ORIGINAL PAPER

Enfortumab vedotin demonstrates consistent efficacy in platinum- and ICI-refractory metastatic urothelial carcinoma patients irrespective of prior response: a multicenter real-world study

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Introduction Enfortumab vedotin (EV) is an antibody–drug conjugate (ADC) targeting nectin-4 and is widely used for patients with locally advanced or metastatic urothelial carcinoma (mUC) after prior systemic therapies. However, it remains unclear whether EV efficacy is influenced by responses to preceding platinum-based chemotherapy (PBC) or immune checkpoint inhibitors (ICI). This study evaluated the relationship between responses to prior therapy and EV outcomes in a real-world cohort.

Material and methods We retrospectively analyzed 60 patients with mUC who received EV between December 2021 and March 2024. All patients had previously undergone PBC and ICI. Based on responses to these therapies, patients were classified as responders (complete or partial response) or non-responders (stable or progressive disease). Primary endpoints were objective response rate (ORR), progression-free survival (PFS), and overall survival (OS). Multivariable Cox proportional hazards models were used to evaluate factors associated with survival outcomes.

Results The overall EV ORR was 41.7%. EV responders demonstrated significantly longer PFS and OS than non-responders. No significant differences in EV efficacy were observed between responders and non-responders to prior chemotherapy or ICI. Multivariable analyses confirmed that prior treatment response was not independently associated with PFS or OS.

Conclusions EV response was strongly associated with prolonged survival. Importantly, consistent antitumor activity was observed regardless of prior treatment efficacy, suggesting that even patients refractory to PBC or ICI may derive meaningful benefit. EV should be offered promptly even for patients with double-refractory mUC, as the presence of a prior non-response does not signify compromised efficacy.

Key Words: enfortumab vedotin ‹› metastatic urothelial carcinoma ‹› objective response rate ‹› overall survival ‹› prior treatment ‹› progression-free survival

INTRODUCTION

Metastatic urothelial carcinoma (mUC), encompassing both bladder and upper tract urothelial cancers, remains a potentially lethal disease. Although platinum-based chemotherapy (PBC) achieves response rates of approximately 40–50%, median overall survival rarely exceeds 15 months, and nearly all patients eventually experience disease progression [1]. The introduction of immune checkpoint inhibitors (ICI) has led to the achievement of durable responses in a subset of patients, but the majority still develop resistance or relapse over time [2].

Enfortumab vedotin (EV) is a first-in-class ADC targeting nectin-4, a cell adhesion molecule highly expressed in urothelial carcinoma. After binding to nectin-4, EV is internalized and releases monomethyl auristatin E (MMAE), a potent microtubule inhibitor that disrupts cell division and induces apoptosis [3]. In the pivotal EV-201 trial, EV led to an objective response rate (ORR) of 44%, including a complete response in 12% of patients previously treated with PBC and PD-1/PD-L1 inhibitors [4]. The phase III EV-301 trial further demonstrated superior survival compared with standard chemotherapy: overall survival (12.9 vs 9.0 months, respectively; HR = 0.70, p <0.001) and progression-free survival (5.6 vs 3.7 months, respectively; HR = 0.62) [5]. Moreover, the EV-302 trial showed that first-line EV combined with pembrolizumab significantly improved PFS and OS compared with PBC [6].

Despite these encouraging results, it remains uncertain whether the efficacy of EV is influenced by prior treatment responses. Some anticancer agents exhibit cross-resistance or dependence on tumor biology influenced by earlier therapy, potentially affecting subsequent treatment outcomes. To clarify this, we investigated whether the efficacy of EV correlates with the response to prior PBC or ICI in patients with mUC.

MATERIAL AND METHODS

Study population

We retrospectively reviewed the medical records of 60 consecutive patients with histologically confirmed locally advanced or metastatic urothelial carcinoma who had been treated with EV monotherapy between December 2021 and March 2024 at our institution or affiliated centers. All patients had received prior systemic therapy, including PBC and PD-1/PD-L1 inhibitors.

For the purpose of this analysis, first-line PBC included regimens administered in the neoadjuvant, adjuvant, and metastatic settings. ICI were likewise administered in different clinical contexts, including adjuvant therapy, maintenance therapy for metastatic disease, and treatment following progression after PBC.

For patients who received multiple chemotherapy or immunotherapy regimens across neoadjuvant, adjuvant, or metastatic settings, the best overall response observed among all regimens was used to define prior responder status. Patients were initially categorized into four groups according to their response to prior therapies: group 1 – responders to both PBC and ICI; group 2 – responders to PBC only; group 3 – responders to ICI only; and group 4 – non-responders to both modalities. Groups 1–3 were then consolidated into a single "Prior responders" category, while group 4 was defined as "Prior non-responders".

Treatment and assessment

EV was administered intravenously at a standard dose of 1.25 mg/kg on days 1, 8, and 15 of a 28-day cycle. However, dosage and scheduling were modified at the discretion of each attending physician. Treatment was continued until disease progression or the development of unacceptable toxicity. Tumor response was evaluated every 4–12 weeks by computed tomography according to RECIST version 1.1. The response to prior chemotherapy and ICI was classified as complete or partial response (CR/PR, responders) or stable or progressive disease (SD/PD, non-responders).

Endpoints and statistical analysis

The primary endpoints were EV objective response rate (ORR), progression-free survival (PFS), and overall survival (OS). PFS was defined as the time from EV initiation to radiographic disease progression or death from any cause, and OS as the time from EV initiation to death from any cause. Survival probabilities were estimated using the Kaplan-Meier method, and group comparisons were made using the log-rank test. Cox proportional hazards regression analyses were performed to evaluate factors associated with PFS and OS. Clinically relevant variables were included in multivariable models, and the hazard ratio (HR) with 95% confidence interval (CI) was reported. Differences between groups were analyzed using the chi-squared or Mann-Whitney U test as appropriate. All statistical analyses were performed using EZR software (Saitama Medical Center, Jichi Medical University, Japan), with p <0.05 considered significant.

Bioethical standards

This study was approved by the Clinical Research Review Board of Hamamatsu University School of Medicine (IRB#21-090). In accordance with the opt-out procedure approved by the institutional review board, individual informed consent was not required.

RESULTS

Patient characteristics

Baseline characteristics of the 60 enrolled patients are summarized in Table 1. The median age was 73 years (IQR: 71–78 years), and 42 patients (70%) were male. The primary tumor originated from the upper urinary tract or bladder in 36 and 24 patients, respectively. The median observation period was 11.0 months (IQR: 3.7–14.5 months). The baseline characteristics stratified according to prior treatment response are presented in Table 2. No significant differences were observed between the two groups.

Efficacy of enfortumab vedotin

Across the entire cohort, EV led to ORR of 41.7% (25/60). Median PFS and OS were 6.9 (IQR: 2.8–13.1) and 11.8 (IQR: 6.0–23.4) months, respectively. Patients who achieved a response to EV exhibited significantly longer survival than non-responders, with median PFS of 10.9 months (IQR: 7.2–21.3 months) vs 3.6 months (IQR: 2.0–10.5 months) (p = 0.0034) and median OS of 21.2 (25th percentile, 10.4 months; upper quartile not reached) vs 9.6 months (IQR: 3.7–23.4 months) (p = 0.0194), respectively (Figures 1 and 2).

Impact of prior treatment response

When stratified by prior therapy, no significant correlation was observed between a prior treatment response and EV outcomes. Among chemotherapy responders (n = 21) and non-responders (n = 39), ORR was 40 and 31%, respectively (p = 0.58). Median PFS was 7.2 months (IQR: 4.7–15.6 months) and 5.2 months (IQR: 2.5–11.5 months) (p = 0.25), and median OS was 16.3 months (IQR, 8.6–23.4 months) and 11.2 months (IQR, 3.7–21.3 months) (p = 0.36), respectively (Figures 3 and 4).
Similarly, among ICI responders (n = 12) and non-responders (n = 48), ORR was 32 and 11% (p = 0.10), median PFS was 7.2 (IQR: 2.8–10.4) and 6.0 (IQR: 2.6–13.2) months (p = 0.91), and median OS was 18.4 (25th percentile, 2.9 months; upper quartile not reached) and 11.8 (25th percentile, 6.3 months; up-

Table 1. *No significant differences in baseline characteristics were observed between EV responders and non-responders*

Characteristics	EV responders (n = 25)	EV non-responders (n = 35)	p-value
Age [years], median (IQR)	75 (71–78)	72 (66–77.5)	0.18
Sex [n (%)]			
Male	19 (76.0)	23 (65.7)	0.57
Female	6 (24.0)	12 (34.3)	
ECOG PS [n (%)]			
0–1	23 (92.0)	32 (91.4)	0.84
≥2	2 (8.0)	3 (8.6)	
BMI, median (IQR)	21.4 (19.5–24.5)	20.9 (18.9–26.0)	0.39
Primary lesion [n (%)]			
Upper urinary tract	13 (52.0)	23 (65.7)	0.59
Bladder or other	12 (48.0)	12 (34.3)	
Site of metastasis [n (%)]			
Regional lymph node	11 (44.0)	23 (65.7)	0.12
Extra-regional lymph node	9 (36.0)	14 (40.0)	0.79
Lung	16 (64.0)	18 (51.4)	0.43
Bone	2 (8.0)	9 (25.7)	0.10
Liver	9 (36.0)	8 (22.9)	0.38
Other	10 (40.0)	8 (22.9)	0.17

BMI – body mass index; ECOG PS – Eastern Cooperative Oncology Group performance status

Table 2. *No significant differences in baseline characteristics according to response to prior systemic therapy were observed between Prior responders and Prior non-responders*

Characteristics	Prior responders (n = 29)	Prior non-responders (n = 31)	p-value
Age [years], median (IQR)	74 (69–76)	72 (66–79)	0.90
Sex [n (%)]			
Male	22 (75.9)	20 (64.5)	0.41
Female	7 (24.1)	11 (35.5)	
ECOG PS [n (%)]			
0–1	27 (93.1)	28 (90.3)	1.00
≥2	2 (8.0)	3 (9.7)	
BMI, median (IQR)	21.1 (19.5–23.0)	21.2 (18.6–23.6)	0.98
Primary lesion [n (%)]			
Upper urinary tract	17 (58.6)	17 (54.8)	1.00
Bladder or other	12 (41.4)	12 (38.8)	
Site of metastasis [n (%)]			
Regional lymph node	17 (58.6)	17 (54.8)	0.80
Extra-regional lymph node	11 (37.9)	12 (38.7)	1.00
Lung	14 (48.3)	20 (64.5)	0.30
Bone	7 (24.1)	4 (12.9)	0.33
Liver	9 (31.0)	8 (25.8)	0.78
Other	11 (37.9)	7 (22.6)	0.26

BMI – body mass index; ECOG PS – Eastern Cooperative Oncology Group performance status

per quartile not reached) months (p = 0.67), respectively (Figures 5 and 6).

Multivariable Cox proportional hazards analysis

To further evaluate factors associated with survival outcomes, multivariable Cox proportional hazards regression analyses were performed including baseline variables (age ≥75 years, ECOG performance status ≥2, primary tumor site [upper tract vs bladder], presence of liver metastasis, prior PBC response, and prior ICI response) (Table 3).
For PFS, liver metastasis was independently associated with an increased risk of disease progression (HR = 2.34, 95% CI: 1.05–5.19, p <0.05). No other baseline variables, including prior PBC or ICI response, were significantly associated with PFS.

For OS, none of the included baseline factors reached statistical significance in the multivariable model. These findings indicate that baseline liver metastasis status, rather than prior treatment response, was the principal factor associated with disease progression following EV therapy in this cohort.
Overall, both univariable and multivariable analyses consistently demonstrated that survival outcomes following EV therapy were not significantly influenced by prior PBC or ICI response.

DISCUSSION

This study demonstrated that EV is of marked clinical benefit to patients with metastatic urothelial carcinoma, regardless of their response to prior treatments. Patients who responded to EV achieved

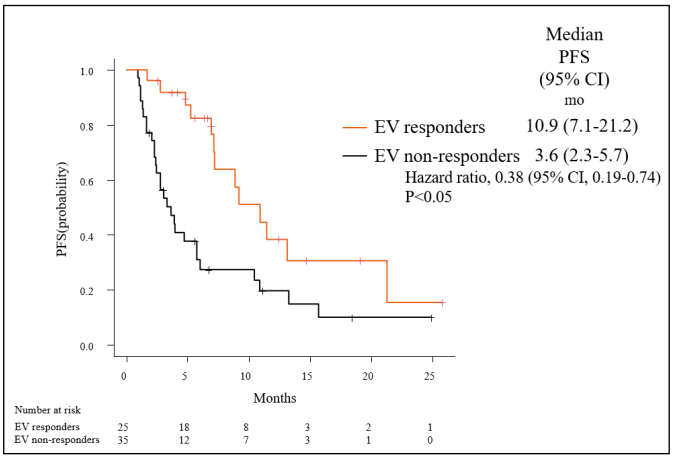

Figure 1. *PFS was significantly longer in EV responders compared with non-responders.*

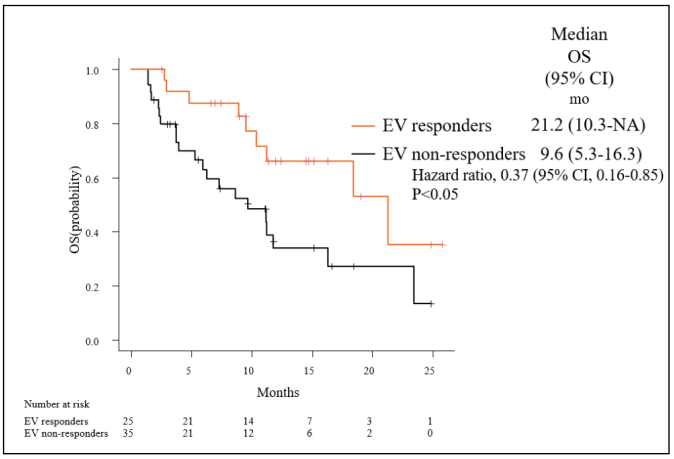

Figure 2. *OS was significantly longer in EV responders compared with non-responders.*

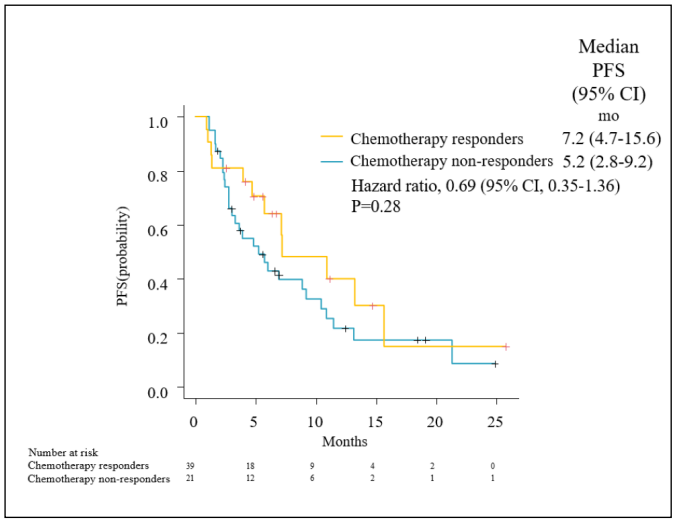

Figure 3. *There was no significant difference in PFS between chemotherapy responders and non-responders.*

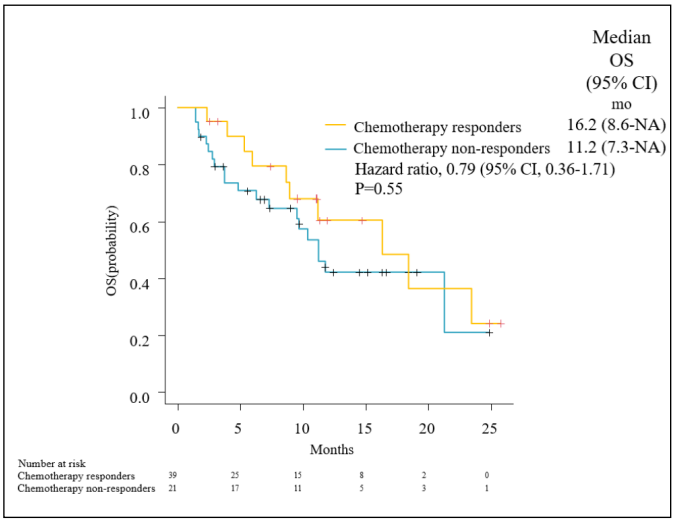

Figure 4. *There was no significant difference in OS between chemotherapy responders and non-responders.*

significantly longer PFS and OS than non-responders, confirming that EV response is a strong predictor of prolonged survival. Although this finding may appear obvious, it remains a clinically important observation because it emphasizes the critical role of a response to EV to achieve improved outcomes.
More importantly, EV efficacy appeared to be independent of prior chemotherapy or immunotherapy outcomes. Multivariable Cox proportional hazards analyses incorporating baseline clinical variables confirmed that prior response to PBC or ICI was not independently associated with either PFS or OS. These findings indicate that resistance to earlier systemic therapies does not necessarily predict diminished benefit from EV and support the concept that EV retains antitumor activity irrespective of prior treatment sensitivity.
In contrast, liver metastasis emerged as an independent adverse prognostic factor for PFS. Hepatic involvement has consistently been associated with poor outcomes in mUC across treatment eras. In the pre-immunotherapy setting, liver metastasis was identified as an independent predictor of worse survival in patients receiving PBC [7]. Similar findings have been reported in the ICI era [8]. These observations likely reflect more aggressive tumor biology and a potentially immunosuppressive microenvironment associated with liver metastases. In our cohort, liver metastasis independently predicted shorter PFS but not OS, possibly due to the influence of subsequent therapies and post-progression management on overall survival.
From a biological perspective, this observation is plausible given the distinct mechanism of action of EV. ADC targeting nectin-4, which is highly and uniformly expressed on urothelial carcinoma cells, delivers the MMAE directly into the tumor microenvironment, thereby inducing apoptosis through microtubule inhibition [3]. This mechanism differs fundamentally from platinum-based cytotoxic chemotherapy and immune checkpoint inhibition and may explain why EV maintains activity in tumors that have developed resistance to conventional cytotoxic or immune-based therapies.
The present findings are consistent with prior pivotal trials. The phase III EV-301 trial demonstrated that EV significantly prolonged both OS and PFS compared with standard chemotherapy in patients who had received prior PBC and ICI [5]. The phase II EV-201 study also showed comparable

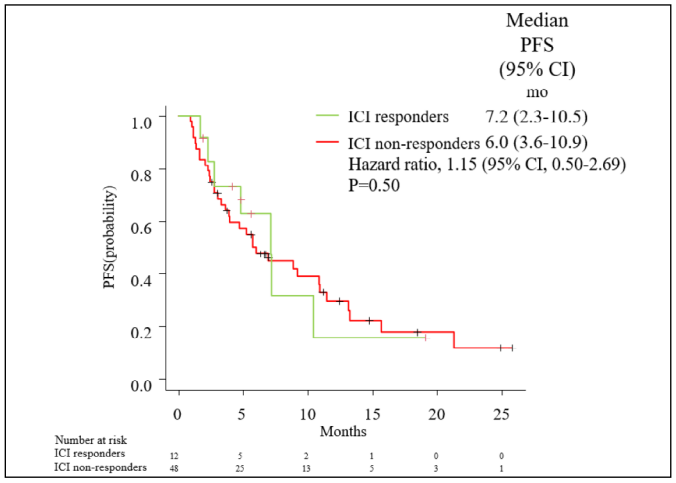

Figure 5. *There was no significant difference in PFS between ICI responders and non-responders.*

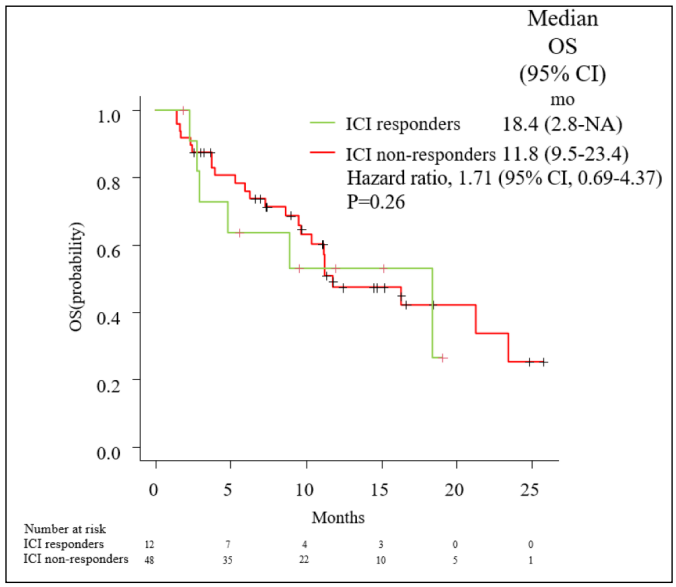

Figure 6. *There was no significant difference in OS between ICI responders and non-responders.*

Table 3. *Multivariable Cox proportional hazards analysis showed no independent association between prior treatment response and either PFS or OS*

Variable	OS HR (95% CI)	p-value	PFS HR (95% CI)	p-value
Age ≥75 years	1.11 (0.49–2.54)	0.80	1.04 (0.52–2.07)	0.90
ECOG PS ≥2	1.80 (0.41–7.82)	0.43	0.62 (0.16–2.46)	0.50
Upper tract	0.98 (0.42–2.27)	0.97	0.92 (0.46–1.85)	0.83
Liver metastasis	1.26 (0.50–3.19)	0.63	2.34 (1.05–5.19)	<0.05
Prior chemo response	0.71 (0.33–1.57)	0.40	0.70 (0.35–1.41)	0.32
Prior ICI response	1.30 (0.47–3.57)	0.61	0.70 (0.29–1.73)	0.45

ECOG PS – Eastern Cooperative Oncology Group performance status; ICI – immune checkpoint inhibitors; OS – overall survival; PFS – progression-free survival

response rates regardless of prior exposure to PD-1 or PD-L1 inhibitors [4]. Furthermore, recent real-world studies confirmed that EV remains effective across a broad range of patients, including those refractory to previous treatments [9].

In the evolving therapeutic landscape, combination strategies such as EV plus pembrolizumab have demonstrated superior survival outcomes in the first-line setting and are reshaping treatment algorithms for mUC. Nevertheless, EV monotherapy remains clinically relevant in several contexts, including patients who are ineligible for combination therapy, those treated in health care systems where combination regimens are not yet accessible, and patients who have already progressed after multiple prior therapies. Real-world data therefore remain essential to define the positioning of EV outside the controlled environment of phase III trials.

In addition to its robust antitumor activity, EV has demonstrated practicality in vulnerable and frail patient populations. For example, in EV-201 cohort 2, which included cisplatin-ineligible patients with prior ICI exposure, the confirmed ORR reached 52%, including a 20% CR rate [10]. Furthermore, real-world evidence supports the feasibility of EV in elderly patients. A multicenter retrospective study from Japan found that patients aged ≥80 years achieved comparable ORR, PFS, and OS compared with younger patients, with no significant differences in the frequency of dose reductions or treatment discontinuation [9]. These findings indicate that EV is a viable treatment option for patients with limited physiological reserve, such as those ineligible for cisplatin or with multiple comorbidities, thereby expanding therapeutic options beyond conventional cytotoxic regimens.

The safety profile of EV is well characterized and manageable. Common adverse events include skin rash, fatigue, and peripheral neuropathy, reflecting ADC's mechanism. In EV-201, grade ≥3 treatment-related adverse events occurred in 55% of patients, with the most frequent being neutropenia, rash, and fatigue [10]. Similarly, in the long-term follow-up study of EV-301, the incidence of high-grade toxicity remained comparable between EV and chemotherapy groups, and no new adverse events were observed [12]. While dermatologic and neurologic events were more frequent with EV, the incidence of hematologic toxicity was lower than that observed with PBC. Importantly, treatment discontinuation due to toxicity was relatively rare, and older patients did not exhibit an increased risk of adverse events [11]. These data suggest that EV can be administered safely in routine practice, with proactive management of its expected toxicities.

Clinically, these results underscore two important implications. First, achieving a tumor response with EV translates directly into survival benefit, reinforcing the importance of timely initiation, optimized dosing, and proactive management of adverse events to maintain treatment continuity. Second, patients with failed prior therapies should still be offered EV without delay, as prior non-responsiveness is not predictive of poor outcomes with EV. Consequently, the therapeutic opportunity for EV should never be overlooked in eligible patients with mUC.

In addition to clinical efficacy, economic considerations are increasingly important in treatment selection. A recent European analysis highlighted the substantial and rising financial burden associated with guideline-recommended systemic therapies for mUC following the introduction of novel immunotherapies and targeted agents [13]. The expanding use of ADC and combination regimens further amplifies cost pressures across healthcare systems. In this context, real-world evaluations of treatment benefit, particularly in heavily pretreated populations, are relevant not only from a clinical perspective but also in terms of resource allocation and sustainability.

Several limitations should be acknowledged. First, this study was retrospective in design and included a relatively small sample, which may limit statistical power. Second, prior systemic therapies were heterogeneous in terms of treatment setting, timing, and intensity. PBC and ICI were administered across neoadjuvant, adjuvant, and metastatic contexts, and several patients received multiple regimens over the course of their disease. Although prior responder status was defined using the best overall response across all regimens to standardize classification, this approach may oversimplify prior treatment exposure, and residual confounding cannot be excluded. Third, molecular or genomic predictors of EV response were not evaluated, despite their increasing relevance in precision oncology. Therefore, the present findings should be interpreted as hypothesis-generating and warrant validation in larger prospective cohorts with more standardized treatment sequences.

CONCLUSIONS

In this real-world retrospective study of 60 patients with metastatic urothelial carcinoma, EV demonstrated clinically meaningful antitumor activity, achieving ORR of 41.7% and prolonging survival among responders. Importantly, EV ef-

ficacy appeared to be independent of prior chemotherapy or immunotherapy response, suggesting that patients refractory to previous regimens may still derive benefit.

These findings support the clinical relevance of EV in heavily pretreated populations in the contemporary treatment landscape. However, given the retrospective design and limited sample size, the present results should be interpreted as hypothesis-generating and warrant validation in larger prospective studies.

CONFLICT OF INTERESTS

Teruo Inamoto co-author have received honoraria and advisory fees from Astellas Pharma Inc. The other authors declare no conflict of interest.

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ETHICS APPROVAL STATEMENT

This study was approved by the Clinical Research Review Board of Hamamatsu University School of Medicine (IRB#21-090).

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