Review Article

Sacituzumab govitecan in breast cancer

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Abstract: Sacituzumab Govitecan (SG, IMMU-132) is an antibody-drug conjugate that links the irinotecan active metabolite, SN-38 to a humanized monoclonal antibody targeting Trop-2, a transmembrane calcium signal transducer. Trop-2 is highly expressed in epithelial cancer cells of multiple tumor types, including breast cancer. Therefore, the anti–Trop-2 monoclonal antibody allows for targeted delivery of SN-38 to tumor cells. SN-38 is membrane permeable and may elicit antitumor effects in adjacent tumor cells (bystander effect) before internalization of the antibody–drug conjugate through hydrolysis of the linker or by intracellular SN-38 release after internalization. SG has been approved to treat patients with triple-negative breast cancer (TNBC) and patients with urothelial cancer in US. In phase III confirmatory ASCENT trials, SG showed more significant efficacy benefit in TNBC patients who had received at least two prior systemic therapies than treatment of physician choice. Neutropenia and diarrhea are the most common adverse events and can be managed with supportive care. The incidence of treatment discontinuation due to adverse events was low (<5%). Evidence has shown that SG should be considered as a new standard of care in patients with pretreated metastatic triple-negative breast cancer (mTNBC). Besides, SG has shown encouraging activity in patients with pretreated $HR^+/HER2^-$ mBC with a predictable, manageable safety profile. Here, in this review article, we summarize recent studies of SG for breast cancer treatment and provide a systematic review of the recently developed therapeutic approach.

Keywords: Antibody-drug conjugate (ADC); sacituzumab govitecan (SG); Trop-2; breast cancer

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Background

Triple-negative breast cancer (TNBC) is a subtype of breast cancer that lacks expression of estrogen receptor (ER), progesterone receptor (PR) and human epidermal growth factor 2 receptor (HER2) on cancer cell surface. Approximately 15% of breast cancer (BC) patients were diagnosed with TNBC and had the worst prognosis among all subtypes of BC (1). Currently, chemotherapy remains to be the backbone for treatment of metastatic TNBC (mTNBC). However, standard chemotherapy is associated with low response rates (10% to 15%) and short progression-free survival (PFS) (2 to 3 months) among patients with pretreated mTNBC. In recent studies, several novel drugs including PD-1/PD-L1 inhibitors and PARP inhibitors showed promising results for the treatment of TNBC in front lines (1,2).

Based on IMpassion 130, FDA approved the combinational treatment of Atezolizumab and nab-paclitaxel in patients with advanced PD-L1-positive (defined as PD-L1 stained tumor-infiltrating immune cells (IC) of any intensity covering ≥1% of the tumor area) TNBC as first-line treatment. The median PFS of Atezolizumab/nab-paclitaxel combinational treatment group was 7.5 months, while it was 5 months with nab-paclitaxel single treatment in PD-L1–positive subset (hazard ratio, 0.62; 95% CI, 0.49–0.78; P<0.001). Overall survival (OS) in the combinational treatment group was 25.0 months, while it was 15.5 months with nab-paclitaxel

Table 1 Clinical results of targeted and immuno-therapeutic drugs

Agent	MOA	Patients	Phase & patient size	Key efficacy results	Main toxicity
Olaparib *vs.* single-agent chemo	PARPi	≤2L chemo for gBRCAm mBC	III, 205 *vs.* 97	BRCAm, PFS, 7.0 *vs.* 4.2 months	Anemia, gastrointestinal toxicity
Talazoparib *vs.* single-agent chemo	PARPi	≤3L chemo for gBRCAm mBC	III, 287 *vs.* 144	BRCAm, PFS, 8.6 *vs.* 5.6 months	Anemia, gastrointestinal toxicity
Pembrolizumab + chemo *vs.* chemo	Anti-PD-1	Untreated mTNBC	III, 566 *vs.* 281	PD-L1+: PFS, 9.7 *vs.* 5.6 months	Gastrointestinal toxicity, myelosuppression
Atezolizumab + Nab-pac *vs.* Nab-pac	Anti-PD-L1	Untreated mTNBC	III, 451 *vs.* 451	PD-L1+: PFS, 7.5 *vs.* 5.0 months; OS, 25 *vs.* 15.5 months	Alopecia, nausea, cough, peripheral neuropathy

mTNBC, metastatic triple-negative breast cancer; PFS, progression-free survival; OS, overall survival.

single treatment in PD-L1–positive subset (hazard ratio, 0.62; 95% CI, 0.45–0.86) (3). Similarly, the phase III KEYNOTE-355 study evaluated the efficacy of pembrolizumab combined with chemotherapy in mTNBC as first-line treatment. Improvement in PFS was noted in patients with PD-L1–positive tumors (defined as a combined positive score >10) treated with immunotherapy plus chemotherapy versus chemotherapy alone (median PFS 9.7 *vs.* 5.6 months; hazard ratio, 0.65; 95% CI, 0.49–0.86; one-sided P=0.0012) (4).

In a phase III OlympiAD trial, the PARP inhibitor olaparib increased PFS compared with chemotherapy in patients with advanced HER2-negative BC with germline BRCA mutation (median PFS 7.0 *vs.* 4.2 months, hazard ratio, 0.58; 95% CI, 0.43–0.80; P<0.001) (5). Similar results were also demonstrated with another PARP inhibitor, talazoparib in advanced BC patients with germline BRCA1 and BRCA2 mutations in the EMBRACA study (8.6 *vs.* 5.6 months; hazard ratio, 0.54; 95% CI, 0.41–0.71; P<0.001) (6). However, a majority of patients have disease progression after receiving PD-1/PD-L1 inhibitors and PARP inhibitors, and standard therapeutic options are limited to chemotherapy. The clinical results of targeted and immune-therapeutic drugs are shown in *Table 1*.

Antibody-drug conjugates (ADC)

Traditional alkylating agents, antimetabolites and anti-mitotic agents are main chemotherapy drugs (7). The emergence of antibody drugs such as HER2 mAb made combinational therapy between antibody and chemotherapy a more promising therapeutic approach for $HER2^+$ breast cancer. With the continuous improvement of antibody technology, several ADC drugs have been approved for cancer therapy.

For the first-generation of ADCs, murine antibodies were highly immunogenic. Human anti-murine antibodies (HAMAs) have reduced efficacy and toxicity. Conventional cytotoxic drugs, including doxorubicin, methotrexate, mitomycin as payload, the active dose can be within the micromolar range and are mainly conjugated with the mouse monoclonal antibody by non-cleavable linkers (amide or succinimide) (8).

For the second generation of ADCs, chimeric, humanized or fully human antibodies have reduced immunogenicity. In addition, active doses of humanized antibody that couples with highly toxic drugs can be of pmol level. However, the second-generation ADCs have a narrow therapeutic window, mainly due to off-target toxicity, fast clearance, and competition with unconjugated antibodies. The second-generation ADC drugs have different drug antibody ratios (DAR) from 0–8. It usually shows low tolerance, high plasma clearance efficiency and low in vivo efficacy when DAR over 4. For example, the DAR of Brentuximab Vedotin is 4, the DAR of Ado-Trastuzumab emtansine DAR is 3.5, and the DAR of Inotuzumab Ozogamicin is 6 (9).

The key to the third-generation ADC drugs is the site-specific binding, which ensures antibody conjugate drugs with a clear DAR. In addition, the third-generation ADC drugs have significantly improved therapeutic efficacy in terms of antibody optimization, linkers, and binding of small-molecule drugs. The representative drugs include sacituzumab govitecan (SG) and Fam-trastuzumab deruxtecan. The third-generation ADC, which coupled with humanized mAb at a higher ratio, uses fewer toxic drugs, allows higher doses of drugs to be delivered to tumor site and executes a bystander effect (10). The major differences from 1st to 3rd generation of ADC are shown in *Table 2*.

Table 2 Differences from 1st to 3rd generation of antibody-drug conjugates

Variables	First generation	Second generation	Third generation
Antibody	Murine antibodies	Human antibodies	Human antibodies
Payloads	Weak potency	High potency	Moderate or high potency
Conjugation strategy	Random coupling	Random coupling	Random or specific coupling
Linkers	Non-cleavable	Non-cleavable	Cleavable
Homogeneous (drug to antibody ratio)	Heterogeneous	Heterogeneous	Homogeneous
Bystander effect	None	None	Most Have

Trop-2

Trop-1, 2, 3, and 4 were first identified as transmembrane proteins expressed on the surface of normal and malignant trophoblast cells in 1981. Abnormally elevated expression of Trop-2 was found in a variety of cancer tissues regardless of the levels of its baseline expression in normal tissues (11). One possible mechanism of high expression of Trop-2 in cancer is that transcription factors that regulate cancer cell progression, like WT1, can enhance the transcription and expression of Trop-2. Trop-2 modulates growth, invasion and proliferation of BC cells and is upregulated in breast cancer cells, making it an ideal target for novel therapies for breast cancer (12).

In a nude mouse model of MDA-MB-231 BC cells, Trop-2-targeted antigen-binding fragments (Fab) has been demonstrated to inhibit the occurrence and progression of breast cancer. Besides, another novel human Fab antibody for Trop-2 inhibits breast cancer growth *in vitro* and *in vivo* (13). IMMU-132 also known as SG is a Trop-2 targeting ADC conjugated with topoisomerase I inhibitor SN-38, which is an active metabolite of irinotecan and has shown anti-tumor effect in multiple cancer types including breast cancer. The SN-38 deliver into the tumor of IMMU-132 was 136 times more than that of irinotecan as demonstrated in a preclinical mouse model of allograft of breast cancer (14).

As a novel therapeutic target, Trop-2, an internalizing antigen/antibody, proprietary linker chemistry, and high drug payload has led to a high therapeutic index of IMMU-132 with acceptable tolerability, with a less occurrence of diarrhea than irinotecan (15).

SG (IMMU-132, Trodelvy®)

SG is a first-in-class Trop-2 ADC drug. It contains three parts: a humanized monoclonal antibody (hRS7), payload SN-38 and a linker. The monoclonal antibody binds to Trop-2, the trophoblast cell-surface antigen. The payload SN-38 is a topoisomerase inhibitor metabolized from Irinotecan that prevents repair of DNA damage and leads to apoptosis and cell death. The monoclonal antibody and SN-38 are conjugated by a hydrolyzable linker with a drug to antibody ratio of 7.6. Preclinical studies have shown that SG targets and binds to Trop-2-expressing cancer cells, which is subsequently internalized by cells. Upon entry into the cell, SG is hydrolyzed to release SN-38. SN-38 inhibits topoisomerase I activity by competitively binding to topoisomerase I, preventing its molecular function, which is involved in single strand break reconnection. Eventually, the accumulated DNA damage induces apoptosis of cancer cells. The key characteristics of SG are shown in *Table 3*.

In April 2020, FDA granted accelerated approval for the use of SG in mTNBC patients, who previously received at least two regimens for metastatic disease, based on clinical efficacy and safety data from a phase I/II basket study (IMMU-132-01) (16). In September 2020, the primary results of phase III ASCENT clinical study confirmed these findings. Recently, based on the published results of ASCNET study, FDA granted a full approval to sacituzumab govitecan-hziy (Trodelvy) for the treatment of patients with unresectable locally advanced or metastatic triple-negative breast cancer (TNBC) who have previously received 2 or more systemic therapies, at least 1 of them for metastatic disease (17). In May 2021, SG also obtained the priority review for treatment of mTNBC by National Medical Products Administration (China NMPA). The milestone of SG's use in BC are shown in *Table 4*.

Clinical data

IMMU-132-01 TNBC cohort

IMMU-132-01 is a phase I/II basket study. In mTNBC (18)

Table 3 The characteristics of sacituzumab govitecan

Properties	Properties
Antibody	A humanized mAb targeting Trop-2
	Trop-2 is a newly discovered cancer protein, which is overexpressed in multiple epithelial cancers and regulates signaling pathways involved in cancer cell proliferation and metastasis
	The humanized mAb shows ADCC activity
Payload	SN-38, as an inhibitor of topoisomerase I, which could induce DNA damage, leading to cancer cell apoptosis and cell death
	SN-38 is 2–3 logs more toxic than its prodrug, irinotecan
	Overcome cross-resistance to prior chemotherapy drugs
	Given its membrane-permeable nature, free intracellular SN-38 may disperse outside of cells and elicit antitumor effects to adjacent tumor cells
Linker	A high drug-to-antibody ratio, with an average of 7 to 8 molecules of SN-38 per antibody molecule
	CL2A linker is moderately stable and pH-sensitive, resulting in payload release from the cell and the tumor microenvironment outside the cell before internalization, providing a bystander effect

Table 4 Key milestone of sacituzumab govitecan in breast cancer

Time	Stone
Jan. 2015	FTD for mTNBC in USA
Feb. 2016	Breakthrough therapy designation
May 2018	Submission by BLA in USA (May)
Jul. 2018	Acceptance and Priority Review granted by BLA in USA
Jan. 2019	Received FDA Complete Response Letter
Dec. 2019	Submission by BLA
Apr. 2020	Accelerated approved
Apr. 2021	Regular approved for mTNBC in US
May 2021	Priority approval status of National Medical Products Administration (China)

FTD, fast track designation; mTNBC, metastatic triple-negative breast cancer.

cohort, a total of 108 patients received SG after at least two systemic treatments for mTNBC. On days 1 and 8 of every 21 days treatment cycle, patients were given SG (10 mg/kg) intravenously. Patients received a median of three previous systemic therapies, like taxanes and anthracyclines. The investigator-assessed objective response rate (ORR) was 33.3%. The median duration of response was 7.7 months. The median progression-free survival was 5.5 months. The median overall survival were 13.0 months. Ninety-two of 108 patients (85%) had Grade 3/4 adverse events (AEs). Most common adverse events related to SG was GI and hematological toxicities. A proportion of 6.5% of patients have Grade 3 febrile neutropenia and 8% patients have Grade 3 diarrhea. Dose reduction occurred in 36 of the 108 patients (33%). Three patients (2.8%) discontinued treatment due to AEs.

IMMU-132-01 HR+ cohort

Fifty-four patients were enrolled in the HR+ cohort of IMMU-132-01 (19). All patients were received at least two kinds of systemic therapies in any setting, one of which must have been endocrine-based therapy. SG (10 mg/kg, I.V.) was applied to them on days 1 and 8 of a 21-day treatment cycle. Patients received a median of three previous endocrine therapy regimens in any setting and

Table 5 Summarized clinical results in breast cancer with sacituzumab govitecan

Variables	IMMU-132-01-TNBC (N=108)	IMMU-132-01-HR+ (N=54)	ASCENT (N=235, SG arm without brain metastasis)
Patients	mTNBC	HR+ mBC	mTNBC
Prior line	≥2 prior treatments for TNBC	Disease progressed on endocrine-based therapy and at least one prior chemotherapy for mBC	≥2 prior treatments
ORR (%)	33.3	31.5	35
PFS, median (95% CI), months	5.5 (4.1–6.3)	5.5 (3.6–7.6)	5.6 (4.3–6.3)
OS, median (95% CI), months	13.0 (11.2–13.7)	12 (9–18.2)	12.1 (10.7–14.0)
Time to response, median (range), months	2.0 (1.6–13.5)	2.1 (1.4–7.8)	1.5 (0.7–10.6)
DOR, median (95% CI), months	7.7 (4.9–10.8)	8.7 (3.7–12.7)	6.3 (5.5–9.0)
Most key reported grade 3 or 4 AEs	Neutropenia (42%), anemia (11%), diarrhea (8%).	Neutropenia (50%), anemia (13%), diarrhea (7.4%)	Neutropenia (52%), anemia (9%), diarrhea (11%)
AEs leading to treatment discontinuations	2.8%	5.6%	4.7%

mTNBC, metastatic triple-negative breast cancer; ORR, objective response rate; PFS, progression-free survival; OS, overall survival; DOR, duration of response; AE, adverse event.

median two prior chemotherapy regimens in metastatic setting. The mean follow-up time was 11.5 months. The ORR, median duration of response, median PFS, and median OS were 31.5%, 8.7 months, 5.5 months, and 12 months, respectively. The most common grade 3/4 treatment-related adverse events were neutropenia (50.0%), anemia (11.1%), and diarrhea (7.4%). Two of the patients stopped the current therapy regimen due to treatment related AE. None of the patients died of SG related AE.

ASCENT study

The ASCENT study was an open label, randomized and controlled phase III trial (20). ASCENT investigated the efficacy and safety of SG versus the treatment of physician's choice (TPC). TPC is single-agent chemotherapy (eribulin, vinorelbine, capecitabine, capecitabine), a standard care in later liner of mTNBC. All patients were relapsed or refractory to two or more previous standard chemotherapy regimens (and without upper limit). The primary endpoint is PFS, assessed by independent central review (ICR). 468 patients without baseline brain metastases were enrolled. The mTNBC patients with disease-free interval less than 12 months after (neo)adjuvant chemotherapy would be counted as 1 prior treatment line. A heavily pre-treated patient population with a median number of 4 prior treatment lines (range, 2–17) were enrolled, including carboplatin and immune checkpoint inhibitors (ICIs) in 65.5% and 27.1% of patients without brain metastasis, respectively. The median progression-free survival was 5.6 months (95% CI, 4.3–6.3; 166 events) with SG and 1.7 months (95% CI, 1.5–2.6; 150 events) with chemotherapy (hazard ratio, 0.41; 95% CI, 0.32–0.52; P<0.001). The median overall survival was 12.1 months (95% CI, 10.7–14.0) with SG and 6.7 months (95% CI, 5.8–7.7) with chemotherapy (hazard ratio, 0.48; 95% CI, 0.38–0.59; P<0.001). The percentage of patients with an objective response was 35% with SG and 5% with chemotherapy. The median duration of response was 6.3 and 3.6 months in SG group and TPC group, respectively. This trial was completed earlier than originally planned upon recommendation by an independent Data Safety Monitoring Committee because compelling evidence of efficacy regardless of BC related biomarkers, like TROP-2 expression and germline BRCA mutations had shown SG had a superior performance than TPC. The treatment-related adverse events were consistent with those previously reported. Neutropenia, alopecia, anemia, nausea, diarrhea and fatigue were the main treatment-related adverse events. 46% patients had alopecia. Neutropenia (51%), diarrhea (10%), anemia (8%) and febrile neutropenia (6%) were the

Table 6 Summarized ongoing clinical trials in breast cancer with SG

Trial	Phase	Population	Regimen	Patients size	Recruitment status
Advanced breast cancer					
TROPiCS-02	III	HR+ mBC	SG	543	Active, not recruiting
EVER-132-001	II	mTNBC	SG	80	Active, not recruiting
Saci-IO TNBC	II	mTNBC	SG + pembrolizumab	110	Recruiting
Saci-IO HR+	II	HR+ mBC	SG + pembrolizumab	110	Recruiting
S2007	II	HER2-negative breast cancer and BM	SG	44	Recruiting
Morpheus-TNBC	I/II	mTNBC	SG + atezolizumab	280	Recruiting
SEASTAR	Ib/II	mBC	SG + rucaparib	329	Active, not recruiting
NCT04039230	I/II	mBC with BRCAm	SG + talazoparib	75	Recruiting
IMMU-132-14	IV	Solid tumor/metastatic cancer	SG	200	Enrolling by invitation
EVER-132-002	III	HR+ mBC	SG	330	Recruiting
Early-stage breast cancer					
NeoSTAR	II	eTNBC	SG + pembrolizumab	100	Active, not recruiting
SASCIA	III	$HER2^-$ eBC	SG	1200	Recruiting

mTNBC, metastatic triple-negative breast cancer; SG, sacituzumab govitecan; BM, brain metastasis.

most common grade 3–4 treatment-related adverse events. The summarized clinical results in BC are shown in *Table 5*.

Ongoing study

A series of studies focusing on the treatment of breast cancer is ongoing. For patients with advanced stage cancer, two ongoing phase III clinical studies aim to evaluate the treatment efficacy of SG and TPC in patients with advanced HR+ BC [TROPiCS-02 (NCT03901339), EVER-132-002 (NCT04639986)]. To explore the new combinational treatment, SG is under evaluation in combination with PD-1/PD-L1 inhibitors [Saci-IO TNBC (NCT04468061), Saci-IO HR+ (NCT04448886), Morpheus-TNBC (NCT03424005)] and PARPi [SEASTAR (NCT03992131), talazoparib (NCT04039230)]. For patients with early-stage breast cancer, SG is under phase III evaluation as post-neoadjuvant treatment in patients with early HER2- BC presenting with residual disease after neoadjuvant treatment (SASCIA, NCT04595565). A neoadjuvant therapy [NeoSTAR (NCT04230109)] aiming to evaluate the efficacy of SG combined with PD-1 in early stage triple negative breast cancer is ongoing. The summarized ongoing clinical trials in BC with SG are shown in *Table 6*.

ADCs have demonstrated significantly improved efficacy and manageable toxicities as a new therapeutic approach for breast cancer. With the rapid development of ADC technology, it deserves further exploration. SG is a novel promising Trop-2 targeting ADC with potent anti-cancer activity showing in multiple epithelial cancer types, especially in breast cancer. Clinical evidence from phase III confirmatory RCT trial has supported that SG would be the standard of care in patients with pretreated mTNBC.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://dx.doi.org/10.21037/tbcr-21-28). All authors are from Everest Medicines Ltd. and they have no other conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are

appropriately investigated and resolved.

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