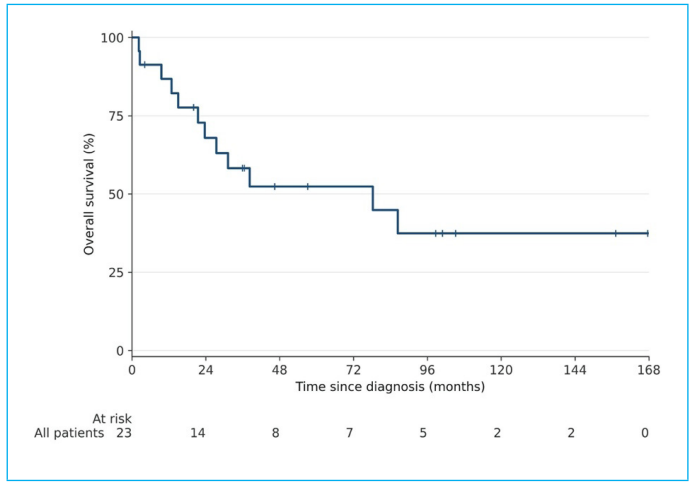

Figure 1. Kaplan–Meier estimate of overall survival for the whole cohort (median 78.3 months; 5-year survival 52.4%). The table below the curve shows the number of patients at risk.

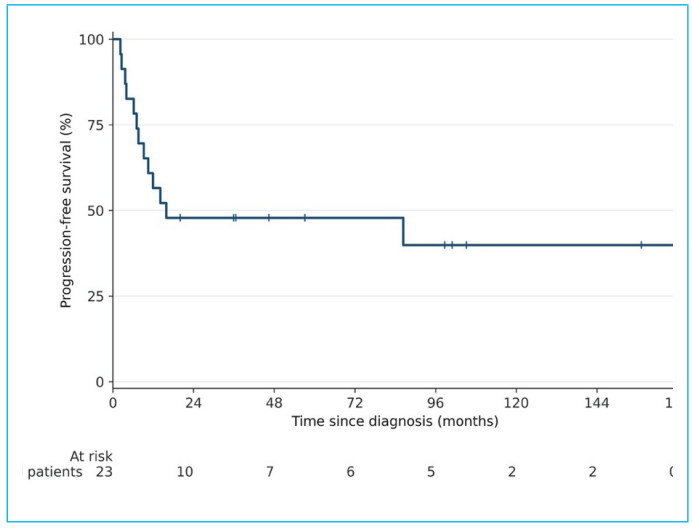

Figure 2. Kaplan–Meier estimate of progression-free survival for the whole cohort (median 15.9 months).

Table 2. Univariable analysis of overall survival

Variable (index vs reference)	HR (95% CI)	Cox p	Log-rank p
Metastatic vs localized	6.14 (1.74–21.7)	0.005	0.002
No primary surgery vs surgery	5.91 (1.76–19.8)	0.004	0.001
ECOG PS ≥1 vs 0	4.33 (1.27–14.8)	0.019	0.012
No adjuvant vs adjuvant	3.22 (1.03–10.1)	0.045	0.034
Age ≥40 vs <40 years	2.80 (0.89–8.81)	0.078	0.066
LDH >259 U/L	6.63 (1.77–24.9)	0.005	0.001
PLR>169	7.08 (1.48–34.0)	0.014	0.005
PNI >49.7	0.25 (0.07–0.94)	0.041	0.028
SIRI>1.74	3.76 (1.01–14.0)	0.048	0.034
SII>1085	2.75 (0.82–9.21)	0.100	0.086

Hazard ratios are from Cox proportional-hazards regression; p values are reported separately for Cox regression and the log-rank test. Indices were dichotomized at the median. NLR, LMR, and CAR were not retained as main findings.
CI: Confidence interval; HR: Hazard ratio; CAR: C-reactive protein-to-albumin ratio; LMR: Lymphocyte-to-monocyte ratio; SII: Systemic immune-inflammation index; SIRI: Systemic inflammation response index.

occurred, and the median PFS was not reached. In patients with metastatic disease (n=9), 8 PFS events occurred, and the median PFS was 6.2 months. The difference between the two groups was statistically significant by the log-rank test (p<0.001; Fig. 4). PFS results according to metastatic status are presented in Table 3.

Exploratory Adjusted Cox Models

Two-variable Cox models adjusted for metastatic disease were used for exploratory assessment of LDH and PNI. Each model included 21 patients and 12 OS events (Table 4).

In the LDH model, metastatic disease remained associated with shorter OS (adjusted HR 4.49, 95% CI 1.16–17.4; p=0.030). LDH >259 U/L also retained its association with worse OS after adjustment for metastatic disease (adjusted HR 6.13, 95% CI 1.55–24.2; p=0.010). The model C-index was 0.79, and the proportional hazards assumption was acceptable.

In the PNI model, metastatic disease remained associated with OS (adjusted HR 4.65, 95% CI 1.33–16.2; p=0.016). PNI >49.7 showed a favorable association after adjustment, but this did not reach conventional statistical significance (adjusted HR 0.27, 95% CI 0.07–1.01; p=0.052). The C-index was 0.79, and the proportional hazards assumption was acceptable. Given the limited number of events, adjusted analyses were interpreted as exploratory.

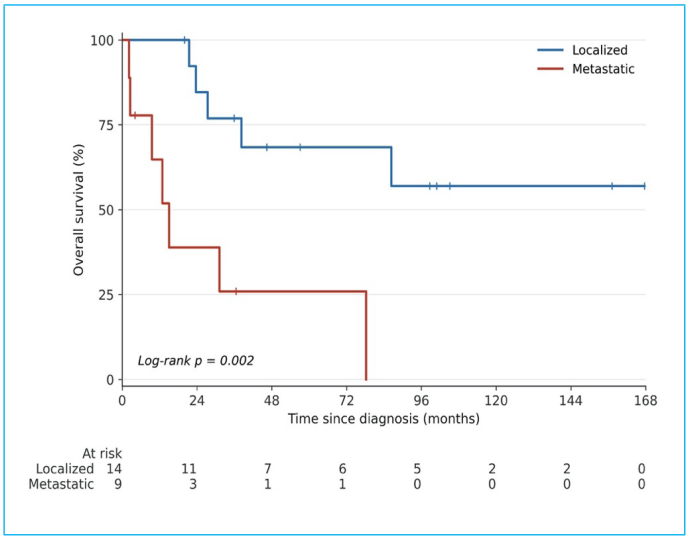

Figure 3. Overall survival according to metastatic status at diagnosis (log-rank p=0.002).

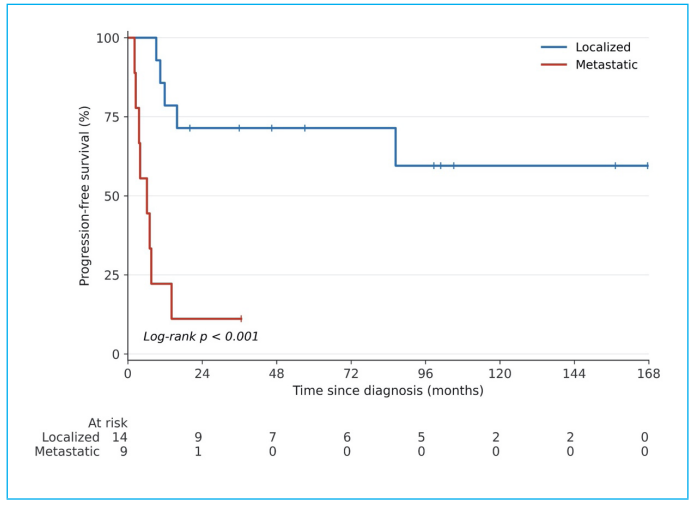

Figure 4. Progression-free survival according to metastatic status at diagnosis (log-rank p<0.001).

Note: Figures are provided as separate high-resolution JPEG files (Figure 1–Figure 4) and are not embedded in the manuscript file.

Table 3. Progression-free survival by metastatic status

Group	n	Events	Median PFS	Log-rank p
Localized	14	5	Not reached	< 0.001
Metastatic	9	8	6.2 months	

PFS was calculated from diagnosis to first progression or death. PFS: Progression-free survival.

Table 4. Exploratory two-variable cox models adjusted for metastatic disease

Model / covariate	Adjusted HR (95% CI)	p
LDH model (n=21, 12 events)		
Metastatic disease	4.49 (1.16–17.4)	0.030
LDH >259 U/L	6.13 (1.55–24.2)	0.010
PNI model (n=21, 12 events)		
Metastatic disease	4.65 (1.33–16.2)	0.016
PNI >49.7	0.27 (0.07–1.01)	0.052

Both models had a C-index of 0.79 with an acceptable proportional-hazards assumption. Given 12 events, models are exploratory. HR: Hazard ratio; CI: Confidence interval; LDH: Lactate dehydrogenase; PNI: Prognostic nutritional index.

Discussion

This adult single-center series provides survival data together with an exploratory evaluation of routine laboratory markers. Median OS was 78.3 months, and median PFS was 15.9 months. Patients with metastatic disease at diagnosis had clearly poorer outcomes, particularly for PFS, for which the median was 6.2 months in the metastatic subgroup. Among the laboratory markers, LDH gave the most consistent signal; LDH >259 U/L remained associated with OS after adjustment for metastatic disease. PNI was favorable in direction but borderline after adjustment, while PLR and SIRI were limited to univariable associations.

Compared with pediatric and adolescent Ewing sarcoma, adult disease is represented by fewer and more heterogeneous cohorts. Differences in age range, stage distribution, primary site, and treatment feasibility make direct comparison difficult. In the present cohort, 9 of 23 patients (39%) had metastatic disease at diagnosis, which closely parallels the 38.5% reported by Uyeturk et al.[6] in a Turkish adult series. Survival also fell within a similar range: our median OS was 78.3 months, with a 5-year OS of 52.4%, whereas Uyeturk et al.[6] reported a median OS of 91 months and a 4-year OS of 54%. The multicenter Anatolian Society of Medical Oncology study also underlined the prognostic role of disease extent and treatment patterns in adults.[5] These comparisons support the clinical plausibility of the present dataset despite its small size. Recent adult cohorts and registry analyses similarly show that age, stage, primary site, local treatment feasibility, and age-related treatment differences influence prognosis.[16–19] Therefore, this study should be read as a real-world exploratory cohort rather than a definitive prognostic model.

The unfavorable effect of metastatic presentation was expected and aligns with the broader Ewing sarcoma literature. In this cohort, metastatic disease was linked to inferior OS and produced a large separation in PFS curves. Median PFS was 6.2 months for metastatic disease, whereas it was not reached for localized disease. This metastatic PFS estimate is low compared with some adult reports, but it is still compatible with the poor event-free survival described in metastatic adult and real-world series.[7,16,17] The estimate should be interpreted cautiously because it comes from only nine metastatic patients and eight PFS events. Metastatic burden, referral timing, treatment feasibility, and progression before completion of multimodal therapy may all have influenced this result. These same selection issues also explain why primary surgery and adjuvant treatment cannot be viewed as treatment-effect variables in this dataset; local therapy is usually offered to patients with more favorable disease distribution, operability, response, and performance status. The associations of primary surgery and adjuvant treatment with longer survival should not be interpreted as evidence of treatment efficacy. Treatment selection in this retrospective cohort was likely influenced by baseline disease characteristics, including metastatic status, operability, and performance status. These factors may have confounded the observed survival differences.

LDH was the most consistent laboratory-based prognostic signal in this cohort. Patients with LDH >259 U/L had shorter OS in univariable analysis, and the association persisted after adjustment for metastatic disease. This is in keeping with the meta-analysis by Li et al.[11], which reported worse OS and 5-year disease-free survival in patients with elevated LDH. Older Ewing sarcoma work also connected high pretreatment LDH with metastatic burden and poorer disease-free survival, supporting the view that LDH partly reflects tumor load or high cellular turnover.[20] Our result also resembles the pediatric bone Ewing sarcoma series by Del Baldo et al.[21], in which LDH retained prognostic value after adjustment for established clinical factors, although that population differs from the adult cohort studied here. The effect size in our data was large, but the confidence intervals were wide, and the adjusted model included only 21 patients with 12 OS events. LDH should therefore be considered a consistent exploratory signal, not a validated independent biomarker.

The inflammatory indices did not show a uniform pattern. PLR >169 and SIRI >1.74 were associated with worse OS in

univariable analysis, but NLR, LMR, CAR, and SII were not retained as main findings. This only partly matches earlier Ewing sarcoma reports. Li et al.[12] described prognostic associations for several inflammatory markers, including NLR, PLR, CRP-to-albumin ratio, and Glasgow Prognostic Score-related indices, while Bal et al.[13] examined practical inflammatory markers such as NLR and SII. In the present study, PLR and SIRI remained limited to univariable associations. This is understandable in a small adult cohort because blood-count-derived indices can be affected by disease burden, infection, corticosteroid exposure, nutritional status, and timing of sampling. These results should therefore be treated as supportive and hypothesis-generating, not as stand-alone prognostic tools.

PNI suggested a favorable but weaker signal. Higher PNI was linked to better OS in univariable analysis, and the direction remained favorable after adjustment for metastatic disease, although the adjusted p value was borderline. Because PNI combines albumin and lymphocyte count, it may capture nutritional reserve, systemic inflammation, and immune status. Still, PNI is not established specifically for Ewing sarcoma. Most available evidence comes from broader soft tissue sarcoma cohorts, where lower PNI has been associated with poorer outcomes.[14,22] In this cohort, PNI should be viewed as hypothesis-generating. Its role may be clinically relevant in adults receiving intensive multimodal treatment, but larger Ewing sarcoma studies with standardized nutritional and inflammatory evaluation are needed.

These findings should be interpreted in light of several limitations. The study was retrospective, single-center, and small, with 23 patients, 12 OS events, and 13 PFS events. This event count restricted modeling, so adjusted analyses were limited to two-variable Cox models. Although this was done to reduce overfitting, the estimates remain unstable and cannot be considered confirmatory.[23] Continuous laboratory markers were dichotomized at cohort-specific medians. This approach may have reduced statistical power, and the resulting thresholds reflect the distribution of the present cohort and should not be regarded as clinically validated cut-off values. Primary surgery and adjuvant treatment are also prone to selection bias**.** A minority of patients were referred after initial management elsewhere, which may have introduced some referral-related selection bias. Molecular confirmation of EWSR1 rearrangement was available in 15 of 23 patients (65.2%), so overlap with Ewing-like round cell sarcomas cannot be fully excluded, especially under the current separation of conventional Ewing sarcoma, CIC-rearranged sarcoma, and BCOR-altered round cell sarcoma.[8–10] The cohort also had limited power to assess tumor size, metastatic sites, chemotherapy regimens, radiotherapy details, and local control strategies. Within these limits, LDH was the most consistent laboratory marker, whereas PNI, PLR, and SIRI remained exploratory.

Conclusion

In this small retrospective adult cohort, metastatic presentation and elevated baseline LDH were associated with worse survival in Ewing sarcoma. PLR, SIRI, and PNI showed exploratory signals, but they should not be regarded as validated prognostic markers. Larger adult Ewing sarcoma cohorts are needed to confirm whether simple laboratory parameters can add value to established clinical risk factors.

Disclosures

Ethics Committee Approval: The study was approved by the Pamukkale University Non-Interventional Clinical Research Ethics Committee (approval number: E-60116787-020-898373 [decision no. 12]; date: 30 June 2026).

Informed Consent: Written informed consent was waived because the analysis was retrospective.

Conflict of Interest: The authors declare that they have no conflict of interest.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Use of Artificial Intelligence–Assisted Technologies

OpenAI ChatGPT (GPT-5.5 Thinking; OpenAI; accessed July 2026) was used only for sentence-level English language and grammar editing. The prompts were limited to language-editing requests and instructed the tool not to change scientific content, study data, statistical analyses, numerical results, tables, figures, references, or interpretation. No AI tool was used to create or modify figures. The authors reviewed the final manuscript and take responsibility for its content.

Authorship Contributions: Concept – TD, AGD; Design – TD, AGD, AY; Supervision – AY, GGD; Data collection and/or processing – TD, BYT, GGD; Analysis and/or interpretation – TD, AGD, AY; Literature search – TD, BYT; Writing – TD; Critical review – AGD, BYT, AY, GGD.

Peer-review: Externally peer-reviewed.

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