GILEAD | Medical Information

Trodelvy® (sacituzumab govitecan-hziy) Use in Patients With Brain Metastases in mBC Studies

This document is in response to your request for information regarding the use of Trodelvy® (sacituzumab govitecan-hziy [SG]) in patients with brain metastases (BrM) in metastatic breast cancer (mBC) clinical studies.

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The full indication, important safety information, and boxed warnings for neutropenia and diarrhea are available at: www.gilead.com/-/media/files/pdfs/medicines/oncology/trodelvy/trodelvy_pi.

Summary

Relevant Product Labeling[1]

No information about whether SG crosses the blood-brain barrier is available in the SG US FDA-approved prescribing information.

Clinical Data on SG Use in Patients With BrM in mBC Studies

In the phase 3 ASCENT study, which evaluated safety and efficacy of SG in patients with mTNBC, 12% of patients had previously treated and stable BrM at baseline (BMPos: SG group, n=32; TPC group, n=29).[2]

- In a post hoc subgroup analysis in patients who were BMPos, the median (95% CI) PFS was 2.8 (1.5–3.9) mo in the SG group and 1.6 (1.3–2.9) mo in the TPC group. The safety profile among the subgroup of patients who were BMPos was consistent with the profile seen in the full ASCENT safety population.[3]

In TROPiCS-02, a phase 3 study of SG vs TPC in HR+/HER2- mBC, patients with previously treated BrM may participate if their BrM are stable for ≥4 wk. At this time, results in the stable BrM subgroup have not been reported.[4]

A phase 0/early phase 1 study evaluated the intratumoral concentrations and intracranial activity of SG in patients undergoing craniotomy for BCBM. In the BCBM cohort, most AEs were Grade 1 or 2. The median (range) PFS was 8 (2–26.5) mo. SN-38 levels showed penetration into intracranial tumor tissue.[5]

Real-World Data on SG Use in Patients With BrM

A real-world study evaluated the use of SG in 103 patients with mTNBC with a focus on BrM and toxicity and included patients with active or stable BrM (n=32, 31.1%) and impaired performance status. The median (95% CI) PFS was 3.7 (2.6–6.2) mo, and the median

(95% CI) OS was 6.7 (56.3–NR) mo in patients who were BMPos. ORR in the BMPos population was 19.8% (n=6).[6]

- In the BMPos population, 26 patients (81.2%) discontinued SG mostly due to disease progression. AEs that led to SG dose reduction were reported in 4 patients (12.5%), with no reports of SG-related death.[6]

A retrospective cohort study (N=98) investigated whether SRT for intact BrM used concurrently with an ADC, including SG, was associated with an increased risk of SRN. Concurrent ADC was associated with a higher risk of SRN in multivariable analyses (*P*<0.001) that also controlled for prior RT (*P*=0.01) and BrM volume (*P*<0.001). No ADC agent was associated with the risk of SRN (*P*=0.74).[7]

In a retrospective, observational study of SG in 33 patients with HER2- mBC and CNS metastases (treated/stable BrM, active BrM, and LMD), the overall median (95% CI) CNS and extra-CNS PFS was 2.9 (2–4.3) mo and 2.6 (1.9–4) mo, respectively. Three patients (TNBC, n=2; HR+/HER2-, n=1) had bicompartmental PFS that was >10 mo; each had surgery and brain RT. No safety data were reported.[8]

In a retrospective, multicenter study of SG in 29 patients with mTNBC and BrM, the median OS and PFS were 8.9 mo and 3.09 mo, respectively. There was no statistically significant association between the number or size of CNS lesions with OS or PFS. AEs resulted in dose delays in 15 patients (51.7%), dose reductions in 11 patients (37.9%), and discontinuations in 2 patients (7.7%).[9]

Ongoing Clinical Study

An open-label, phase 2 study (NCT04647916) is evaluating the efficacy and safety of SG in HER2- breast cancer that has metastasized to the brain.

Clinical Data on SG Use in Patients With BrM

ASCENT Study in mTNBC

The ASCENT study, a global, open-label, randomized, confirmatory, phase 3 study, was conducted to investigate efficacy and safety of SG vs chemotherapy TPC in patients with refractory or relapsed mTNBC who received ≥2 prior chemotherapies for unresectable, locally advanced, or metastatic disease.[2]

Patients with mTNBC (N=529) were enrolled and randomly assigned (1:1) to receive SG 10 mg/kg IV on Days 1 and 8 of a 21-d cycle (n=267) or single-agent TPC (n=262 [ie, eribulin, n=139; vinorelbine, n=52; gem, n=38; and cape, n=33]). Treatment was continued until the first of the following events: disease progression, unacceptable toxicity, discontinuation from the study, or death. The study protocol allowed a predefined maximum of 15% for patients with BrM for ≥4 wk. Patients with known BrM required baseline brain MRI/CT scans that were conducted every 6 wk for 36 wk and every 9 wk thereafter until disease progression led to study treatment discontinuation; confirmatory imaging was performed 4 to 6 wk after attaining responses.[2]

Post hoc subgroup analysis of patients with stable BrM[3]

A post hoc subgroup analysis evaluated the efficacy and safety of SG in patients who were BMPos, defined as having stable CNS disease for ≥4 wk by MRI, ≥2 wk from discontinuation

of antiseizure medication, and corticosteroid dose (≤20 mg prednisone equivalent) that was stable or decreasing for ≥2 wk. Among the patients (N=529) enrolled in the ASCENT study, 61 (12%) patients were BMPos (SG, n=32; TPC, n=29). From the SG and TPC groups, 2 (6%) and 6 (21%) patients, respectively, did not receive treatment. Baseline characteristics were generally balanced between the ITT and BMPos study populations (Table 1).

Table 1. ASCENT Subgroup Analysis in the ITT and BMPos Populations: Select Baseline Patient and Disease Characteristics[3]

Key Demographics and Characteristics	ITT (N=529)	BMPos (n=61)
Female, n (%)	527 (99)	61 (100)
Age, median (range), y	54 (27–82)	53 (27–81)
Race (self-reported), White, n (%)	418 (79)	49 (80)
ECOG PS, 0/1, n (%)	229 (43)/300 (57)	23 (38)/38 (62)
Number of prior chemotherapies, 2–3/>3, n (%)	365 (69)/164 (31)	35 (57)/26 (43)
Prior systemic regimens, median (range), n	4 (2–17)	5 (2–10)

Patient disease characteristics were generally similar between patients in the BMNeg and BMPos populations with a few exceptions.[3] *BRCA* mutations were reported in 15% vs 7% of patients who were BMPos and BMNeg, respectively. In patients who were BMPos and BMNeg, 82% vs 69% of patients were diagnosed with TNBC initially, the median (range) number of prior anticancer regimens was 4 (2–9) vs 3 (1–16), 43% vs 30% had >3 prior chemotherapies, 79% vs 65% had prior cape therapy, and 43% vs 27% had previous checkpoint inhibitors prior to enrollment, respectively. Compared to 44% and 22% of patients who were BMNeg, 67% and 36% of patients who were BMPos had major tumor locations in the lung and bone, respectively.

Efficacy

In the SG vs TPC group in the BMPos population, the median PFS (95% CI) was 2.8 (1.5–3.9) mo vs 1.6 (1.3–2.9) mo (HR, 0.65; 95% CI: 0.35–1.22), and the median OS (95% CI) was 7 (4.7–14.7) mo vs 7.5 (4.7–11.1) mo (HR, 0.96; 95% CI: 0.55–1.68). Response rates are presented in Table 2.

Table 2. ASCENT Subgroup Analysis in the BMPos Population: Response Rates (N=61)[3]

Efficacy Measure		SG (n=32)	TPC (n=29)
ORR, n (%)		1 (3)	0
Clinical benefit rate,[a] n (%)		3 (9)	1 (3)
Best overall response, n (%)	CR	0	0
	PR	1 (3)	0
	SD	15 (47)	9 (31)
	SD for >6 mo	2 (6)	1 (3)
	PD	11 (34)	11 (38)
	Not evaluable	5 (16)	9 (31)
DOR, median (95% CI), mo		2.9 (NE–NE)	N/A[b]
TTR, median (range), mo		1.5 (1.5–1.5)	0

Abbreviations: DOR=duration of response; NE=not estimable; TTR=time to response.
[a]Proportion of patients with a confirmed best overall response of CR or PR and SD for ≥6 mo.
[b]No patients to report.

Safety

The safety profile among the subgroup of patients who were BMPos was consistent with the profile observed in the full ASCENT safety population. In the BMPos subgroup, the most common (>20%) TEAEs of any grade were fatigue (63% vs 52%), neutropenia (63% vs 52%), diarrhea (50% vs 13%), nausea (43% vs 26%), decreased appetite (30% vs 17%), anemia (23% vs 35%), alopecia (23% vs 13%), and constipation (23% vs 22%) in the SG vs TPC groups, respectively.

TEAEs leading to treatment discontinuation in the BMPos subgroup occurred in 2 (7%) and 2 (9%) patients in the SG and TPC groups, respectively. In this subgroup, there were no reports of treatment-emergent death and 1 report of treatment-emergent death in the SG and TPC groups, respectively.

TROPiCS-02 Study in HR+/HER2- mBC

In TROPiCS-02, an open-label, randomized, multicenter, phase 3 study comparing the safety and efficacy of SG with TPC in patients with HR+/HER2- mBC who have received ≥2 and ≤4 prior chemotherapy regimens for metastatic disease, as well as ≥1 endocrine therapy, taxane, and cyclin-dependent kinase 4/6 inhibitor,[10] patients with previously treated BrM may participate provided they meet specific criteria, such as having clinically stable signs and symptoms for ≥4 wk, and more.[4]

At this time, the efficacy and safety of SG in the patient subgroup with stable BrM have not been reported.[10,11]

Prospective, Single-Center Study: SG in BCBM

A prospective, single-center, window-of-opportunity, phase 0/early phase 1 study evaluated intratumoral concentrations and intracranial activity of SG in patients (aged ≥18 y) undergoing craniotomy for BCBM or rGBM. Patients received a single IV dose of SG 10 mg/kg 1 d before surgical resection. Patients resumed treatment with SG 10 mg/kg on Days 1 and 8 of 21-d treatment cycles following recovery from surgery. Tumor and serum levels of SG, SN-38, and SN-38G were collected during surgery.[5]

In the BCBM cohort, 13 female patients were screened for inclusion and 1 patient failed screening; patients had a mean age (range) of 48.5 (33–70) y, 12 (93%) were White, 1 (7%) was Black/African American, 7 (54%) were HR+, 7 (54%) were HER2+, and 3 (23%) had TNBC.[5]

Preliminary safety and efficacy results

In the BCBM cohort, most AEs were Grade 1 or 2, which included diarrhea, neutropenia, nausea, hypokalemia, and fatigue.[5]

Efficacy outcomes in the BCBM cohort are shown in Table 3.

Table 3. Preliminary Efficacy Results for the BCBM Cohort[5]

	OS, Median (Range), Mo	PFS, Median (Range), Mo	iORR, %	iPR, %	iCR, %
BCBM	35.2 (2.7–37)[a]	8 (2–26.5)	50	25	25

Abbreviations: iORR=intracranial objective response rate, iPR=intracranial partial response, iCR=intracranial complete response.
[a]Median OS for HER2+ BCBM for patients with CNS metastasis at or after diagnosis was 30 to 38 mo, respectively, and was 12.5 mo for patients with HR+ BCBM.

Primary outcome: SN-38 levels and biomarker data

In the BCBM cohort, SN-38 levels showed penetration into intracranial tumor tissue (n=13, matching tissue and serum samples; n=3, CSF samples).[5,12] SN-38 levels, molarity, and minimum IC_{50} values are shown in Table 4.[5,12] Within the BCBM cohort, SN-38G was only detected in small amounts or not at all in the CSF.[5,12]

Table 4. Total SN-38 Levels, Molarity, and Minimum IC_{50} in Tumor Tissue, Serum, and CSF From Patients in the BCBM Cohort[5a]

Total SN-38 Tumor Tissue			Total SN-38 Serum			Total SN-38 CSF			Observed SN-38 Level, mcM	Minimum IC_{50}, mcM[b]
n	Level, Median (Range), ng/g	Molarity, mcM	n	Level, Median (Range), ng/mL	Molarity, mcM	n	Level, ng/mL	Molarity, mcM		
13	197.3 (86.5–652)	0.0523	13	2462.4 (1266.8–5659.6)	6.27	3	9.4[c]	0.035	0.662	0.000551

[a]Used brain tissue density of 1.04 g/mL. [b]Molecular weight=392.4 g/mol. [c]Median value range (5–26.5 ng/mL).

Additionally, several assays were evaluated to investigate potential mechanisms of action for SG in the CNS and included quantification of tumor expression for Trop-2 (a marker of antigen expression), γH2AX (a marker of DNA damage), and CAIX (a marker of intratumoral hypoxia).[5]

In the BCBM cohort, 12 samples were sufficient for Trop-2 analysis, and all had an H-score of 3+. Trop-2 expression did not correlate with % SN-38 tissue-to-serum ratio (Pearson r^2, 0.018; 95% CI: -0.23 to 0.81; *P*=0.18). Expression of γH2AX (n=11) ranged from 33.1% to 81.2% (mean, 54.4%), and 5 of the 11 samples indicated high CAIX expression.[5]

Real-World Data on SG Use in Patients With BrM

Real-World Study in mTNBC[6]

An ambispective, bicentric cohort study assessed the real-world efficacy and safety of SG in patients with mTNBC treated through the French Early Access Program (May 2021 to January 2023). Of the 103 patients enrolled, the median age (range) at baseline was 55 (26–89) y, 80.6% of patients had an ECOG PS of 0 to 1, 64.1% of patients had 1 to 2 prior lines of treatment in the advanced setting, and 31.1% (n=32) were BMPos.

In the BMPos population, the median (95% CI) PFS was 3.7 (2.6–6.2) mo, the median (95% CI) OS was 6.7 (56.3–NR) mo, and the ORR was 19.8% (n=6). SG treatment discontinuation was reported in 26 patients (81.2%) and was mostly due to disease progression. AEs that led to SG dose reduction were reported in 4 patients (12.5%), with no reports of death.

Retrospective Cohort Study: ADCs With SRT for BrM[7]

A retrospective cohort study (N=98) investigated whether SRT for intact BrM used concurrently with an ADC, including SG, was associated with an increased risk of SRN. Patients who had received ≥1 course of SRT for intact BrM between January 1, 2014, and October 24, 2022, and ≥1 dose of ADC were eligible for inclusion (Table 5). The control cohort consisted of patients with BrM who received ADCs sequentially with SRT. Results were pooled across ADCs, and SG-specific data were not provided.

Table 5. Select Baseline Demographics, Disease Characteristics, and ADC Received: Overall and With or Without Concurrent ADC Treatment[7]

Select Demographics and Characteristics and ADCs Received		Concurrent ADC[a] (n=42)	No Concurrent ADC (n=74)	All (N=98)
Age, median (range), y		54 (27–77)	55 (34–77)	55 (27–77)
Female, n (%)		33 (78.6)	66 (89.2)	82 (83.7)
Primary cancer diagnosis,[b] breast, n (%)		30 (71.4)	55 (74.3)	71 (72.4)
ADC,[c] n (%)	SG	7 (16.7)	23 (31.1)	26 (26.5)
	Trastuzumab emtansine	21 (50)	43 (58.1)	52 (53.1)
	Trastuzumab deruxtecan	14 (33.3)	42 (56.8)	50 (51)
Lesion volume, median (range), cm^3		0.3 (0.01–43.15)	0.25 (0.01–15.6)	0.26 (0.01–43.15)
Any prior radiation,[d] n/N (%)		20/156 (12.8)	81/408 (19.9)	101/564 (17.9)

[a]SRT was considered concurrent if delivered ≤7 d before or ≤21 d after ADC administration.
[b]Other cancers in the overall population included the following: non-small cell lung cancer, *ERBB2* variant (n=13); esophageal and/or gastric cancer, *ERBB2* amplified (n=6); salivary, *ERBB2* amplified (n=4); and colon, melanoma, urothelial, and unknown (each, n=1).
[c]ADCs given concurrently or sequentially with SRT. Numbers total >100% and do not total across rows as patients could have received ≥1 course of ADC.
[d]n/N refers to the number of lesions. Lesions could have been treated with >1 type of prior RT.

Results

The median (IQR) time from SRT to last imaging was 12.4 (0–80.4) mo. Across the entire cohort, the 24-mo cumulative incidence (range) of SRN was 8.5% (6.2–11%). The use of concurrent ADC was associated with a higher risk of SRN in univariable and multivariable analyses that also controlled for prior RT and BrM volume (Table 6). For previously radiated lesions in patients with and without concurrent ADC therapy, the 24-mo risk of SRN was 42% and 9.4%, respectively. Among patients with BrM who were treated with concurrent ADC therapy, no ADC agent was associated with the risk of SRN (*P*=0.74). Grade 4 to 5 SRN after SRT was observed in 11 of 156 (7.1%) and 3 of 408 (0.7%) lesions treated with and without concurrent ADC, respectively.

Table 6. Risk of SRN With Concurrent ADC[7]

Concurrent ADC (n=42)	Subdistribution HR (95% CI)	*P*-Value
Univariable analysis	4.01 (1.79–9.01)	<0.001
Multivariable analysis	4.31 (1.95–9.5)	<0.001
Prior RT	2.99 (1.26–7.09)	0.01
BrM volume, per cm^3	1.14 (1.09–1.19)	<0.001

Retrospective, Observational Real-World Study[8]

A retrospective, observational real-world study evaluated clinical outcomes with SG (≥1 dose of SG between 2018 and 2022) treatment in patients with HER2- mBC and CNS metastases. Of the 33 patients included, 23 and 10 had TNBC and HR+/HER2- mBC, respectively. Overall, 18 patients had treated/stable BrM (defined as lesions that had been previously treated with CNS-specific therapies), 7 had active BrM (defined as those with new BrM or progressed BrM that had not been treated with CNS-specific therapies since progression), and 8 had LMD (defined as metastases in the leptomeningeal space with or without BrM). One patient had CNS metastases without extra-CNS involvement. Additional baseline characteristics are shown in Table 7.

Table 7. Select Baseline Demographics and Disease Characteristics: Overall and by Subtype[8]

Select Demographics and Disease Characteristics		Overall (N=33)	Treated/Stable BrM (n=18)	Active BrM (n=7)	LMD (n=8)
Age at start of SG, median (range), y		56.7 (51.3–65.7)	59.59 (42.9–75.1)	59.1 (52.9–72.8)	50.88 (41.4–68.3)
Diagnosis of BrM to SG, median (range), mo		7.5 (0.6–94.8)	4.6 (0.7–94.8)	9.3 (0.6–21)	9.3 (2.7–21.5)
HER2 status at SG start, n (%)	HER2-0	22 (66.7)	9 (50)	7 (100)	6 (75)
	HER2-low	11 (33.3)	9 (50)	0 (0)	2 (25)
Metastatic lines prior to SG, median (range), n		3 (0–10)	3 (0–9)	5 (2–10)	3.5 (2–6)

Results

The overall median (95% CI) duration of follow-up was 6.7 (3.1–10) mo; responses were evaluable in 30 patients in the CNS subgroup and 29 patients in the extra-CNS subgroup. The CNS PFS was 2 (1.2–7.7) mo and 3.2 (2.2–5.1) mo in patients with HR+/HER2- and TNBC, respectively. In patients with >3 and ≤3 prior lines, the CNS PFS was 2.7 (1.2–4.8) mo and 3.2 (2–7.1) mo, respectively; additional results are found in Table 8. The CNS ORR by mBC type was as follows: HR+/HER2-, 11.1%; TNBC, 14.3%.

Table 8. Outcomes Overall and by Subgroup[8]

Response, n (%)	Overall (n=30)		Treated/Stable BrM (n=16)		Active BrM (n=7)		LMD (n=7)	
	CNS	Extra-CNS[a]	CNS	Extra-CNS[b]	CNS	Extra-CNS	CNS	Extra-CNS
Response[c]	4 (13.3)	1 (3.4)	2[d] (12.5)	0	0	0	2 (28.6)	1 (14.3)
SD	16 (53.3)	12 (41.4)	10 (62.5)	6 (40)	3 (42.9)	4 (57.1)	3 (42.9)	2 (28.6)
PD	10 (33.3)	16 (55.2)	4 (25)	9 (60)	4 (57.1)	3 (42.9)	2 (28.6)	4 (57.1)
DCR	20 (66.7)	13 (44.8)	12 (75)	6 (40)	3 (42.9)	4 (57.1)	5 (71.4)	3 (42.9)
CBR at 6 mo	8 (26.7)	4 (13.8)	6 (37.5)	2 (13.3)	1 (14.3)	1 (14.3)	1 (14.3)	1 (14.3)
PFS,[e] median (95% CI), mo	2.9 (2–4.3)	2.6 (1.9–4)	3.4 (2.2–10)	2.7 (1.9–4.2)	1.9 (1.2–16.5)	1.9 (1.5–10.1)	2.1 (0.4–7.7)	2 (0.4–6.9)
OS, median (95% CI), mo	6.9 (3.1–10.2)		10 (4.3–15.9)		3.1 (1.9–21.6)		3.8 (1.7–11.9)	

Abbreviation: DCR=disease control rate.
[a]n=29. [b]n=15. [c]CR or PR for CNS response. [d]Both received RT <2 mo before SG began.
[e]Bicompartmental median (95% CI) PFS by subgroup was as follows: overall, 2.6 (1.9–4) mo; treated/stable BrM, 2.7 (1.9–4.1) mo; active BrM, 1.8 (1.5–10.1) mo; LMD, 2.4 (0.4–5.1) mo.

Overall, 32 patients discontinued treatment due to disease progression (53.1% had both CNS and extra-CNS progression), and 1 discontinued SG due to toxicity; 5 patients died during SG treatment.

Three patients had bicompartmental PFS that was >10 mo; each had surgery and brain RT. Two of these patients had TNBC with stable BrM and received SG early (≤2 prior therapies); the third patient with HR+/HER2- mBC had 95 mo between BrM diagnosis and SG initiation.

No safety data were reported.

Retrospective, Multicenter Study in mTNBC and BrM[9]

A retrospective, multicenter study evaluated effectiveness and safety in patients with mTNBC and radiologically confirmed BrM treated with SG at centers in the Czech Republic,

Poland, and Slovakia between August 2021 and May 2025. Of the 29 patients included in the analysis, the median (IQR) age was 48.5 (42.1–58) y; the median number (range) of prior palliative systemic therapy lines was 2 (1–3); 24.1%, 72.4%, and 3.4% had ECOG PS scores of 0, 1, and ≥2, respectively. Overall, 89.7% had invasive ductal histological subtype, and 3.4% each had invasive lobular, metaplastic, and secretory subtypes; 51.7%, 27.6%, 6.9%, and 13.8% had HER2 IHC of 0, 1+, 2+, and unknown status, respectively; 13.8% had a history of ER- or PgR+ BC and 6.9% had a history of HER2+ BC; 24.1% had a history of *BRCA1* or *BRCA2* mutation; and 72.4% had a history of breast RT. The median (range) number of BrMs was 2 (1–20), and the median (range) size of the biggest lesion was 16.5 (2–50) mm. Four patients (14.3%) had neurosurgical resection and 28 (89.3%) had CNS-directed RT. In the palliative setting, the most common (≥20%) prior treatments included the following: platinum compounds (63.3%), taxanes (50%), anthracyclines (43.3%), cape (30%), gem (26.7%), and pembrolizumab (20%).

Results

After a median (IQR) follow-up of 6.05 (3.66–11.66) mo, 22 patients had died and 26 had stopped treatment with SG. The median OS was 8.9 mo, and estimated OS rates at 3, 6, 9, and 12 mo were 85.2%, 60.3%, 42.3%, and 28.2%, respectively. The median PFS was 3.09 mo, and PFS rates at 3, 6, 9, and 12 mo were 53.6%, 31.4%, 22.5%, and 12%, respectively. Of the 26 patients with evaluable data, the ORR was 30.8% (n=8). CNS progression occurred in 23.1% of patients (n=6), and per univariable Cox proportional hazards model, there was no statistically significant association between the number or size of CNS lesions with effectiveness outcomes (OS and PFS; *P*-value not provided).

No new safety signals were reported. AEs resulted in dose delays in 15 patients (51.7%) and dose reductions in 11 (37.9%). SG was discontinued due to toxicities in 2 patients (7.7%).

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Abbreviations

γH2AX=phosphorylated form of histone H2AX
ADC=antibody drug conjugate
AE=adverse event
BCBM=breast cancer brain metastases
BMNeg=negative for brain metastasis
BMPos=positive for brain metastasis
BrM=brain metastases
CAIX=carbonic anhydrase IX
cape=capecitabine
CNS=central nervous system
CR=complete response
CSF=cerebrospinal fluid
ECOG PS=Eastern Cooperative Oncology Group performance status
ER=estrogen receptor
ERBB2=HER2 mutation
extra-CNS=lesions or disease outside of the central nervous system
gem=gemcitabine
H-score=histochemical scoring
HER2-=human epidermal growth factor receptor 2-negative
HR=hazard ratio
HR+=hormone receptor-positive
IC_{50}=half maximal inhibitory concentration
IHC=immunohistochemistry
LMD=leptomeningeal disease
mBC=metastatic breast cancer
mTNBC=metastatic triple-negative breast cancer
NR=not reported
ORR=objective response rate
OS=overall survival
PD=progressive disease
PFS=progression-free survival
PgR=progesterone receptor
PR=partial response
rGBM=recurrent glioblastoma
RT=radiotherapy
SD=stable disease
SG=sacituzumab govitecan-hziy
SN-38=active metabolite of irinotecan; topoisomerase I inhibitor
SN-38G=glucuronidation metabolite of SN-38
SRN=symptomatic radiation necrosis
SRT=stereotactic radiotherapy
TNBC=triple-negative breast cancer
TPC=treatment of physician's choice
TEAE=treatment-emergent adverse event
Trop-2=trophoblast cell surface antigen 2

Product Label

For the full indication, important safety information, and boxed warning(s), please refer to the Trodelvy US Prescribing Information available at: www.gilead.com/-/media/files/pdfs/medicines/oncology/trodelvy/trodelvy_pi.

Follow-Up

For any additional questions, please contact Trodelvy Medical Information at:

☎1-888-983-4668 or www.askgileadmedical.com

Adverse Event Reporting

Please report all adverse events to:

Gilead Global Patient Safety ☎ 1-800-445-3235, option 3 or
www.gilead.com/utility/contact/report-an-adverse-event

FDA MedWatch Program by ☎ 1-800-FDA-1088 or ✉MedWatch, FDA, 5600 Fishers Ln, Rockville, MD 20852 or www.accessdata.fda.gov/scripts/medwatch

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