Adverse events induced by the subcutaneous HER2-targeted antibody-drug conjugate JSKN033 in a male patient with breast cancer: A case report

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Abstract. JSKN033 is a fixed-dose combination of JSKN003 [a biparatopic HER2-directed antibody-drug conjugate (ADC)] and envafolimab (a programmed death-ligand 1 inhibitor). JSKN033 is formulated for subcutaneous administration and is currently in the investigational stage, with limited safety profile data. The present report highlights the case of a male patient with breast cancer with HER2 2+ expression who experienced disease progression after multiline therapy, including modified radical mastectomy, adjuvant chemotherapy, adjuvant radiotherapy and trastuzumab emtansine. Following enrollment in the JSKN033 clinical trial, the patient developed systemic and localized adverse reactions after treatment, including fatigue, diarrhea and injection-site reactions, such as pruritus, pain, skin indentation and induration. The present case report discusses potential etiologies of these adverse events and issues to consider during subcutaneous administration of HER2-directed ADCs, and provides supplementary clinical data to potentially advance the understanding of their adverse event profiles, mechanisms, prevention and management strategies.

Introduction

Male breast cancer (MBC) is a rare malignant tumor that accounts for ~1% of all breast cancer cases (1). Globally, the incidence of MBC is relatively low, with GLOBOCAN 2020 data reporting an annual rate of 0.5-1.0 cases per 100,000 individuals (2). The mortality rate of MBC in 2021 was 0.34 cases per 100,000 individuals [95% uncertainty interval (UI), 0.23-0.41], which was markedly lower compared with the rate of female breast cancer (FBC) at 14.55 cases per 100,000 individuals (95% UI, 13.45-15.56) (3). Due to its rarity, MBC is underrepresented in breast cancer trials, resulting in a lack of prospective or randomized data specific to men. Therefore, treatment decisions are typically extrapolated from data derived from female patients (4), whose adverse event (AE) profiles often lack characteristics specific to men. Therefore, notable emphasis should be placed on including male patients in clinical trials of breast cancer and reporting AE data.

Primary treatment modalities for MBC include surgery, radiotherapy, chemotherapy, endocrine therapy, targeted therapy and immunotherapy (IO) (5,6). Mastectomy has traditionally been considered the standard surgical approach for early-stage MBC due to limited breast tissue in men and the typical proximity of tumors to the nipple-areolar complex (5). Since the majority of MBCs are hormone receptor-positive, adjuvant endocrine therapy, primarily tamoxifen, constitutes the cornerstone of treatment for hormone receptor-positive disease. Radiotherapy is considered to provide clinically notable benefits for male patients with early-stage and locally advanced disease. Furthermore, the management of advanced MBC typically aligns with established approaches used for female patients, including chemotherapy, HER2-targeted agents, IO and poly(ADP ribose) polymerase inhibitors (5,6).

HER2 is expressed in tumor tissues and cells of various advanced malignant solid tumors, such as breast (7), gastric (8), pancreatic (9), lung (10), colorectal (11) and ovarian (12) cancer. In breast cancer, the incidence of HER2 gene amplification and upregulation can reach 15-20% (13). The HER2 receptor is recognized as an effective therapeutic target for tumors with HER2 amplification or upregulation. Antibody-drug conjugates (ADCs) combine the high specificity of monoclonal antibodies with the potent antitumor activity of cytotoxic payloads. The targeted delivery mechanism of ADCs enhances safety profiles, making them a prominent research focus in oncology therapeutics (14). Although several HER2-directed ADCs, such as trastuzumab emtansine (T-DM1) (15), trastuzumab deruxtecan (16) and disitamab vedotin (17), have been approved for clinical use, subcutaneous (SC) formulations of HER2-directed ADCs remain in the emerging phase of

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clinical investigation, with limited reported data on associated AEs.

In January 2025, the Phase I Unit of Fudan University Shanghai Cancer Center (Shanghai, China) initiated a phase I/II clinical trial evaluating the safety, tolerability, pharmacokinetics/pharmacodynamics and antitumor activity of JSKN033 (18). JSKN033 is a fixed-dose combination for SC injection that comprises JSKN003, a biparatopic HER2-directed ADC, and envafolimab, a programmed death-ligand 1 (PD-L1) inhibitor approved by the National Medical Products Administration of China (18). In the dose-escalation phase, patients received SC JSKN033 across three doses (5.6, 6.7 and 8.4 mg/kg, once weekly) following a modified 3+3 design. Safety evaluations were based on treatment-related AEs (TRAEs) and dose-limiting toxicities to assess the primary endpoints of safety and tolerability. The dose-limiting toxicity assessment period was 21 days. Investigators evaluated tumor response every 6 weeks using the Response Evaluation Criteria in Solid Tumors (version 1.1; RECIST) (19) and performed tumor imaging assessments with computed tomography (CT) or magnetic resonance imaging (MRI). Preliminary study data indicated that the most common TRAE of JSKN033 was mild to moderate (grade 1 and 2) injection site reactions (90.9%). This was followed by diarrhea (54.5%), nausea (45.5%), increased aspartate aminotransferase (27.3%), decreased appetite (27.3%), increased alanine aminotransferase (18.2%) and maculo-papular rash (18.2%). No grade 3 or higher TRAEs or serious AEs were observed and no TRAEs led to treatment discontinuation (18). During the same month of JSKN033 initiation, a male patient with breast cancer was enrolled and subsequently developed evident systemic and injection-site adverse reactions after treatment, including fatigue, diarrhea and localized injection-site manifestations of pruritus, pain, cutaneous depression and SC induration.

The development of SC formulations for anticancer agents has gained increasing attention in oncological therapeutics due to their notable convenience and improved treatment experience. Notable examples include subcutaneous formulations of the monoclonal antibodies trastuzumab (20), the fixed-dose combination of pertuzumab and trastuzumab (21), and the PD-L1 inhibitor envafolimab (22). JSKN033 is the first global SC coformulation consisting of an ADC and an immune checkpoint inhibitor, and to the best of our knowledge, research on the underlying mechanisms of JSKN033-associated AEs and their clinical management is limited (18,23). The present case report focuses on JSKN033-associated AEs, which may contribute to the understanding of the toxicity profiles of SC-administered ADCs and the optimization of management strategies. Furthermore, as the present case includes a male patient with breast cancer, it may provide distinctive data and insights that are potentially valuable for future research.

Case report

A 38-year-old man underwent a right-sided simple mastectomy at Changzhou Wujin People's Hospital (Changzhou, China) in March 2023 after self-identifying a 7x7-cm mass in the right breast. Postoperative pathology [based on histopathological slides from Changzhou Wujin People's Hospital and subsequent consultation with the Department of Pathology of Fudan University Shanghai Cancer Center (Shanghai, China)] revealed grade 3 invasive ductal carcinoma according to the Nottingham histological grading system (24) [estrogen receptor (ER), 90%; progesterone receptor, 90%; HER2, 2+; and Ki-67, 40%). The immunohistochemistry and H&E images are not available as the original histopathological slides were processed and diagnosed by Changzhou Wujin People's Hospital (data not shown). In April 2023, the patient received a modified radical mastectomy followed by adjuvant chemotherapy consisting of 4 cycles of cyclophosphamide [1.5 g intravenously (IV) every 3 weeks] plus epirubicin (180 mg IV every 3 weeks) and 4 cycles of trastuzumab (1,120 mg IV every 3 weeks), pertuzumab (840 mg IV every 3 weeks) and docetaxel (202.4 mg IV every 3 weeks). The patient subsequently received adjuvant radiotherapy in November 2023 (total dose of 4,256 cGy in 16 fractions at 266 cGy/fraction), followed by 2 cycles of T-DM1 (500 mg IV every 3 weeks), which was discontinued due to disease progression. In December 2023, positron emission tomography-CT revealed disease recurrence in the chest wall with multiple pulmonary and lymph node metastases (Fig. 1). The patient was subsequently enrolled in two Phase 1 clinical trials [SMP-656 (CTR20233290) and ASKG-915 (CTR20232767)] from January to June 2024 but was withdrawn from both trials due to progressive disease.

The patient was enrolled in the JSKN033 phase I/II clinical trial (CTR20244896) at the Phase I Clinical Trial Center of Fudan University Shanghai Cancer Center in January 2025. The patient had a medical history of type 2 diabetes mellitus and hypertension, with concomitant medications, including metformin (0.5 g twice daily, orally), protamine human insulin mixed injection (30 IU once daily, administered subcutaneously in the bilateral upper arms), amlodipine besylate (5 mg once daily, orally), valsartan (100 mg three times daily, orally) and arotinolol (10 mg three times daily, orally). No history of drug or disinfectant allergies was reported. Physical examination revealed no ecchymoses, erythema or SC nodules on the skin or mucous membranes. Baseline target lesion selection was performed in January 2025, identifying a left pulmonary nodule as the target lesion, with a sum of the longest diameters measuring 17.3 mm. JSKN033 [180 mg (1 ml)/vial] consisted of 80 mg of JSKN003 and 100 mg of envafolimab. The JSKN033 treatment regimen included weekly SC administration. The dose administered to the patient was calculated as 560 mg (3.1 ml) per administration on the basis of body weight, which was divided into two SC injections (2+1.1 ml) due to protocol recommendations (maximum 2 ml per injection) (25,26), which meant that the patient received two SC injections per weekly dose. The first dose of JSKN033 was administered in January 2025. As of April 2025, the patient had received 13 weekly doses, totaling 26 SC injections over the 13-week period. On the basis of the protocol recommendation that the optimal injection sites are the abdomen and thighs (25,26), the injection sites were selected in the following order: i) Left/right mid-abdomen, ii) left/right upper abdomen, iii) left/right lower abdomen, iv) medial mid-to-lower left/right thigh; and v) lateral mid-to-lower left/right thigh. New injection sites were selected for each administration and no single site was injected twice consecutively; when repeated injections in the same anatomical region were necessary due to practical constraints, subsequent

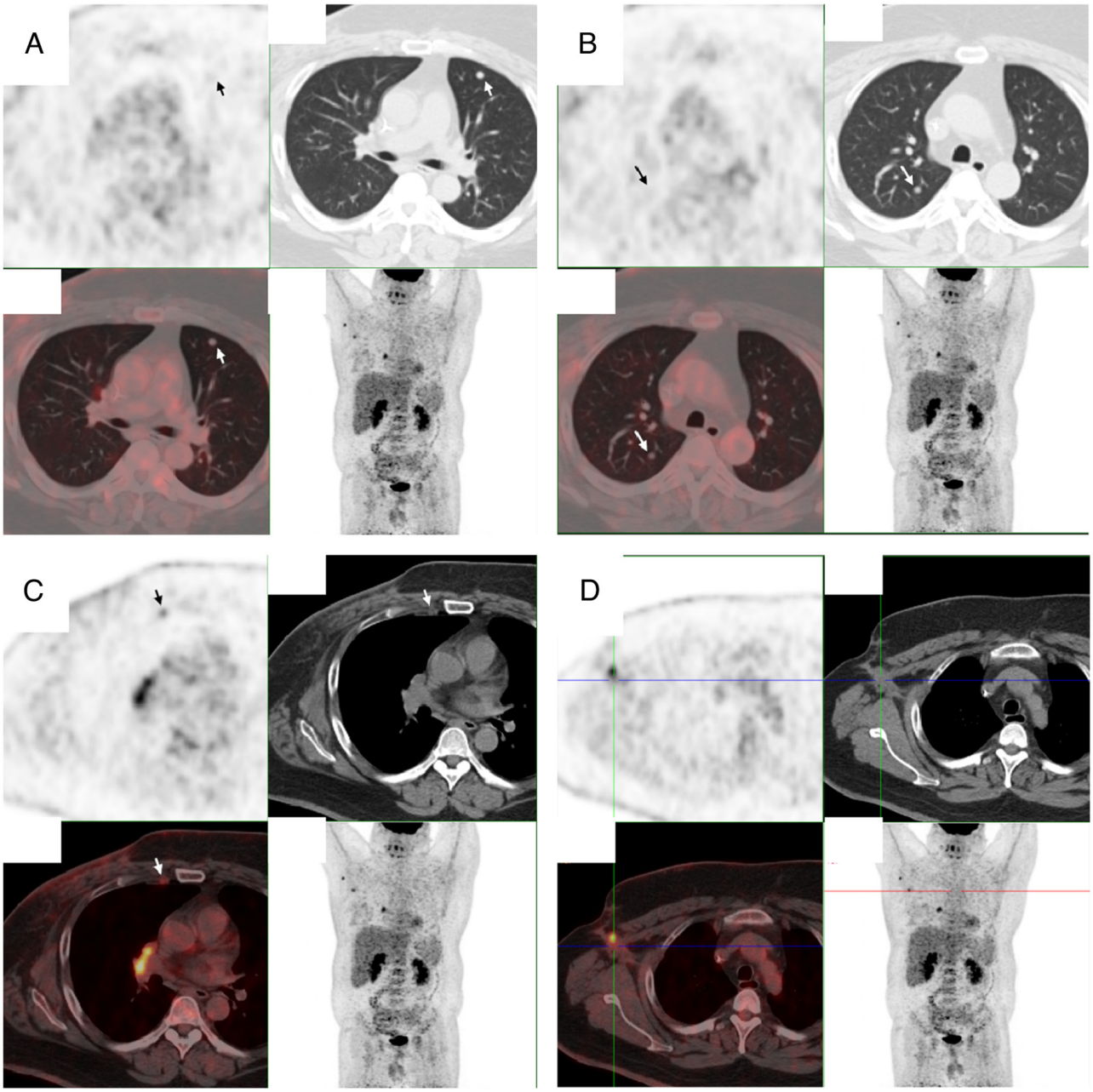

Figure 1. PET-CT scan (December 2023) revealing metastatic recurrence. (A and B) Multiple pulmonary metastases in both lungs. (C and D) Chest wall recurrence and axillary lymph node metastases. In panels (A-D), the top-left image is a PET scan, the top-right is a CT scan, the bottom-left is a fused PET-CT image and the bottom-right is a whole-body PET scan. PET, positron emission tomography; CT, computed tomography.

injections were administered ≥2.5 cm from previous sites. Please refer to Fig. 2 for a detailed illustration of the injection site rotation strategy. With the protocol recommended injection rate of ≤0.06 ml/sec, the minimum injection time of each administration was calculated as 52 sec. Postinjection observation was required for 1 h before discharge.

AEs were classified and graded according to the Common Terminology Criteria for Adverse Events (CTCAE) version 5.0 (27). The patient developed systemic reactions 2 weeks after administration (January 2025), presenting with fatigue (CTCAE grade 1) and diarrhea (watery stools, 4-5 episodes/day; CTCAE grade 2). The patient demonstrated no evidence of anemia or thyroid dysfunction at baseline or during treatment. Therefore, these factors were excluded as potential causes of fatigue. The diarrhea improved to 1-2 episodes/day (CTCAE grade 1) following antidiarrheal treatment with smectite (3 g twice daily, orally) and loperamide capsules (2 mg once daily, orally). Furthermore, 5 weeks after administration (February 2025), the patient exhibited an elevated high-sensitivity troponin T concentration of 0.042 ng/ml (normal range, 0-0.014 ng/ml). Supplementary echocardiography demonstrated a preserved left ventricular ejection fraction of 63% (normal range, 50-80%). No elevated liver enzymes or hematological toxicity was observed during the treatment course. The patient self-reported postinjection localized tension-type pain, quantified as 2 on a 0-10 numeric rating scale, where 0 indicates no pain and 10 represents the worst possible pain (28). Mild pruritus (CTCAE grade 1) at the

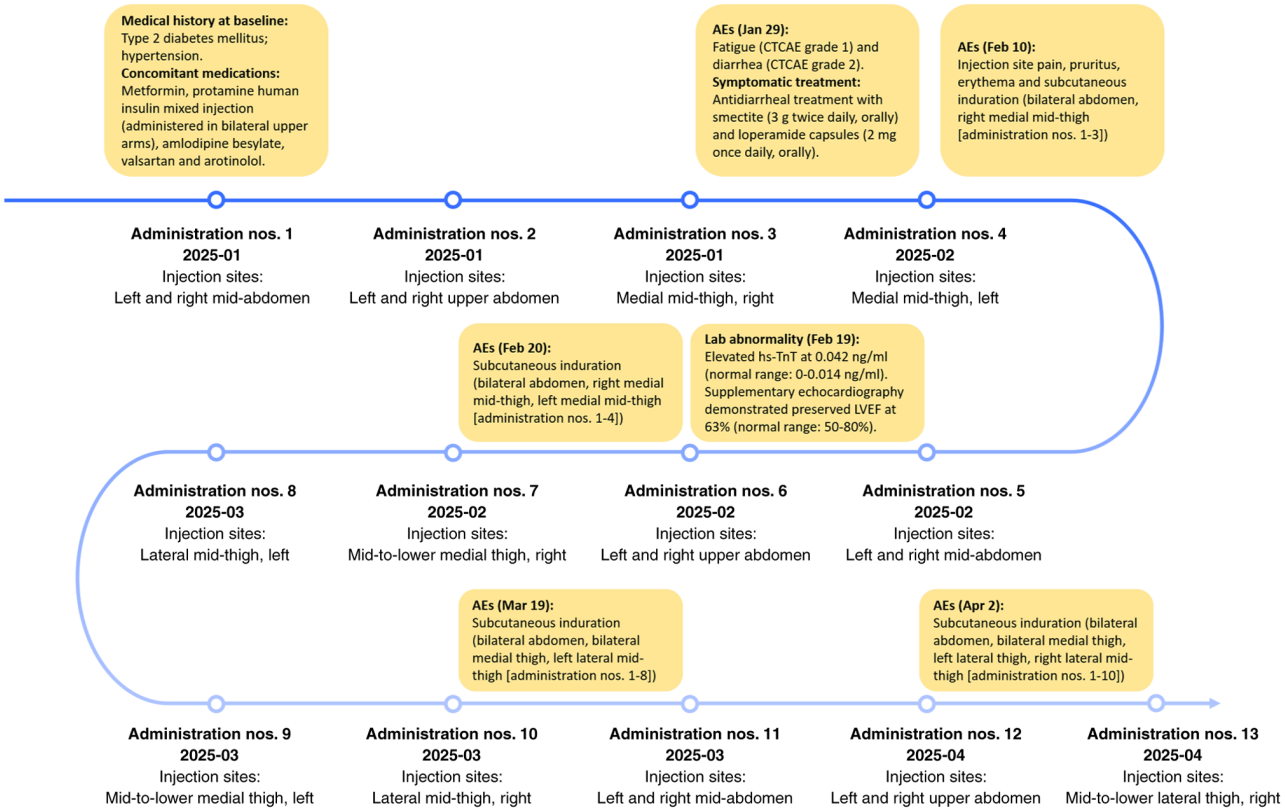

Figure 2. Timeline of treatment interventions and AEs. Each administration required two separate subcutaneous injections (2+1.1 ml), with injection sites within the same anatomical region spaced ≥2.5 cm apart (administration nos. 3, 4, 7, 8, 9, 10 and 13). CTCAE, Common Terminology Criteria for Adverse Events; AEs, adverse events; LVEF, left ventricular ejection fraction; hs-TnT, high-sensitivity troponin T.

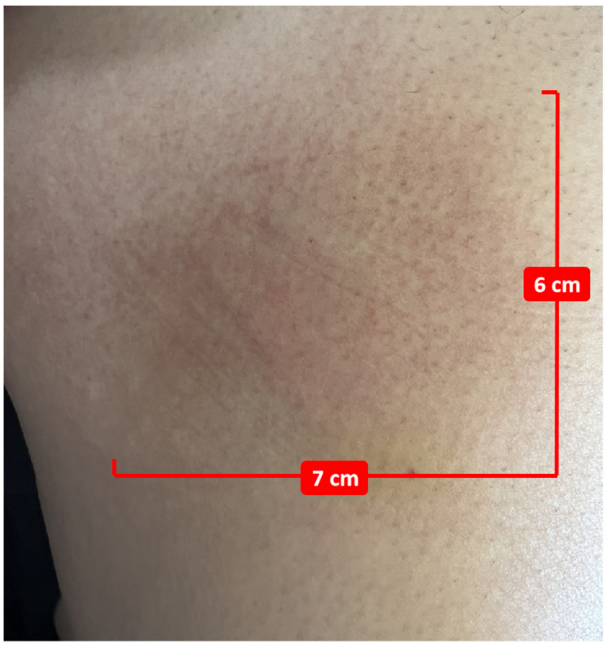

Figure 3. Erythema and hyperpigmentation following left medial thigh injection (image captured in March 2025). Appearance at 1 week after the ninth injection in March 2025, showing an area measuring ~7x6 cm.

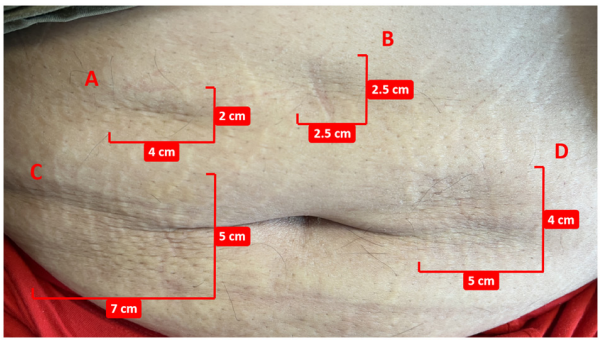

Figure 4. Subcutaneous indentation and induration following abdominal injection (image captured in March 2025). (A and B) Appearance at 8 weeks after the second injection in January 2025, showing an area measuring ~4x2 and 2.5x2.5 cm. (C and D) Appearance at 9 weeks after the first injection in January 2025, showing areas measuring ~7x5 and 5x4 cm. All cutaneous abnormalities presented occurred following single injections at each site.

injection site was noted and worsened with thermal stimulation. Furthermore, the patient experienced local erythema (Fig. 3) without swelling at ~1 week, which typically resolved within 2-3 weeks, resulting in post-inflammatory hyperpigmentation. Persistent cutaneous changes included injection-site depression (Fig. 4), enlarged pores, deepened skin folds and SC induration resistance to resolution. All cutaneous abnormalities presented in Figs. 3 and 4 occurred following single injections at each site. The overall treatment process and progression of AEs are shown in Fig. 2.

The patient was followed up weekly, and tumor assessments were performed using CT or MRI every 6 weeks (±7 days). At the 12-week follow-up (April 2025), the therapeutic response

was assessed as a partial response according to RECIST (version 1.1), with the target left pulmonary nodule demonstrating a sum of the longest diameters of 9.8 mm (baseline, 17.3 mm). All observed AEs were mild and clinically manageable. Treatment continuation was approved, with ongoing monitoring.

Discussion

JSKN033 is a fixed-dose combination of JSKN003 and envafolimab (18). JSKN003 is a biparatopic HER2-directed ADC composed of a recombinant humanized anti-HER2 bispecific antibody, a linker and a topoisomerase I inhibitor payload. The JSKN003 mechanism of action includes specific recognition and binding to HER2 on tumor cell surfaces, followed by HER2-mediated internalization, intracellular release of the topoisomerase I inhibitor payload, induction of DNA damage and subsequent apoptosis, thereby exerting antitumor effects (29). The combination of ADCs with IO may provide potential synergistic effects to enhance clinical benefits, representing a promising direction for future ADC development (30). The currently approved HER2-targeted ADCs (for example, T-DM1, disitamab vedotin and trastuzumab deruxtecan) (17,31) are all administered via intravenous (IV) infusion. Furthermore, both ADC-IO combinations and SC ADC formulations remain under clinical investigation (18,32).

Based on current reports of HER2-targeted ADCs, the commonly observed AEs include elevated liver enzymes, fatigue, decreased left ventricular ejection fraction, interstitial lung disease/non-infectious pneumonitis, loss of appetite, nausea, vomiting, diarrhea, hematological toxicity and infusion-associated reactions (14,33-35). The toxicity profile of ADCs is primarily associated with target selection and payload characteristics (33). In the present case, the systemic reactions of the patient manifested mainly as diarrhea and fatigue, which may be attributed to cross-reactivity of the HER2-targeted ADC with gastrointestinal HER2 receptors during tumor antigen binding (36) and the payload, a topoisomerase I inhibitor, which can induce early-onset and delayed-onset diarrhea (37). With respect to non-infectious etiologies, mild diarrhea typically responds to loperamide supplemented by fluid/electrolyte replacement when necessary (38). Overall, these systemic reactions remain mild and manageable through symptomatic treatment and lifestyle modifications. From both theoretical and clinical data, the systemic adverse reactions associated with SC administration are generally consistent with those associated with the IV route (in terms of classification rather than specific incidence rates) (14,18,33-35).

Compared with IV administration, SC delivery avoids infusion-associated reactions but may induce injection site reactions (ISRs). In accordance with the preliminary clinical data of JSKN033, 11 patients were enrolled in the dose-escalation phase in Australia, 10 of whom (90.9%) experienced ISRs following administration (18), all of which were mild in severity, with no reported cases requiring dose reduction or treatment discontinuation. To the best of our knowledge, current research on the mechanisms underlying ISRs associated with SC administration of HER2-targeted ADCs remains limited. Potential contributing factors to these local reactions may include the following: i) Drug-associated factors: The cytotoxic payloads of ADCs are likely the primary causes of ISRs (23). The slow absorption process and prolonged local retention of large-molecule ADCs can lead to sustained local drug exposure at the injection site. Furthermore, enhanced immune cell uptake of the antibody-conjugated payload may exacerbate immune system and skin inflammation (23). Furthermore, envafolimab in JSKN033 is the first approved global SC-administered PD-L1 inhibitor and is marketed in China. Current data indicates that envafolimab induces ISRs with no more than a 5% incidence rate, potentially associated with immune system activation and inflammatory responses, possibly contributing to the ISR presentation of the patient (39). ii) Injection technique factors: The stability of the linker markedly influences payload release, where premature release of the cytotoxic payload may result in off-target toxicity (33). Improper handling practices such as excessive heating or agitation before injection could compromise linkers, thereby impairing therapeutic efficacy and exacerbating AEs. Furthermore, injection parameters, including volume, injection rate, technique and repeated dosing at the same site, contribute to ISRs (40). iii) Patient-specific factors: The present patient had a baseline body weight of 120 kg, with a BMI >37 kg/m^2. Previous studies have indicated that pro-inflammatory immune cells and adipocytes secrete inflammatory cytokines (TNF-α, IL-6, IL-1β and leptin), creating a chronic low-grade inflammatory state in obesity that likely exacerbates injection-site inflammatory responses and induration formation (41-43). Furthermore, obesity affects skin barrier integrity, which gets translated into clinical xerosis. Obesity also causes altered collagen structure and impaired wound healing due to decreased mechanical strength (44). These factors may have collectively contributed to the development of ISRs in the present patient.

Furthermore, MBC is usually ER$^+$. A large multicenter retrospective cohort study demonstrated strong ER expression in >90% of MBC cases (45). The gene encoding ERα acts as a key regulator of several key hepatic drug-metabolizing enzymes, including cytochrome P450 families, exerting broad effects on drug metabolism (46). Therefore, the high ER-positive profile characteristic of MBC may also represent one of the factors influencing drug metabolism and the development of AEs. It has been reported that certain sex-based differences exist in the risk of AEs between male and female patients with cancer receiving IO, targeted therapy or chemotherapy (47). Women are at a notably greater risk of severe symptomatic AEs across multiple treatment domains, including patients receiving immune checkpoint inhibitor therapy and targeted therapies with kinase inhibitors (47). This disparity may be associated with factors such as differences in sex hormone levels affecting drug metabolism and sex variations in pharmacokinetics, pharmacogenomics and treatment adherence (47). JSKN033 is a combination of JSKN003 (a biparatopic HER2-directed ADC) and envafolimab (a PD-L1 inhibitor) (18); therefore, its treatment-associated AEs may also exhibit sex-specific variations. However, as JSKN033 is still in the clinical trial stage, robust statistical evidence regarding sex differences in its toxicity profile remains unavailable. Future research on sex-specific differences in JSKN033-associated AEs would be valuable to inform optimized dosing strategies for both male and female patients.

SC administration improves patient experience, treatment adherence and quality of life by markedly reducing administration time, increasing treatment convenience, decreasing hospitalization frequency, lowering both temporal and economic health care burdens, and eliminating complications associated with venipuncture (48). Therefore, the development of SC formulations for anticancer agents has gained increasing attention in oncological therapeutics. However, compared with IV administration, the localized reactions associated with SC injection may pose a limiting factor for its widespread adoption. Therefore, proper injection techniques and skin protection measures should be implemented when SC ADCs are administered to minimize or prevent these localized reactions.

Excessive injection rates, and improper injection angles or needle gauges may exacerbate ISRs (40,49). Therefore, standardized SC administration is essential for prevention. Since current research on the SC administration of ADCs is limited, the present case report offers recommendations for combining the pharmacological mechanisms of ADCs with those of established SC injection techniques for biologics such as monoclonal antibodies (40,49,50). i) Medication preparation: After removal from 2-8˚C refrigeration, allow 30 min to let the medication reach room temperature prior to injection, while avoiding improper heating or vigorous agitation to maintain pharmaceutical stability. ii) Needle: SC injections typically employ short (4-8 mm) and thin-wall needles with small gauges (25-27 G) and sharp tips to minimize pain. iii) Injection volume: The amount of drug injected should not be >2.0 ml per injection site to prevent injection pain, leakage and tissue distortion. iv) Injection site: Adipose-rich areas such as the abdomen and thighs should be chosen for injection, ensuring that the injection site exhibits no erythema, damage, ecchymosis, scarring, induration or hyperpigmentation. v) Rotation techniques: Injection sites should be rotated systematically to reduce irritation and ISRs, with subsequent injection administered ≥2.5 cm from previous sites. vi) Injection angle/technique: The non-dominant hand should be used to elevate a skin fold and the dominant hand should be used to insert the needle at a 30-40˚ angle, which can be adjusted on the basis of SC fat thickness to achieve optimal deposition. vii) Injection rate: The injection rate should not exceed 0.06 ml/sec to ensure patient comfort and proper drug dispersion. viii) Post-injection monitoring: A minimum of 1 h of observation is needed after injection to confirm patient safety before discharge.

Current clinical data indicates that ISRs are the most frequently reported AEs associated with SC-administered JSKN033, primarily manifesting as localized erythema, swelling, pain and pruritus (18). Most ISRs are mild in severity and typically resolve without intervention or can be effectively managed with physician-directed antihistamine therapy when necessary. For localized skin temperature elevation or erythema, cold compresses or topical corticosteroids may be applied (50). Analgesics should be considered for patients with notable pain. Warm compresses and physiotherapy may help alleviate induration. For severe cutaneous reactions such as ulceration or necrosis, immediate treatment discontinuation or regimen adjustment is warranted.

Although ISRs are usually mild and rarely classified as severe AEs, they may have a notable effect on patient satisfaction with treatment and even contribute to treatment discontinuation (51). Adequate patient education on the following aspects can help improve treatment experience. Before injection, patients should be informed about the procedure, precautions, common AEs and corresponding management measures. During the injection, any discomfort, such as pain at the injection site or systemic/local allergic reactions (for example, fever, chills, rash, dizziness or chest tightness), should be reported to medical staff immediately. After the injection, patients should be instructed to apply proper pressure to the injection site to minimize bleeding. The patients should also be educated on self-identifying AEs and promptly reporting them to medical staff, keeping the injection site clean and dry, avoiding water exposure for ≥24 h and bathing only after the injection site has fully healed. Furthermore, at 24 h post-injection, patients should not apply heat, undergo physiotherapy or vigorously massage the area in order to prevent capillary rupture and bleeding (52).

Due to study limitations and the personal preference of the patient, the present case report was unable to obtain additional imaging data (for example, ultrasound and MRI scans) or histological data regarding the AEs of the patient. The results described in the study are based on pathology reports rather than retrievable image files. Therefore, there is a lack of histopathological or biopsy evidence to further support diagnostic evaluation. Furthermore, during the current therapeutic response assessment, HER2 fluorescence *in situ* hybridization analysis was not performed, which represents a limitation of the present case report. JSKN033 remains in the emerging phase of clinical investigation. The AE mechanisms of JSKN033 and their management are still being investigated, with little existing research reported, to the best of our knowledge. Based on the present case report and the pharmacological mechanisms of JSKN033, potential AE mechanisms and management strategies were analyzed. However, these analyses had limitations. Notably, the small sample size inherent in a case report design limits the generalizability of the study findings and precludes definitive statistical conclusions. Furthermore, as the present case had only completed 13 weeks of administration at the time of manuscript preparation, longer-term follow-up outcomes were not yet available.

ADCs are currently a key research focus in oncology therapeutics. SC administration has demonstrated notable potential to improve the treatment experience and quality of life of patients due to its enhanced convenience. However, SC ADC formulations remain in the investigational stage and further study is warranted to fully characterize their AE profiles, and the mechanisms, prevention and management strategies of these AEs. The present study reported a case of systemic and localized AEs associated with SC ADC administration, aiming to provide data and potential insights for associated research in this field. JSKN033 remains in the emerging phase of clinical investigation, including the underlying mechanisms and management of its associated AEs, with, to the best of our knowledge, limited existing research. Based on the present case report and the underlying pharmacological mechanisms of JSKN033, the potential underlying mechanisms and management strategies of JSKN033-associated AEs were analyzed.

However, these analyses had limitations. Future studies may collect more comprehensive and long-term evidence to potentially improve the mechanistic understanding and clinical management of AEs associated with SC-administered ADCs to guide targeted management strategies.

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Availability of data and materials

The data generated in the present study are included in the figures and/or tables of this article.

Authors' contributions

RY wrote the original draft, devised the methodology, conducted the investigation and conceptualized the present case report. JZ advised on patient treatment, analyzed patient data, and critically reviewed and revised the manuscript. YW reviewed and edited the manuscript, devised the methodology and conceptualized the present case report. RY and YW confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The present case report was approved by the Ethics Committee of Fudan University Shanghai Cancer Center (Shanghai, China; approval no. 2409305-22).

Patient consent for publication

Written informed consent was obtained from the patient for the publication of the case report, including any potentially identifiable images or data.

Competing interests

The present case report has been published with the written permission of the sponsor of JSKN033 (Jiangsu Alphamab Biopharmaceuticals Co., Ltd.) from Clinical Trials ID no. NCT06226766 and Chinese Clinical Trial Registry ID no. CTR20244896. The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential competing interest.

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