

# Steroid Premedication Impact on Efficacy and Cutaneous Toxicity of Enfortumab Vedotin for Advanced Urothelial Carcinoma

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## Abstract

*Background/Aim:* The impact of steroid premedication on the efficacy and cutaneous toxicity of enfortumab vedotin (EV) in advanced urothelial carcinoma (UC) is unclear.

*Patients and Methods:* We retrospectively analyzed consecutive patients with advanced UC who received EV after the failure of platinum-based chemotherapy and immune checkpoint inhibitors from December 2021 to November 2024.

*Results:* Twenty-eight patients (male, n=16; median age, 71 years) were enrolled. Dexamethasone 6.6 mg was administered intravenously prior to EV in six (21.4%) patients. There were no differences in the overall response and disease control rates between patients with and without steroid premedication ( $p=0.653$  and  $p>0.99$ , respectively). The progression-free survival was not significantly associated with or without steroid premedication (not estimable vs. 4.3 months,  $p=0.501$ ). There were no marked differences in the incidence of all grades of EV-related cutaneous adverse events (AEs) between patients with and without steroid premedication (33.3% vs. 45.5%,  $p=0.673$ ). There was no significant difference in the incidence of grade  $\geq 3$  EV-related cutaneous AEs between the patients with and without steroid premedication (16.7% vs. 36.4%,  $p=0.630$ ). Multivariate analysis revealed that a performance status  $\geq 2$  was an independent prognostic factor for progression-free survival (hazard ratio=4.653, 95% confidence interval=1.263-17.140,  $p=0.021$ ), and steroid premedication was not ( $p=0.869$ ).

*Conclusion:* In EV treatment, steroid premedication did not affect clinical outcomes. The incidence and severity of EV-related cutaneous toxicity tended to improve in patients who received steroid premedication, although no significant differences were observed.

**Keywords:** Urothelial carcinoma, enfortumab vedotin, cutaneous toxicity, steroid premedication.



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## Introduction

Enfortumab vedotin (EV) is an antibody-drug conjugate (ADC) consisting of a human monoclonal antibody targeting nectin-4, linked to the chemotherapeutic payload monomethyl auristatin E (1).

In Japan, EV was approved in November 2021 as a third-line therapy for patients with advanced urothelial carcinoma (UC) that is refractory to prior platinum-based chemotherapy and immune checkpoint inhibitors (ICIs), such as PD-1/programmed death ligand 1 (PD-1/L1) inhibitor (2), based on the results of a randomized controlled phase III pivotal EV-301 trial (3).

A companion diagnostic is not required to administer EV to patients with advanced UC, as nectin-4 has been shown to be highly expressed in both lower and upper urinary tract cancers (4). Conversely, nectin-4 is normally expressed in human epidermal keratinocytes and skin appendages (*e.g.*, sweat glands and hair follicles) (1). Therefore, cutaneous toxicities are expected to occur as natural potential toxicities, and their incidence is reported to be higher than that of other EV-related AEs in the EV-301 trial (3).

The incidence of cutaneous toxicity has been reported in the early phase of EV treatment (5), and corticosteroids are recommended for the management of dermatologic events (6); however, even during steroid treatment, failure to successfully manage cutaneous AEs may result in EV dose interruption, dose reduction, or treatment withdrawal.

EV are classified as having low emetic risk in the National Comprehensive Cancer Network (NCCN) guidelines, and dexamethasone 8-12 mg per os (PO)/ intravenous (IV) once is also recommended as a supportive care drug to start before anticancer therapy in cases with a low emetic risk (7). However, whether steroid premedication affects the clinical outcomes of EV monotherapy and improves the incidence and severity of EV-related cutaneous toxicities remains unclear.

Therefore, we retrospectively evaluated the association between steroid premedication, clinical outcomes, and EV-

related cutaneous toxicity in patients with advanced UC who received EV after being refractory to prior platinum-based chemotherapy and ICIs.

## Patients and Methods

*Patients.* A total of 29 consecutive patients who received EV monotherapy for advanced UC (metastatic or locally advanced) that had progressed radiologically with platinum-based chemotherapy and ICIs were enrolled from December 2021 to November 2024. One patient was excluded due to steroid use in the present study, resulting in a final analysis set of 28 patients.

EV was administered at a dose of 1.25 mg/kg on days 1, 8, and 15 at 28-day intervals until disease progression or unacceptable AEs occurred. Dose modifications were performed by physicians to manage AEs according to the patient's condition. Based on steroid premedication, dexamethasone 6.6 mg before EV was administered intravenously by each physician.

Chest-abdominal-pelvic computed tomography scans were generally performed for tumor measurements at baseline and after every one-three cycles of EV, or when deemed clinically necessary. The Response Evaluation Criteria in Solid Tumors, version 1.1 was used to assess treatment responses (8). The Common Terminology Criteria for Adverse Events (version 5.0) were used for the assessment of EV-related cutaneous toxicity grades (9). This study was approved by the Institutional Review Board of the National Hospital Organization Kyushu Cancer Center (2014-99). Informed consent was obtained through an opt-out process owing to the retrospective nature of the study.

*Statistical analyses.* EZR ver.1.40 (Easy R, Saitama Medical Center, Jichi Medical University, Saitama, Japan), a graphical user interface for R (The R Foundation for Statistical Computing, Vienna, Austria), was used for all statistical analyses (10). The overall response rate (ORR) was defined as the proportion of patients with partial response (PR) or complete response (CR) to EV, and the

disease control rate (DCR) was a composite of ORR and stable disease (SD). Fisher's exact probability test was used to compare categorical variables.

Progression-free survival (PFS) was analyzed using the Kaplan-Meier method and defined as the time from the first day of EV exposure to radiographic progression or death, whichever occurred first. For patients without adverse events, follow-up was censored on the date of the last follow-up examination. The log-rank test was used to analyze the differences in PFS between the groups with and without steroid premedication. All statistical analyses were 2-sided, with statistical significance set at  $p < 0.05$ .

## Results

*Patient characteristics (Table I).* Clinical characteristics of the 28 patients are shown in Table I. The median follow-up period after receiving EV was 7.1 [interquartile range (IQR)=3.7-12.7] months. In this cohort, the median age was 71 years (IQR=67-77 years), and 16 patients were male (57.1%). Twenty-two patients had an ECOG performance status (PS) of 0 or 1 (78.6%). Regarding the primary tumor site, 15 patients (53.6%) had tumors located in the lower urinary tract and 13 (46.4%) had a tumor located in the upper urinary tract. A total of 20 patients had pure UC according to the histological analysis (71.4%). Regarding prior ICIs, 15 patients had received anti-PD-1 (53.6%) and others had received anti-PD-L1. In addition, ORR was confirmed in four patients (14.3%) and DCR in 11 patients (39.3%) according to their response to ICIs. The number of regimens administered prior to EV was 2 in 24 patients (85.7%). Eighteen patients (64.3%) had visceral metastases when EV were initiated. Eighteen (64.3%) patients died during the follow-up period.

*Efficacy.* The ORR and DCR in patients treated with EV were 39.3% and 67.9%, respectively (best response: CR [n=0], PR [n=11], SD [n=8], and progressive disease [n=9]). Overall, steroid premedication with dexamethasone 6.6 mg was administered intravenously prior to EV in six (21.4%)

Table I. *Patients' characteristics.*

Characteristic (n=28)	
Age (years), median (IQR)	71 (67-77)
Male sex, n (%)	16 (57.1)
ECOG PS score, n (%)	
≤1	22 (78.6)
≥2	6 (21.4)
Primary tumor site, n (%)	
Lower urinary tract	15 (53.6)
Upper urinary tract	13 (46.4)
Pure UC in histologic testing, n (%)	20 (71.4)
Prior immune checkpoint inhibitors, n (%)	
Anti-PD-1 antibody	15 (53.6)
Anti-PD-L1 antibody	13 (46.4)
Response to immune checkpoint inhibitors, n (%)	
ORR (CR+PR)	4 (14.3)
DCR (without PD)	11 (39.3)
Number of regimens prior to EV	
2	24 (85.7)
≥3	4 (14.3)
Visceral metastases, n (%)	18 (64.3)

IQR: Interquartile range; ECOG PS: Eastern Cooperative Oncology Group performance status; UC: urothelial carcinoma; PD-1: programmed cell death 1; PD-L1: programmed cell death ligand 1; ORR: objective response rate; CR: complete response; PR: partial response; DCR: disease control rate; PD: progressive disease; EV: enfortumab vedotin.

patients. ORR was confirmed in three patients (50.0%) with steroid premedication and in eight patients (36.4%) without steroid premedication. DCR was confirmed in four patients (66.7%) with steroid premedication and in 15 patients (68.2%) without steroid premedication. There were no marked differences in the ORR and DCR between patients with and without steroid premedication ( $p=0.653$  and  $p>0.99$ , respectively) (Figure 1 and Figure 2).

The median PFS values in patients treated with and without steroid premedication were not estimable (NE) [95% confidence interval (CI)=1.4-NE] months and 4.3 (95%CI=2.6-6.4) months, respectively. The log-rank test revealed no significant difference in PFS between patients with and without steroid premedication ( $p=0.501$ ) (Figure 3).

*Safety.* Overall, EV-related cutaneous AEs occurred in 2 (33.3%) patients receiving steroid premedication and 10

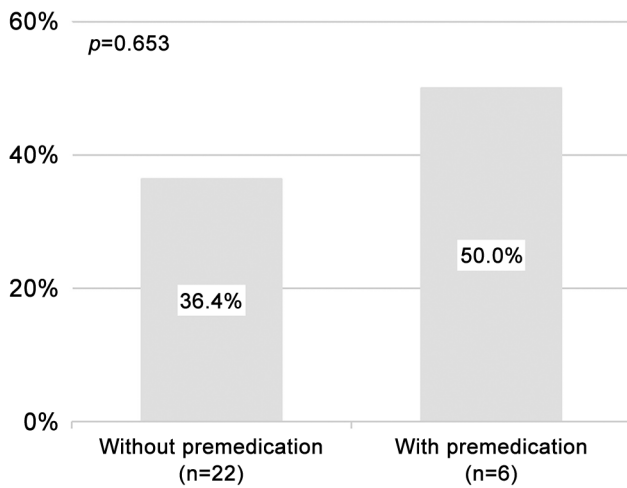


Figure 1. Objective response rates with and without steroid premedication in patients treated with enfortumab vedotin.

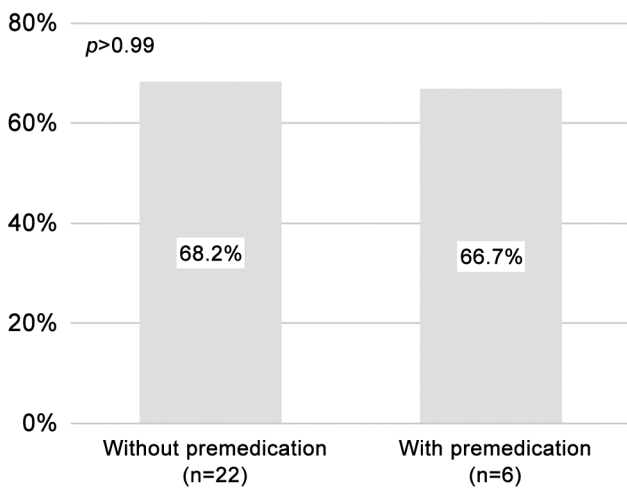
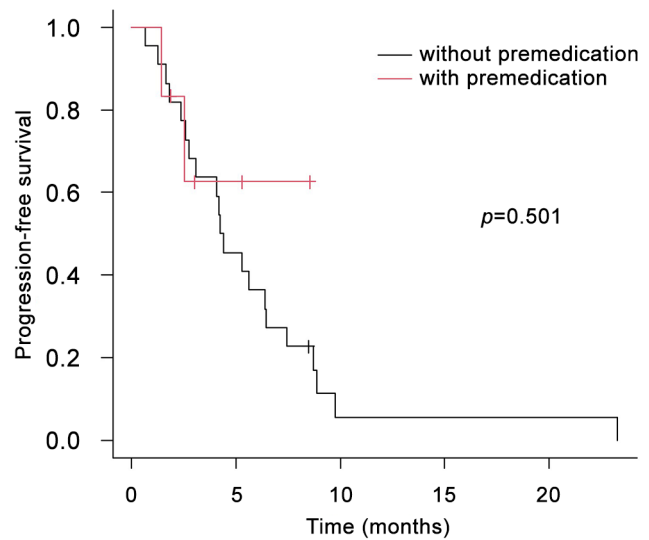


Figure 2. Disease control rates with and without steroid premedication in patients treated with enfortumab vedotin.

(45.5%) patients without steroid premedication ( $p=0.673$ ) (Figure 4). There was no significant difference in the incidence of grade  $\geq 3$  EV-related cutaneous AEs between the patients with and without steroid premedication (16.7% vs. 36.4%,  $p=0.630$ ) (Figure 5).

*Univariate and multivariate analyses for the PFS.* Univariate Cox regression analysis revealed that  $PS \geq 2$  was



Number at risk		0	5	10	15	20
Without	22	10	1	1	1	1
With	6	2	0	0	0	0

Figure 3. Progression-free survival rates with and without steroid premedication in patients treated with enfortumab vedotin.

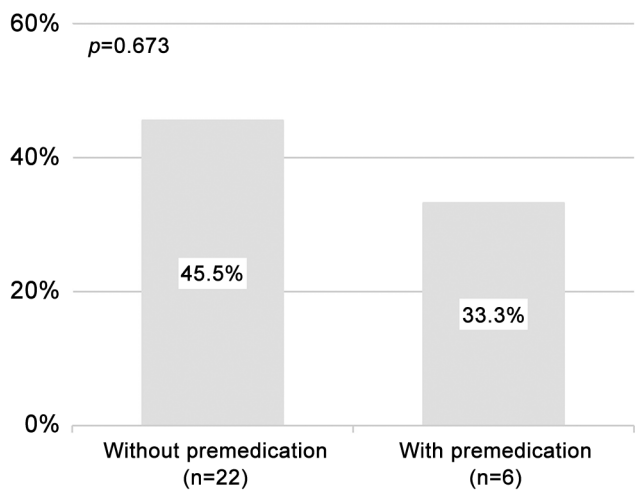


Figure 4. Incidence of enfortumab vedotin-related cutaneous toxicity (all grade) according to steroid premedication (with and without).

significantly associated with prognosis (HR=3.126, 95%CI=1.153-8.478,  $p=0.025$ ), although other variables, including steroid premedication, were not associated with prognosis. Multivariate analyses revealed that  $PS \geq 2$  was independently associated with prognosis (HR=4.653, 95%CI=1.263-17.140,  $p=0.021$ ) (Table II).

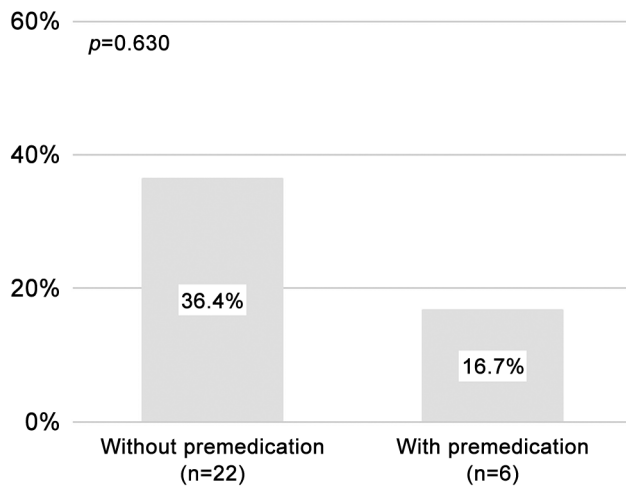


Figure 5. Incidence of enfortumab vedotin-related cutaneous toxicity (grade  $\geq 3$ ) according to steroid premedication (with and without).

## Discussion

This retrospective study was designed to evaluate whether steroid premedication prior to EV affects efficacy, and whether steroid premedication prior to EV improves the incidence and severity of cutaneous toxicity in patients with advanced UC treated with EV. The present study confirmed that the efficacy of steroid premedication prior to EV was not associated with a significantly lower ORR or DCR, or a shorter PFS than in patients without steroid premedication prior to EV. No significant improvement in the incidence or severity of cutaneous toxicity was observed with steroid premedication, although the incidence and severity of cutaneous toxicity showed some improvement.

EV showed an impressive result, with an overall survival (OS) benefit of approximately four months in comparison to chemotherapy (HR=0.70, 95%CI=0.56-0.89;  $p=0.00142$ ), despite being a third-line treatment for advanced UC in the EV-301 trial (3). Furthermore, a subgroup analysis of OS reported HRs of less than 1 in cases with liver metastases, cases with primary sites in the upper urinary tract, and cases that did not respond to pretreatment with ICI, which are traditionally considered difficult to treat (11).

However, treatment-related toxicities occur early during EV treatment. Specifically, skin reactions have been reported to have a median (range) time to onset of 0.43 (0.03-12.68) months (3). Therefore, failure to successfully control AEs that occur early in the course of treatment with EV may result in dose interruption, dose reduction, or treatment discontinuation, thereby preventing the full benefit of EV. We previously reported that clinical outcomes, such as ORR, DCR, PFS, and OS, were not significantly associated with EV-related cutaneous toxicities of any grade or grade  $\geq 3$ . However, PFS and OS tended to be worse in patients with EV-related grade  $\geq 3$  cutaneous toxicity (with vs. without: PFS 2.7 vs. 5.6 months,  $p=0.053$ ; OS 4.6 vs. 11.4 months,  $p=0.168$ ), suggesting that severe cutaneous toxicities may lead to dose interruption of EV for the management of AEs (12).

The management of AEs with EV is basically symptomatic treatment, along with reducing the dose of EV and extending the dosing interval; however, for skin disorders, corticosteroid treatment is indicated based on expert clinical recommendations (6). Among the treatment-emergent AEs in EV, nausea of any grade was reported in 22.6% of patients in the EV-301 trial (3). The NCCN classifies EV as having a low emetic risk (10%-30% frequency of emesis) with regard to the emetogenic potential of parenteral anticancer agents. For agents with low emetic risk, dexamethasone 8-12 mg PO/IV once, metoclopramide 10-20 mg PO/IV once, prochlorperazine 10 mg PO/IV once, or 5-hydroxytryptamine-3 receptor antagonists are recommended before anticancer therapy with daily repetition for multiday doses of anticancer therapy, although there is no clear evidence of acute-phase nausea and vomiting with low-emetic-risk anticancer agents (7). Therefore, the NCCN guidelines also state that clinicians should avoid overuse of antiemetics, especially in settings where the anticancer agents are of minimal or low emetic risk, in order to avoid exposing patients to AEs from antiemetics, to decrease possible drug-drug interactions, and to prevent unnecessary expenditures.

Whether steroid premedication affects the clinical outcomes of EV monotherapy and improves the incidence

Table II. Univariate and multivariate analyses for the progression-free survival in patients treated with enfortumab vedotin.

Variable	Univariate		Multivariate	
	HR (95%CI)	p-Value	HR (95%CI)	p-Value
Age	0.986 (0.933-1.043)	0.631	0.966 (0.903-1.034)	0.321
Sex	1		1	
	1.054 (0.422-2.631)	0.911	1.386 (0.524-3.667)	0.511
ECOG PS	1		1	
	3.126 (1.153-8.478)	0.025	4.653 (1.263-17.140)	0.021
Histologic testing	1		1	
	2.301 (0.845-6.266)	0.103	1.748 (0.595-5.139)	0.310
Visceral metastases	1		1	
	0.743 (0.316-1.751)	0.498	0.530 (0.193-1.456)	0.219
Steroid premedication	1		1	
	0.607 (0.139-2.641)	0.505	0.876 (0.181-4.241)	0.869

ECOG PS: Eastern Cooperative Oncology Group Performance Status; UC: urothelial carcinoma; HR: hazard ratio; CI: confidence interval.

and severity of EV-related cutaneous toxicities remains unclear. Dexamethasone, owing to its broad immunosuppressive effects, can theoretically blunt the effects of ICIs. Regarding the relationship between the clinical outcomes of ICIs and steroid administration, it has been reported that in patients treated with PD-(L)1 blockade monotherapy for advanced non-small cell lung cancer (NSCLC) who were receiving daily corticosteroids ( $\geq 10$  mg prednisone equivalent) at baseline, baseline corticosteroid treatment was associated with a decreased ORR, PFS and OS. In a multivariable analysis of the pooled population adjusted for smoking history, performance status, and history of brain metastases, baseline corticosteroids remained significantly associated with decreased PFS (HR=1.3;  $p=0.03$ ) and OS (HR=1.7;  $p<0.001$ ) (13). In contrast, it was also reported that steroid administration due to immune-related AEs did not affect OS ( $p=0.38$ ) compared with the steroid-naïve group in patients with advanced NSCLC treated with ICIs. Only steroid administration for palliation of cancer-related symptoms was an independent predictor of shorter OS (HR=2.7; 95%CI=1.5-4.9) (14).

According to the relationship between the clinical outcomes of chemotherapy and steroid administration, dexamethasone used for supportive care has no clinically significant impact on the pathological complete response, recurrence-free survival, or OS when administered

concurrently with neoadjuvant chemotherapy in patients with curative triple-negative breast cancer (15). This report is supported by the results of the present study, which found that steroid premedication, dexamethasone 6.6 mg IV before EV, did not affect the clinical outcome (ORR, DCR, and PFS) and that steroid premedication was not an independent variable associated with PFS according to multivariate analyses. Overall, this suggests that dexamethasone in chemotherapy is a safe option for symptom control, given its apparent lack of impact on clinically relevant survival outcomes.

Given the expression of nectin-4 in epidermal keratinocytes and skin appendages, skin disorders are anticipated following the administration of EV. These disorders can include rare but serious and potentially fatal cutaneous adverse events, such as Stevens-Johnson syndrome and toxic epidermal necrolysis. However, to date, no drug has been reported to be effective in the prevention and reduction of EV-related cutaneous toxicity, although regular application of topical emollients, moisturizers, or barrier-protecting agents may provide effective prophylaxis (16).

When cutaneous adverse events occur in patients treated with EVs, expert clinical recommendations suggest treating mild to moderate cutaneous manifestations with moderate-potency topical corticosteroids in combination

with topical antibiotics at intertriginous sites. For severe (grade 3) cutaneous manifestations, referral to a dermatology specialist should be considered, and the use of corticosteroids is recommended. For grade 4 or recurrent grade 3 dermatologic events or confirmed Stevens-Johnson syndrome and toxic epidermal necrosis, treatment should be permanently discontinued, and admission to the intensive care or burn unit may be warranted (6). Considering the results of the present study, premedication with steroids used as treatment at the onset of cutaneous damage may have the potential to improve the incidence and severity of EV-related cutaneous toxicity, although no significant differences were observed compared with patients without steroid premedication.

*Study limitations.* As this was a retrospective study conducted at a single institution, the sample size is small to have adequate statistical power to draw firm conclusions about the effect on cutaneous toxicities. However, despite these limitations, this study showed that the efficacy of steroid premedication was similar to that without steroid premedication, and that the incidence and severity of cutaneous AEs could potentially be improved by steroid premedication in EV therapy.

## Conclusion

In EV treatment, steroid premedication did not affect clinical outcomes. The incidence and severity of EV-related cutaneous toxicity tended to improve in patients who received steroid premedication, although no significant differences were observed.

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No funding was received.

## Conflicts of Interest

The Authors declare that they have no conflicts of interest in relation to this study.

## Authors' Contributions

Study concept and design: N.F., M.N. and T.N.; acquisition of data: N.F., M.M., A.K., Y.F., M.N. and N.T.; statistical analysis: N.F., and T.N.; analysis and interpretation of data: all Authors; drafting of the original manuscript: N.F., M.N. and T.N.; critical revision of the manuscript for important intellectual content: all Authors; supervision: M.N. and T.N. All Authors have read and approved the final version of the manuscript.

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## References

- 1 Challita-Eid PM, Satpayev D, Yang P, An Z, Morrison K, Shostak Y, Raitano A, Nadell R, Liu W, Lortie DR, Capo L, Verlinsky A, Leavitt M, Malik F, Aviña H, Guevara CI, Dinh N, Karki S, Anand BS, Pereira DS, Joseph IB, Doñate F, Morrison K, Stover DR: Enfortumab vedotin antibody-drug conjugate targeting nectin-4 is a highly potent therapeutic agent in multiple preclinical cancer models. *Cancer Res* 76(10): 3003-3013, 2016. DOI: 10.1158/0008-5472.CAN-15-1313
- 2 Minato A, Kimuro R, Ohno D, Tanigawa K, Kuretake K, Matsukawa T, Takaba T, Jojima K, Harada M, Higashijima K, Nagata Y, Tomisaki I, Harada K, Fujimoto N, Miyamoto H: Efficacy and tolerability of enfortumab vedotin for metastatic urothelial carcinoma: early experience in the real world. *Anticancer Res* 43(9): 4055-4060, 2023. DOI: 10.21873/anticancer.16594
- 3 Powles T, Rosenberg JE, Sonpavde GP, Loriot Y, Durán I, Lee JL, Matsubara N, Vulsteke C, Castellano D, Wu C, Campbell M, Matsangou M, Petrylak DP: Enfortumab vedotin in previously treated advanced urothelial carcinoma. *N Engl J Med* 384(12): 1125-1135, 2021. DOI: 10.1056/NEJMoa2035807
- 4 Tomiyama E, Fujita K, Hashimoto M, Adomi S, Kawashima A, Minami T, Yoshimura K, Uemura H, Nonomura N: Comparison of molecular profiles of upper tract urothelial carcinoma vs. urinary bladder cancer in the era of targeted therapy: a narrative review. *Transl Androl Urol* 11(12): 1747-1761, 2022. DOI: 10.21037/tau-22-457
- 5 Rosenberg JE, O'Donnell PH, Balar AV, McGregor BA, Heath EI, Yu EY, Galsky MD, Hahn NM, Gartner EM, Pinelli JM, Liang SY, Melhem-Bertrandt A, Petrylak DP: Pivotal trial of

- enfortumab vedotin in urothelial carcinoma after platinum and anti-programmed death 1/programmed death ligand 1 therapy. *J Clin Oncol* 37(29): 2592-2600, 2019. DOI: 10.1200/JCO.19.01140
- 6 Lacouture ME, Patel AB, Rosenberg JE, O'Donnell PH: Management of dermatologic events associated with the nectin-4-directed antibody-drug conjugate enfortumab vedotin. *Oncologist* 27(3): e223-e232, 2022. DOI: 10.1093/oncolo/oyac001
  - 7 NCCN guidelines. Antiemesis. Available at: <https://www.nccn.org/guidelines/guidelines-detail?category=3&id=1415> [Last accessed on May 1, 2024]
  - 8 Eisenhauer EA, Therasse P, Bogaerts J, Schwartz LH, Sargent D, Ford R, Dancey J, Arbuck S, Gwyther S, Mooney M, Rubinstein L, Shankar L, Dodd L, Kaplan R, Lacombe D, Verweij J: New response evaluation criteria in solid tumours: Revised RECIST guideline (version 1.1). *Eur J Cancer* 45(2): 228-247, 2009. DOI: 10.1016/j.ejca.2008.10.026
  - 9 US Department of Health and Human Services. NIOH, National Cancer Institute. Common Terminology Criteria for Adverse Events (CTCAE) version 5.0, 2017. Available at: [https://ctep.cancer.gov/protocolDevelopment/electronic\\_applications/docs/CTCAE\\_v5\\_Quick\\_Reference\\_8.5x11.pdf](https://ctep.cancer.gov/protocolDevelopment/electronic_applications/docs/CTCAE_v5_Quick_Reference_8.5x11.pdf) [Last accessed on May 1, 2024]
  - 10 Kanda Y: Investigation of the freely available easy-to-use software 'EZ' for medical statistics. *Bone Marrow Transplant* 48(3): 452-458, 2013. DOI: 10.1038/bmt.2012.244
  - 11 Rosenberg JE, Powles T, Sonpavde GP, Loriot Y, Duran I, Lee JL, Matsubara N, Vulsteke C, Castellano D, Mamtani R, Wu C, Matsangou M, Campbell M, Petrylak DP: EV-301 long-term outcomes: 24-month findings from the phase III trial of enfortumab vedotin *versus* chemotherapy in patients with previously treated advanced urothelial carcinoma. *Ann Oncol* 34(11): 1047-1054, 2023. DOI: 10.1016/j.annonc.2023.08.016
  - 12 Furubayashi N, Minato A, Tomoda T, Masaoka H, Hori Y, Kiyoshima K, Negishi T, Haraguchi Y, Koga T, Song Y, Harada K, Kuroiwa K, Seki N, Fujimoto N, Nakamura M, Uro-Oncology Group in Kyushu (UROKYU): Cutaneous and renal toxicities of enfortumab vedotin for advanced urothelial carcinoma: the UROKYU study. *Anticancer Res* 44(7): 3025-3032, 2024. DOI: 10.21873/anticancer.17115
  - 13 Arbour KC, Mezquita L, Long N, Rizvi H, Auclin E, Ni A, Martínez-Bernal G, Ferrara R, Lai WV, Hendriks LEL, Sabari JK, Caramella C, Plodkowski AJ, Halpenny D, Chaft JE, Planchard D, Riely GJ, Besse B, Hellmann MD: Impact of baseline steroids on efficacy of programmed cell death-1 and programmed death-ligand 1 blockade in patients with non-small-cell lung cancer. *J Clin Oncol* 36(28): 2872-2878, 2018. DOI: 10.1200/JCO.2018.79.0006
  - 14 Skribek M, Rounis K, Afshar S, Grundberg O, Friesland S, Tsakonas G, Ekman S, De Petris L: Effect of corticosteroids on the outcome of patients with advanced non-small cell lung cancer treated with immune-checkpoint inhibitors. *Eur J Cancer* 145: 245-254, 2021. DOI: 10.1016/j.ejca.2020.12.012
  - 15 Johnson KCC, Goldstein D, Tharakan J, Quiroga D, Kassem M, Grimm M, Miah A, Vargo C, Berger M, Sudheendra P, Pariser A, Gatti-Mays ME, Williams N, Stover D, Sardesai S, Wesolowski R, Ramaswamy B, Tozbikian G, Schnell PM, Cherian MA: The immunomodulatory effects of dexamethasone on neoadjuvant chemotherapy for triple-negative breast cancer. *Oncol Ther* 11(3): 361-374, 2023. DOI: 10.1007/s40487-023-00235-6
  - 16 Brower B, McCoy A, Ahmad H, Eitman C, Bowman IA, Rembisz J, Milowsky MI: Managing potential adverse events during treatment with enfortumab vedotin+pembrolizumab in patients with advanced urothelial cancer. *Front Oncol* 14: 1326715, 2024. DOI: 10.3389/fonc.2024.1326715