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Review Article

Antibody drug conjugates: Next generation targeted cancer therapy

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ABSTRACT

Background: Antibody–drug conjugates (ADCs) are an evolving class of targeted anticancer therapeutics that combine monoclonal antibodies, linkers, and potent cytotoxic payloads to selectively deliver drugs to cancer cells expressing specific antigens. Advances in antibody engineering, linker stability, payload development, and conjugation technologies have expanded their therapeutic potential across multiple malignancies.

Objective: This review examines the structure, mechanisms of action, clinical applications, safety considerations, emerging developments, and role of pharmacists in the effective use of ADCs in cancer treatment.

Methods: A narrative literature review was conducted using PubMed/MEDLINE, Scopus, Embase, and Google Scholar for publications from January 2000 to November 2026. The search included literature on ADCs, therapeutic payloads, linkers, resistance mechanisms, interstitial lung disease, and pharmaceutical care. Following screening, 69 unique articles were selected from 103 identified records, prioritising peer-reviewed reviews and primary research, including phase I–III clinical trials.

Results: ADCs have demonstrated clinically meaningful benefits across breast, urothelial, lung, gastric, gynaecological, and haematological cancers. Agents such as trastuzumab deruxtecan, sacituzumab govitecan, enfortumab vedotin, and mirvetuximab soravtansine illustrate the expanding applications of antigen-directed therapy. Their effectiveness is influenced by antigen expression, linker stability, drug-to-antibody ratio, payload activity, and intracellular drug delivery. Important safety concerns include interstitial lung disease, myelosuppression, peripheral neuropathy, ocular toxicity, gastrointestinal disturbances, and hepatotoxicity. Antigen heterogeneity, acquired resistance, manufacturing complexity, treatment costs, and limited predictive biomarkers remain significant challenges. Emerging approaches include bispecific ADCs, novel payloads, site-specific conjugation, and combination regimens. Pharmacists contribute to safe preparation, dispensing, toxicity monitoring, supportive care, patient education, and pharmacovigilance.

Conclusion: ADCs represent an important advancement in precision oncology, offering targeted treatment opportunities across diverse cancer types. Further progress depends on optimising ADC design, improving patient selection, managing treatment-related toxicities, overcoming resistance, and enhancing equitable access. Multidisciplinary care, including oncology pharmacy expertise, will remain essential to maximising therapeutic benefit.

Keywords: Antibody–drug conjugates; targeted cancer therapy; precision oncology; monoclonal antibodies; cytotoxic payloads; drug-to-antibody ratio; adverse drug reactions; oncology pharmacy.

Introduction:

Antibody, drug conjugates (ADCs), which are basically molecules that combine three things a monoclonal antibody, a chemical linker, and a very strong cytotoxic medication, allow for targeted delivery of therapy. Monoclonal antibody binds to tumor-associated antigens and then is chemically attached to a very toxic payload (drug) through a linker.[1–3] The idea is to have high doses of toxic medicines be delivered specifically to cancer cells

expressing the antigen without causing harm to the normal tissue. Gemtuzumab ozogamicin, first approved drug in this class in 2000,[4] was the first ADC to reach clinical use, after which came brentuximab vedotin[5], and trastuzumab emtansine (T-DM1)[6] that further established CD30 and HER2 as suitable antigens for ADC therapies. Owing to the development of better monoclonal antibodies, more stable linkers, and novel cytotoxic payloads, subsequent generations of ADCs were able to deliver greater efficacy with better tolerability,[8] allowing them to be used earlier in treatment regimens and for treating different types of cancers. In this review, we present the general background and mechanism of action of ADCs, summarise the available clinical data, report on safety, highlight current progress and also consider the pharmacist’s responsibilities in ADC related drug therapy.

Methodology / Literature Search Strategy

Narrative review was the approach chosen. Literature searches were run across PubMed/MEDLINE, Scopus, Embase and Google Scholar from January 2000 through November 2026 by utilizing several combinations of the keywords "antibody-drug conjugate", "ADC", "trastuzumab deruxtecan", "sacituzumab govitecan", "enfortumab vedotin", "linker", "payload", "interstitial lung disease", "resistance" and "pharmacist". Peer-reviewed review articles and primary research articles (phase 1 to 3 trials, pharmacokinetic and translational studies) were considered as sources. Conference abstracts, press releases, guidelines, websites and material that is not peer-reviewed were not referenced. Pivotal randomised trials and the most recent high-quality reviews were selected above the rest. Two sources were identified as duplicates and 69 unique articles were selected from the total of 103 identified as most relevant. Since approvals after the latest peer-reviewed publication changes rapidly, the regulatory situation of individual ADCs (as of October 2026) was verified against regulator and sponsor announcements; such were only used for giving context and therefore should not be considered as references. Readers are advised to check current labeling before utilizing for clinical purposes.

Background

Structure and components

An ADC is comprised of three parts: an antibody, a linker, and a therapeutic agent (payload).[1] The best targets for ADC therapy are those that are greatly and evenly expressed on malignant cells, have minimal expression on essential normal tissues, and undergo internalisation only after antibody binding.[9],[10] The majority of commercially available ADCs employ the IgG1 or IgG4 framework. The stability in the bloodstream and the release of chemotherapy from the antibody is largely determined by the linker. Payloads in cleavable linkers such as acid-labile hydrazones, disulfides, proteased-cleavable valine, citrulline or tetrapeptide sequences, and glucuronide, are released when the lysosomes (tumour-microenvironment) break down, whereas payload release via non-cleavable thioether requires total lysosomal digestion of the antibody.[11–13] Payloads can broadly be categorised into three groups: microtubules poisons (auristatins like MMAE and MMAF; maytansinoids like DM1 and DM4), DNA-damaging chemotherapeutics (calicheamicin, pyrrolobenzodiazepine dimers), and topoisomerase I inhibitors (SN-38 and the exatecan derivative DXd).[14],[15]

The drug-to-antibody ratio (DAR) plays a critical role in drug potency, the drug's ability to be cleared from the body, and its toxic effects: a higher DAR results in greater delivery of chemotherapeutic drug; however, this can also lead to quicker clearance of the drug and higher toxicity.[16]

Site-specific covalent linking enables the drug to be attached at specific locations, which produces better quality and very uniform products with a higher safety margin.[17] A representative list of approved ADCs is presented in Table 1.

Mechanism of action

Once bound to its target antigen, the ADC-antigen complex is internalised by endocytosis and is directed to the lysosome, where the payload is released to show an effect of cell death (Figure 1).[10] Membrane-permeable payloads such as MMAE, SN-38, DXd, can exit the target cell and cause death of adjacent antigen-negative cells, termed the "bystander effect", a phenomenon very important when tumour has heterogeneous antigen expression.[18] From the pharmacokinetic perspective,

ADC exposure is mainly determined by antibody clearance, linker deconjugation in plasma and disposal of the free(payload), which altogether have a strong influence on effectiveness and toxicity respectively.[19]

Evolution of ADC design

Early ADCs (antibody-drug conjugates) combined murine antibodies or chimeratic antibodies with weak linkers and limited drug delivery.

In the subsequent generation of antibody-drug conjugates, apart from using humanized antibodies, better stability of linkers, and a much greater variety and potency of payloads were also introduced, examples include Brentuximab Vedotin, T-Dm1.

The third generation of ADCs features the use of highly targeted site-specific conjugation methods, optimization of DAR (Drug-to-Antibody Ratio), and the incorporation of super potent topoisomerase I inhibitors that can work on the bystander for example, Trastuzumab Deruxtecan.[7,8]

Current Evidence

Breast cancer

HER2-positive disease. In the TH3RESA study, T-DM1 extended the overall survival of heavily pretreated HER2-positive patients with metastatic breast cancer to 22.7 vs 15.8 months respectively, physician's choice, which adds to the previous progression-free survival benefit shown in EMILIA.On the one hand, KATHERINE was the trial that demonstrated that the use of T-DM1 as adjuvant treatment for patients with residual invasive disease after neoadjuvant therapy gave a significantly higher 3-year invasive disease-free survival (88.3% vs 77.0%).On the other hand, trastuzumab deruxtecan (T-DXd) produced an objective response rate of 60.9% in DESTINY-Breast01 after heavily pretreated metastatic breast cancer patients had failed the T-DM1 treatment and had the trastuzumab/chemotherapy regimen, i.e., a patient had received several drugs that include trastuzumab followed by several drugs without it after the progression.In the DESTINY-Breast03 trial, T-DXd showed very good results for progression-free survival compared to T-DM1 as T-DXd had 12-month PFS of 75.8% vs 34.1% of T-DM1.After these trial results, T-DXd was approved for first-line metastatic HER2-positive disease besides being an anti-cancer drug for neoadjuvant and post- neoadjuvant residual-disease settings in which, for example, DESTINY-Breast09, -11 and -05 were involved.

HER2-low disease. DESTINY-Breast04 trial identified HER2-low metastatic breast cancer as a new potential treatment target as T-DXd offered improvement in PFS (9.9 vs 5.1 month) and OS (23.4 vs 16.8 months) over chemotherapy.[24] The DAISY phase 2 trial demonstrated response against all HER2 levels, including very minimal HER2 staining tumours, and explained the mechanism of bystander effect.[25]

TROP2-directed ADCs. In the case of metastatic triple-negative breast cancer (TNBC), the drug sacituzumab govitecan (SG) proved effective in a phase 1/2 basket trial26 and then it significantly increased the median progression-free survival (PFS) (5.6 vs 1.7 months) and overall survival (OS) (12.1 vs 6.7 months) in comparison to chemotherapy through the phase 3 ASCENT trial.27 For the hormone receptor-positive/ HER2-negative subtype, SG improved both PFS and OS in the TROPiCS-02 trial 28, and datopotamab deruxtecan was also able to improve PFS from 6.9 to 4.9 months in the TROPION-Breast01 trial without a significant change in overall survival(OS).29 After these results, both SG and datopotamab deruxtecan received first-line indications for the treatment triple-negative breast cancer through further phase 3 trials.

Urothelial carcinoma

Enfortumab vedotin (Nectin-4, directed, MMAE) demonstrated efficacy in EV-201 showing 44% response rate following treatment with platinum and checkpoint-Inhibitors as well.[30] In study EV-301, this therapy has been found to prolong OS significantly as compared to chemotherapy 12.9 months versus 9.0 months[31]. In Phase III clinical trial EV-302 the use of combination therapy of enfortumab vedotin with pembrolizumab in patients with previously untreated advanced urothelial cancer significantly improved the median PFS (12.5 versus 6.3 months) and median OS (31.5 versus 16.1 months) compared to that of only platinum chemotherapy. The new approach will become standard for first-line treatment.[32]

Lung cancer

T-DXd exhibited a quite impressive 55% response rate and a median progression-free survival (PFS) duration of 8.2 months in HER2-mutant non-small-cell lung cancer (DESTINY-Lung01).[33] Datopotamab deruxtecan

enhanced PFS in comparison to docetaxel in TROPION-Lung01 (the result was 4.4 vs 3.7 months), but there was no significant overall survival (OS) improvement in the total population.[34]

Gastrointestinal and tumour-agnostic evidence

Destiny-Gastric01 found that for previously treated HER2-positive gastric cancer, T-DXd not only improved response rates (51% vs 14%) but chemotherapy extended overall survival (OS) as well (12.5 months vs 8.4 months).[35] A Western population showed similar support results, as reported in Destiny-Gastric02.[36] A trial of Destiny-PanTumor02, showed long-lasting responses in HER2-expressed solid tumors and particularly the patients having IHC 3+ tumors, thereby a tissue-specific approach is supported.[37]

Gynaecological cancers

Mirvetuximab soravtansine, a folate receptor alpha-targeted DM4 antibody-drug conjugate, elicited an overall response rate of 32.4% in SORAYA[38], significantly prolonging PFS (5.6 vs 4.0 months) and OS (16.5 vs 12.8 months) compared with chemotherapy in MIRASOL for platinum-resistant ovarian cancer[39]. Tisotumab vedotin, a tissue factor-targeted MMAE antibody-drug conjugate, resulted in 24% best overall response in innovaTV 204[40] and OS was favorably impacted as compared with chemotherapy (11.5 vs 9.5 months) in patients with recurrent cervical cancer in innovaTV 301.[41]

Haematological malignancies

Combining brentuximab vedotin with chemotherapy yielded better modified 2-year PFS outcomes in the management of advanced Hodgkin lymphoma (ECHELON-1)[42]. Polatuzumab vedotin plus bendamustine, rituximab enhanced complete response rates (40% vs 17.5%) in patients with relapsed/refractory DLBCL [43], while polatuzumab vedotin combined with R-CHP proved more effective for PFS than R-CHOP in the treatment of the untreated DLBCL patients (POLARIX).[44] Loncastuximab tesirine led to a good response rate of 48.3% in a study of patients with relapsed/refractory DLBCL (LOTIS-2).[45] Inotuzumab ozogamicin produced a significantly higher complete remission rate than placebo (80.7 vs 29.4%) among the patients with relapsed/refractory acute lymphoblastic leukaemia.[46] Meanwhile, the addition of gemtuzumab ozogamicin to the course of induction chemotherapy enhanced the event-free survival in newly diagnosed cases of acute myeloid leukaemia.[47] Belantamab mafodotin (anti-BCMA, MMAF) produced the response rate of 31% in the DREAMM-2[48] study. Moreover, the addition of belantamab to the chemotherapy regimens improved the PFS in DREAMM-7 (median 36.6 vs 13.4 months)[49] and in DREAMM-8,[50] even though the drug's eye toxicity limited the dose administration.

Clinical Applications

The usage of ADCs is now mainly guided by biomarker testing. Target expressions are evaluated by various techniques such as IHC and in situ hybridization (eg, HER2 IHC 0, 1+, 2+, 3+; folate receptor alpha; PD-L1 for combinations), and it turns out that HER2 low or ultralow disease still can benefit from ADCs [24,25]. Tropa blast surface antigen TROP2 and Nectin-4 are so broadly expressed that the only test for predicting the biomarker has not yet been established for SG or enfortumab vedotin's indication [27,32]. They are often prescribed as the last line of treatment alone, but they are becoming more and more popular in the front-line or curative-intent scenarios, both with immune checkpoint inhibitors or alone. The first is enfortumab vedotin plus pembrolizumab.

The T-DXd and T-DM1, though having similar names, are not interchangeable medicines because they differ in terms of payload, DAR, linker drug release, dose, and toxicity profile [23,54].

It is not known precisely where ADC treatment should come in after ADC disease progression. Class switching of payloads, like going from, topoisomerase to microtubule inhibitor, seems quite a reasonable idea and is being explored.[3]

See Table 1 for a list of cancer-specific targets and indications.

Abbreviations: ALCL, anaplastic large-cell lymphoma; ALL, acute lymphoblastic leukaemia; AML, acute myeloid leukaemia; DLBCL, diffuse large B-cell lymphoma; HR, hormone receptor; NSCLC, non-small-cell lung cancer; PBD, pyrrolobenzodiazepine; TNBC, triple-negative breast cancer. Approved indications vary by jurisdiction and change frequently; verify against current labelling.

Safety & Limitations

Class and payload-related toxicities

ADC toxicity mainly occurs as a consequence of on-target/off-tumour binding, payload released too soon

and also non-specific uptake, and it is mostly the payload class that determines toxicity instead of antibody target.[51–54] A clinical toxicity meta-analysis by payload demonstrated the characteristic toxicity pattern: auristatins caused neutropenia and neuropathy, whereas maytansinoids were responsible mainly for thrombocytopenia, and calicheamicin resulted in liver toxicity.[52] In contrast, topoisomerase I-inhibitor ADCs have been reported for myosuppression as well as gastrointestinal toxicity besides some cases having interstitial lung disease, e.g. T-DXds.[54] The toxic effect of eyes (keratopathy, conjunctivitis, and dry eyes) is related to three MMAFs (MMAE, MMAF and DM1) and some ADCs (Tisotumab Vedotin),[39–41,55] of which corneal epithelial changes are observed in the majority of patients who received belantamab mafodotin in DREAMM-2. Table 2 lists the main toxicities and management options together with strategies for prevention.

Interstitial lung disease:

In a combined analysis of the nine T-DXd monotherapy trials drug-related ILD/pneumonitis was drug-related ILD/pneumonitis occurred in approxl 15% mostly mild but including a small percentage fatal drug-related ILD/pneumonitis. The multidisciplinary guidelines stress the importance of regular monitoring, early imaging, immediate stop of medication and corticosteroid administration based on severity level. Recommendations for managing T-DXd also include the measures for treating nausea, monitoring neutropenia, and monitoring the card.

Haematological and gastrointestinal toxicity of SG

In the ASCENT safety analysis, neutropenia and diarrhoea were the most common grade 3 or worse side effects and they were treated successfully through dose manipulation, G-CSF, and antidiarrhea drugs [59]. These adverse effects are mostly related to the SN-38 payload, while the homozygous UGT1A1*28 patients have a higher chance of neutropenia [59].

Resistance mechanisms

Cancer cells may find various ways to avoid ADC therapy which includes downregulation/mutation of target molecule; problems in internalisation/trafficking; changed lysosomal function; activation of drug transporter pumps; changes in ADC-cell complex formation.[61,62] It was reported that mutations in antigen and payload target genes were involved in SG resistance in TNBC.[63]

Limitations

In addition to these limitations, there are several challenges like antigen heterogeneity which is very important for the success of ADCs. One more limiting factor would be a narrow therapeutic window for some payloads, plasma instability of some linkers, immunogenicity, the high complexity and cost of manufacturing.[1,64]

It has turned out, for example, that some otherwise active antigen-targeted drugs did not result in an improvement of overall survival. Also, the exact way of giving the doses, in particular for drugs having narrow safe-dose ranges, is a subject of ongoing investigations.[29,34,64]

Emerging Advances

Bispecific ADCs, targeting two distinct epitopes or antigens, are meant to enhance specificity, internalization, and effectiveness especially in the cases of tumors that present different subpopulations of cells.65 Antibodies delivering immune-stimulation conjugates (Toll-like receptor agonists) can be delivered myeloid cells resulting in durable antitumor immunity. This was observed in preclinical studies with immune-stimulating antibody conjugates.66 Antibody-conjugated PROTACs (protein degraders) have demonstrated HER2-dependent degradation and destruction of intracellular targets such as BRD4, thereby broadening the concept of payload beyond cytotoxics.67 New paths include payloads with mechanisms of non-cytotoxic effects, a dual-payload ADC, an activated antibody, a condition dependent one, identification of new targets (For example, B7-H3, c-Met, CDH6) and combinations with drugs inhibiting checkpoints and targeted therapies. 7,64 Site-specific conjugation techniques are being improved, as well as hydrophilic linkers, with an aim to achieve a higher drug-to-antibody ratio (DAR) without compromising the body's pharmacokinetics.[17]

Pharmacist's Role

Pharmacists play a role in ensuring ADC safety and its benefits at all drug use stages.[68,69] Verification and dispensing: double-check the correct medication, dose

based on weight, diluent type and compatible infusion set; prevent look-alike/sound-alike drug errors (e.g., T-DM1 vs T-DXd), and follow the chemotherapy safe-handling and administration guidelines.[23,54,69]

Compounding and handling drug: prepare the drug as aseptically as possible with hazardous-drug precautions, use manufacturer-specified reconstitution and storage and shield from light where necessary[69].

Premedication and supportive care: administer antiemetic prophylaxis based on emetic risk, use G-CSF, apply antidiarrhoeal protocols for SG and provide corticosteroid eye drops or lubricants if ocular toxicity is expected.[56,59,60]

Toxicity monitoring: ILD symptoms (cough, dyspnoea, fever), ocular symptoms, neuropathy, liver function and blood counts, etc. should be assessed at baseline and then periodically, and prompt actions should be taken when necessary.[57,58]

Pharmacogenomics and interactions: providing a supporting role in the interpretation of SG's UGT1A1 genotype, reviewing the list of interfering medications, especially potent CYP3A4 inhibitors which may raise serum exposure of the free payload for certain ADCs.[51,59]

Patient education and compliance: explaining to the patient about the infusion time line, when to seek urgent care, contraception requirements, and highlighting the importance of informing the healthcare provider in case one experiences any new or increased respiratory issues or visual symptoms after the start of the treatment.[60]

Pharmacovigilance and accessing: adverse drug reaction reporting, dose modification auditing, access and cost-effectiveness review support as well as formulary decisions are carried out as the number of ADCs increases.[68]

Research Gaps

Major gaps that remain are:

(1) proven biomarkers for predicting drug action apart from the target molecule (i.e., those related with payload sensitivity, internalisation, SLFN11, TOP1 status)

(2) best use strategy for ADCs together with the possible development of resistance to different ADC payloads

(3) ILD and ocular toxicity, their occurrence mechanisms and how to prevent them

(4) defining the most suitable dose and doing the exposure, responsiveness analysis

(5) identifying the safety of long-lasting treatments and the well-being of survivorships if the intent of treatment is cured

(6) clinical trials to determine if ADCs or combinations are the best; and

(7) health-economic assessment and ensuring the fairness of global distribution.

Future Perspectives

The future of ADC innovation will primarily include design features such as bispecific ADC formats, activation under certain conditions, new payloads, quantitative target assessment, circulating tumour DNA and payload-sensitive biomarkers for the selection of patients more accurately, and also earlier-line and combination use of the therapies. [7,65–67] The potential of combining ADCs together with immunotherapies such an example is given by enfortumab vedotin plus pembrolizumab which have shown a significant improvement in survival[32] will need to be explored further.

To ensure safe implementation of the benefits seen in clinical trials into daily clinical practice, monitoring pathways by the pharmacist, shared decision tools and real world pharmacovigilance will play a very important role.

Conclusion

Antibody-drug conjugates, which had been the kind of therapy to which few resort, are now among the top cancer therapies with the proven life prolonging effects in breast, urothelial, gastric, gynaecological and haematological cancers. To achieve a full benefit of the drugs, it is necessary to properly select the target, optimise the linker-payload chemistry, and be able to handle the unique side-effects of the therapy. The main limitations are resistance, heterogeneous antigen expression and access. We believe that by continued conjugate design innovations, better biomarkers, and combinations, as well as team-based, holistic care that includes oncology pharmacists, ADCs will become more widely used drug therapy options, with reduced toxicity profiles.

Tables and Figures

Table 1. Representative approved ADCs and key supporting evidence

ADC	Target	Payload (linker)	Principal indication(s)
Gemtuzumab ozogamicin	CD33	Calicheamicin (cleavable, acid-labile)	AML
Brentuximab vedotin	CD30	MMAE (cleavable, protease)	Hodgkin lymphoma, ALCL
Inotuzumab ozogamicin	CD22	Calicheamicin (cleavable, acid-labile)	B-cell ALL
Polatuzumab vedotin	CD79b	MMAE (cleavable, protease)	DLBCL
Loncastuximab tesirine	CD19	PBD dimer (cleavable)	DLBCL
Belantamab mafodotin	BCMA	MMAF (non-cleavable)	Multiple myeloma
Trastuzumab emtansine	HER2	DM1 (non-cleavable)	HER2+ breast cancer
Trastuzumab deruxtecan	HER2	DXd, topoisomerase I inhibitor (cleavable, tetrapeptide)	HER2+/HER2-low breast, gastric, HER2-mutant NSCLC, HER2 IHC 3+ solid tumours
Sacituzumab govitecan	TROP2	SN-38 (cleavable, hydrolysable)	TNBC, HR+/HER2- breast cancer, urothelial cancer
Datopotamab deruxtecan	TROP2	DXd (cleavable, tetrapeptide)	Breast cancer, NSCLC
Enfortumab vedotin	Nectin-4	MMAE (cleavable, protease)	Urothelial carcinoma
Tisotumab vedotin	Tissue factor	MMAE (cleavable, protease)	Cervical cancer
Mirvetuximab soravtansine	Folate receptor alpha	DM4 (cleavable, disulfide)	Platinum-resistant ovarian cancer

Table 2. Key ADC toxicities and management considerations

Toxicity	Associated ADCs / payloads	Management considerations (ref.)
Interstitial lung disease/pneumonitis	T-DXd (topoisomerase I payload)	Baseline and ongoing symptom review, early imaging, interrupt/discontinue by grade, corticosteroids (57, 58)
Ocular toxicity (keratopathy, conjunctivitis)	Belantamab mafodotin (MMAF), mirvetuximab (DM4), tisotumab vedotin (MMAE)	Ophthalmic examination, lubricants/steroid drops as specified, dose modification (39, 41, 55, 56)
Peripheral neuropathy	MMAE conjugates	Neurological assessment, dose reduction or discontinuation (52, 54)
Neutropenia and febrile neutropenia	SG, T-DXd, MMAE conjugates	Blood counts, G-CSF, dose modification (52, 59)
Thrombocytopenia, hepatotoxicity	DM1 (T-DM1); calicheamicin (hepatic veno-occlusive disease)	Platelet and liver monitoring (51, 52)
Diarrhoea, nausea/vomiting	SG (SN-38), T-DXd	Antidiarrhoeal and antiemetic prophylaxis; UGT1A1 awareness for SG (59, 60)
Cutaneous reactions	Enfortumab vedotin	Early recognition, dermatological referral, withhold for severe reactions (31, 32)

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