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[^{18}F]Fluorodeoxyglucose-PET/MRI-based response assessment following BCMA-directed CAR-T-cell therapy with ciltacabtagene autoleucel in relapsed/refractory multiple myeloma

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Contributions

RT, KTG, RW and MM conceptualized and designed the study. RT, KTG, LK, KE, SB, HW, RW and MM collected and interpreted data, AG and IG provided statistical input, RT, KTG, RW wrote the manuscript. All authors reviewed and approved the final manuscript.

Data-sharing statement

The data that support the findings of this study are available from the corresponding author upon request.

Disclosures:

RT received consulting and/or lecture fees from AbbVie, Amgen, BMS, Gilead, GSK, Johnson & Johnson, Oncopeptides, Pfizer, Sanofi, Stemline and Takeda, received research grants from Johnson & Johnson; KTG received Honoraria from Amgen, Novartis, Grifols, Roche, Sanofi, SOBI, Takeda and served in advisory boards from Amgen, Grifols, Novartis, Novonordisk, SOBI, Sanofi; MM reports consulting fees from Novartis and Roche; payment or honoraria for Telix and Novartis. All outside the submitted work.

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Letter to the editor

Chimeric Antigen Receptor (CAR)-T-cell therapy has demonstrated remarkable efficacy in patients with relapsed or refractory multiple myeloma (RRMM), inducing high response rates even in heavily pretreated patients.[1] Several diagnostic modalities are currently used for response assessment, including classical serologic evaluation of monoclonal proteins, bone marrow–based assessment of minimal residual disease (MRD), and functional imaging such as PET. However, it remains unclear how treatment response after CAR-T-cell therapy should best be evaluated, as conventional serologic assessments and bone marrow MRD testing alone are unlikely to adequately capture the depth, spatial heterogeneity, and kinetics of response induced by cellular immunotherapy, especially in patients with extramedullary disease (EMD). In this context, the role of PET-based diagnostics continues to be investigated. Still, data on PET-based response assessment after CAR-T-cell therapy remain limited in multiple myeloma, particularly with regard to its optimal application, timing, and added value compared to other available response assessment methods. PET/MRI is an evolving technology with promising results mainly in newly diagnosed MM[2], but data on its use before and after CAR-T-cell therapy are lacking.

To specifically address the current knowledge gap regarding PET/MRI-based response assessment in ciltacabtagene autoleucel (cilta-cel) recipients, we retrospectively evaluated RRMM patients exclusively treated with cilta-cel, eliminating the confounding prognostic heterogeneity introduced by different CAR-T-cell constructs. A particular focus was placed on the prognostic impact and outcome of patients with EMD.

A total of 26 patients undergoing CAR-T-cell therapy with cilta-cel from December 2023 until April 2025 at an academic hospital in Germany were included. Per institutional standard, whole-body [^{18}F]Fluorodeoxyglucose-PET/MRI was performed prior to CAR-T-cell therapy, at 2 months, and at ≥12 months post cilta-cel in all patients with respective follow-up and without prior clinical or serological relapse. Additionally, a standardized flow-cytometry–based MRD assessment[3] (sensitivity: 10^{-5}) was performed 2 and 8–12 months post cilta-cel. Serological response assessments according to IMWG criteria[4] were conducted at 2, 6, 8, and 12 months after cilta-cel.

PET/MRI scans were assessed by two nuclear medicine specialists and radiologists using the IMPeTUs criteria[5], focusing on Deauville score (DS), bone marrow patterns, focal lesions, and extent of EMD. A methodological limitation is that MRI does not allow reliable assessment of

CT-based osteolytic lesions (including their progression) or fractures. Diffuse myeloma involvement was defined as homogenous uptake above liver threshold (PET) or diffuse increased signal on high b-value images (DW-MRI). For evaluation of therapeutic response, complete metabolic response (CMR) was defined as a DS <4 in all focal lesions and bone marrow, whereas a persistent DS ≥4 was considered non–complete metabolic response (Non-CMR), as previously described.[6] Based on PET/MRI findings, patients were categorized as either having bone-restricted (intraosseous and/or paraskeletal) lesions only (noEMD; n=17), or exhibiting extramedullary involvement not adjacent to bone (EMD; n=9). The study was approved by the institutional review board and local ethics committee (BO-EK-57022024).

The median patient age was 63 years at the time of cilta-cel infusion. Patients were heavily pretreated (median of 4 prior lines of therapy). All patients were triple-class exposed and 19/26 (73%) of patients were triple-class refractory. A significant proportion of patients (73%) had cytogenetic high-risk disease (Table 1).

In total, 22/26 patients were evaluable by serology, while 4 patients had oligo-/asecretory disease and could not be evaluated accordingly. All patients with measurable disease (n=22) achieved an objective serological response at month +2 (≥CR=12; VGPR=7; PR=3), with deepening of response over time (Figure 1A). Bone marrow assessment at month +2 showed a high rate of early MRD negativity (MRDneg) (24/26 patients; 92%), with 11/26 patients achieving MRDneg and CR. Of 24 patients with MRDneg at month +2, 17 were evaluable for sustained MRD negativity (>6 months), which maintained in 13 patients. Three patients developed early progressive disease, and one converted to MRD positivity (MRDpos) at the second assessment. Seven patients were excluded due to immature follow-up (n=6) or refusal (n=1). Of the two patients, with MRDpos at month +2 one converted to MRDneg, while the other patient experienced a clinical relapse 7 months post cilta-cel (Figure 1B/D).

Diffuse infiltration at baseline was detected in 5 patients by PET compared to 10 patients by DW-MRI. All diffuse manifestations detected by PET displayed also diffuse uptake in MRI. Discrepancies between the two modalities were either due to diffuse uptake in PET not extending above liver threshold or inhomogeneous, micronodular signal in MRI classified as diffuse uptake. At month +2 diffuse uptake consistently resolved for all patients in PET and in all but one patient in DW-MRI.

Further, 19/26 patients (73%) showed CMR on PET/MRI at month +2, whereas in 7/26 patients at least one lesion (average of 1,7 +/- 0,76) with persistent increased metabolic uptake (Non-

CMR) was detectable. All except one lesion were classified as DS 4 (one lesion DS 5) on PET. On DW-MRI, all of the lesions were also classified as responders (MY-RADS 1 or 2), except the one lesion with persistent DS 5 in PET. Of the 7 patients with Non-CMR at month +2, 5 converted to CMR over time, whereas 2 patients experienced clinical relapse, both of whom had EMD prior to CAR-T-cell therapy (Figure 1C/D). All patients with conversion to CMR at a later time point remained in ongoing complete remission at last follow up. This raises further questions regarding the optimal time point and interpretation of early PET-based response assessment after CAR-T-cell therapy in multiple myeloma. The high rate of later conversion to CMR in our cohort might indicate that month +2 is not the optimal time point for PET evaluation alone, as it might be too early to sufficiently distinguish between residual metabolic uptake and clinically relevant active myeloma. Here, the addition of DW-MRI might help for further distinction. In previous reports different potential reasons for persistent residual metabolic uptake such as spatially heterogeneous delayed clearance, or inflammatory and immune-mediated flare-up phenomena after BCMA-directed therapy have been discussed.[7,8] This could be particularly relevant to avoid unnecessary intensification of therapy, for example through consolidative radiotherapy of residual lesions.

Another key area of interest is the evaluation of potential biomarkers and their prognostic impact on outcomes after CAR-T-cell therapy. Recent studies evaluated the role of PET-based metabolic tumor volume (MTV) and soluble B-cell maturation antigen (sBCMA) in mixed cohorts treated with cilta-cel, ide-cel, or an academic CAR-T-cell construct.[9–11] High MTV, defined by different thresholds, was reported as a negative prognostic factor in two studies. Furthermore, elevated sBCMA levels were associated with high baseline tumor burden and inferior outcomes.

We assessed PET-based MTV using PERCIST 1.0 liver reference normalization with automated lesion detection. Levels of sBCMA were measured by ELISA (R&D®).

Serum levels of sBCMA before cilta-cel infusion correlated well with pre–CAR-T-cell MTV ($R=0.72$; $p<0.001$; Supplementary figure 1A). Furthermore, we observed a strong correlation between the change in MTV from baseline to month +2 after CAR-T-cell therapy and the change in sBCMA over the same period ($R=0.84$; $p<0.001$; Supplementary Figure 1B). Baseline MTV also correlated with the rate of serological CR ($p=0.008$), but not with CMR ($p>0.9$) at month +2 and it did not correlate with outcome (Supplementary figure 2C/D). This supports the potential role of sBCMA and MTV as biomarkers of tumor burden in this setting. These

findings could be particularly relevant in patients with high tumor burden, especially patients with EMD.

EMD is a well-established adverse prognostic factor in multiple myeloma, particularly in the context of conventional therapies. Although early trials of BCMA-directed CAR-T-cell therapies demonstrate superior efficacy over standard treatments, published data on their use in patients with EMD remain sparse. Available evidence suggests that, whilst response rates are higher than with conventional therapy, outcomes for patients with extramedullary involvement are still markedly inferior to those without such disease.[12,13] Cilta-cel is presumably the most effective approved CAR-T-cell in myeloma and can also lead to durable responses in patients with high-risk features such as adverse cytogenetics and triple-class refractory status.[1415] Nevertheless, its efficacy in patients with EMD remains insufficiently defined.

To further assess the influence of extramedullary disease, the cohort was divided into noEMD vs. EMD patients. Baseline MTV (p=0.047) and levels of sBCMA (p=0.038) were significantly higher in patients with EMD. Further patients with EMD had a higher number of lesions per patient (p=0.021).

When comparing response patterns, patients without EMD achieved higher rates of early serological ≥CR (63% vs. 33%) as well as higher rates of CMR (82% vs. 56%) at month +2. However, these differences were not statistically significant based on the given sample size. In contrast, comparison of bone-marrow MRD-levels revealed similarly high rates of MRDneg at month +2 in both groups (93% vs. 92%).

After a median follow up of 12 months (range=5.8-23.2) patients with EMD prior to CAR-T-cell therapy showed a worse PFS (12-month-PFS=65% in EMD-group versus 94% in noEMD-group, p=0.044), while OS did not reach significance (12-month-OS=88% in EMD-group versus 93% in noEMD-group, p=0.37). Comparing specific response patterns of EMD lesions, MRI showed reduction of EMD lesion size at month +2, with persistent measurable disease in most patients, whereas PET demonstrated CMR in all but one patient despite residual morphology. Relapses after CAR-T in the EMD group occurred at previously involved EMD in all patients (Supplementary table 1).

Our data, derived from a small but well-defined and uniformly treated RRMM patient cohort, provide detailed insight into multimodal and longitudinal response assessments after CAR-T-cell therapy including preliminary data on PET/MRI-based imaging. Nevertheless, given the

limitations of a small, single-center cohort, the results of this exploratory analysis should be considered in context and require confirmation in larger, prospective cohorts to further determine the use of PET/MRI and its optimal timing after CAR-T-cell therapy. Our findings suggest that EMD may remain an adverse prognostic factor even in cilta-cel treated patients and indicate, that PET imaging prior to CAR-T-cell therapy and the incorporation of other biomarkers like sBCMA could add essential information for prognostic stratification. This may facilitate the implementation of more effective bridging and debulking strategies to further improve outcomes in this difficult-to-treat patient population.

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Table 1: Patient and PET/MRI characteristics at baseline

Baseline patient characteristics	N = 26
Age at CAR-T infusion (median, range)	63 (41-74)
Sex, n (%) • Male • Female	 14/26 (54%) 12/26 (46%)
Prior lines of therapy (median, range)	4 (2-9)
R-ISS-Score at CAR-T, n (%) • I • II • III	 10/26 (38%) 9/26 (35%) 7/26 (27%)
Triple class exposed, n (%)	26/26 (100%)
Triple class refractory, n (%)	19/26 (73%)
Prior autologous SCT, n (%)	24/26 (92%)
Cytogenetic high-risk *, n (%)	19/26 (73%)
Time from primary diagnosis to CAR-T infusion in months (median, range)	57 (15 - 205)
Baseline PET/MRI characteristics (prior to CAR-T)	
Diffuse bone marrow uptake per modality, n (%) • PET • DW-MRI	 5/26 (19%) 10/26 (38 %)
Number of metabolic active lesions per patient, median (range)	5 (0-17)
Localization of lesions, patients in n (%) • Bone-restricted (intraosseous and/or paraskeletal) • Extramedullary (not adjacent to bone)	 17/26 (65%) 9/26 (35%)
Metabolic Tumor Volume (MTV) in cm^3, median (range)	8 (0 - 1.430)

sBCMA in ng/ml, median (range)	18 (2 - 186)

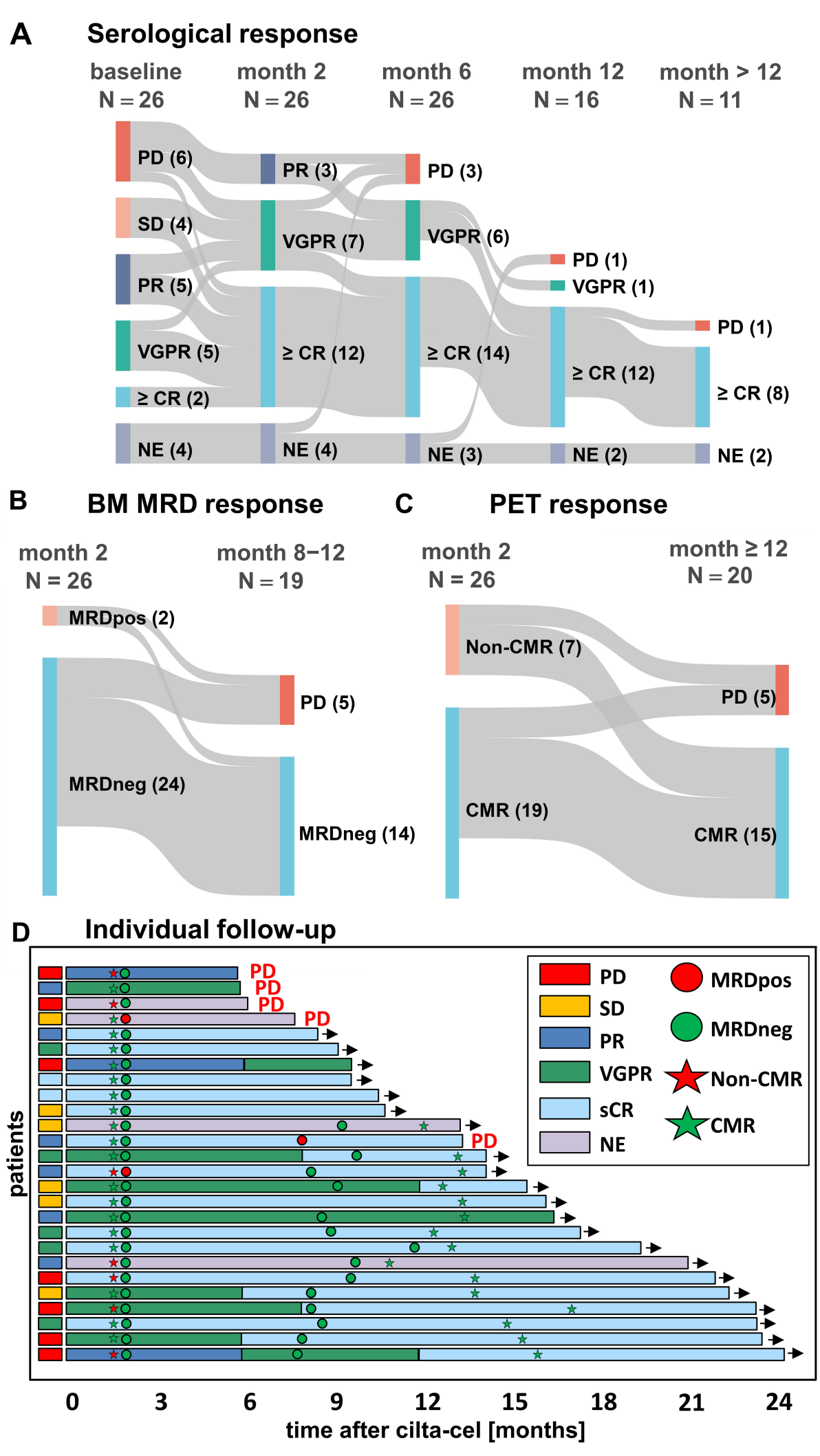
*Defined as presence of one or more: del17p13; t(4;14); t(14;16); gain/amp1q21

Abbr.: CAR-T - chimeric antigen receptor T-cell; EMD – extramedullary disease; [^{18}F]FDG – fluorodeoxyglucose; MRI - magnetic resonance imaging; MTV – metabolic tumor volume; PET - positron emission tomography; R-ISS – Revised International Scoring System; sBCMA – soluble b-cell maturation antigen; SCT – stem cell transplantation

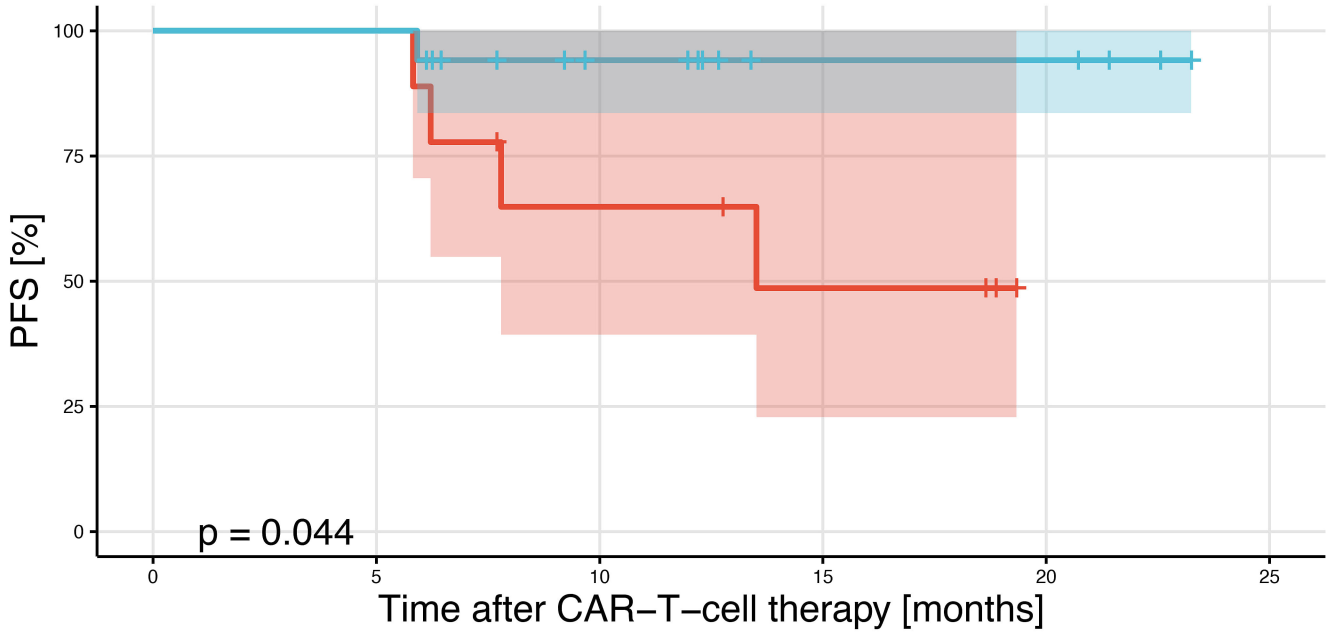
Legend to figures:

Figure 1. **Different patterns of response over time**. (A) Longitudinal serologic response after CAR-T-cell. (B) Bone marrow MRD-status (sensitivity-level: 10^{-5}) at months +2 and at months +8-12 post cilta-cel. (C) PET response measured by [^{18}F]FDG-PET/MRI at months +2 and at ≥ 12 months post cilta-cel. Patients with progressive disease (PD) after cilta-cel are illustrated in each figure irrespective to the modality defining progression. Response categories are defined by IMWG-criteria: PD= progressive disease; SD= stable disease; PR= partial response; VGPR= very good partial response; ≥ CR= Complete response (includes stringent CR and CR); NE= Non evaluable. (D) Patient individual follow-up. Colored bars prior to cilta-cel infusion (time point "0") represent the overall response (imaging and serology) to bridging therapy (all patients received bridging therapy). After car-t cell infusion, colored bars indicate serological response categories according to IMWG-criteria. PET/MRI assessments are indicated by stars (red star= Non-CMR; green star= CMR). Bone marrow MRD assessments are indicated by dots (red dot= MRDpos; green dot= MRDneg). An arrow indicates ongoing response at the time of data cut-off. Progression ("PD") is shown irrespective to the modality defining progression (imaging, serological or clinical).

Figure 2. **PFS and OS in EMD vs. noEMD patients**. (A) Progression-free survival in patients with EMD (red curve) vs. patients without EMD (blue curve). (B) Overall survival in patients with EMD (red curve) vs. patients without EMD (blue curve). Survival analyses were performed using the Kaplan-Meier estimator for survival curve estimation, and group differences were assessed using the log-rank test.


A
Serological response
baseline
N = 26
month 2
N = 26
month 6
N = 26
month 12
N = 16
month > 12
N = 11
PD (6)
SD (4)
PR (5)
VGPR (5)
≥ CR (2)
NE (4)
PR (3)
VGPR (7)
≥ CR (12)
NE (4)
PD (3)
VGPR (6)
≥ CR (14)
NE (3)
PD (1)
VGPR (1)
≥ CR (12)
NE (2)
PD (1)
≥ CR (8)
NE (2)
B
BM MRD response
month 2
N = 26
month 8−12
N = 19
MRDpos (2)
MRDneg (24)
PD (5)
MRDneg (14)
C
PET response
month 2
N = 26
month ≥ 12
N = 20
Non-CMR (7)
CMR (19)
PD (5)
CMR (15)
D
Individual follow-up
PD
SD
PR
VGPR
sCR
NE
MRDpos
MRDneg
Non-CMR
CMR
patients
0
3
6
9
12
15
18
21
24
time after cilta-cel [months]

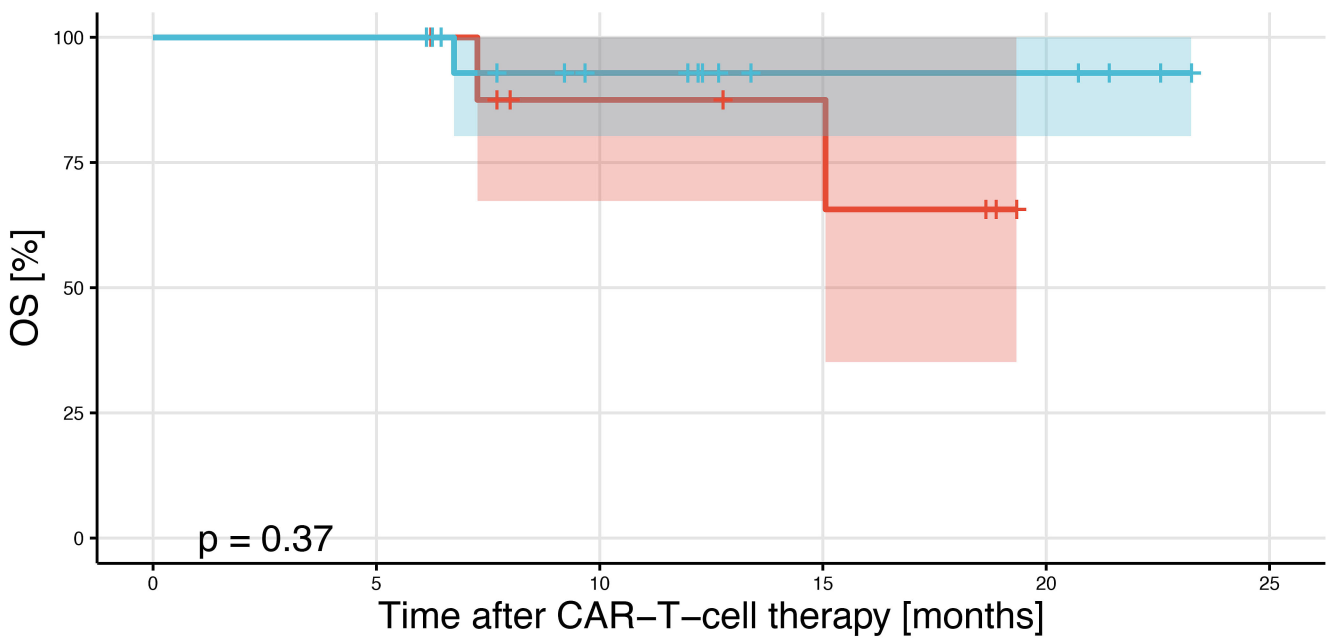
A



Number at risk

EMD	9	5	3	0	0
noEMD	17	10	4	4	0

B

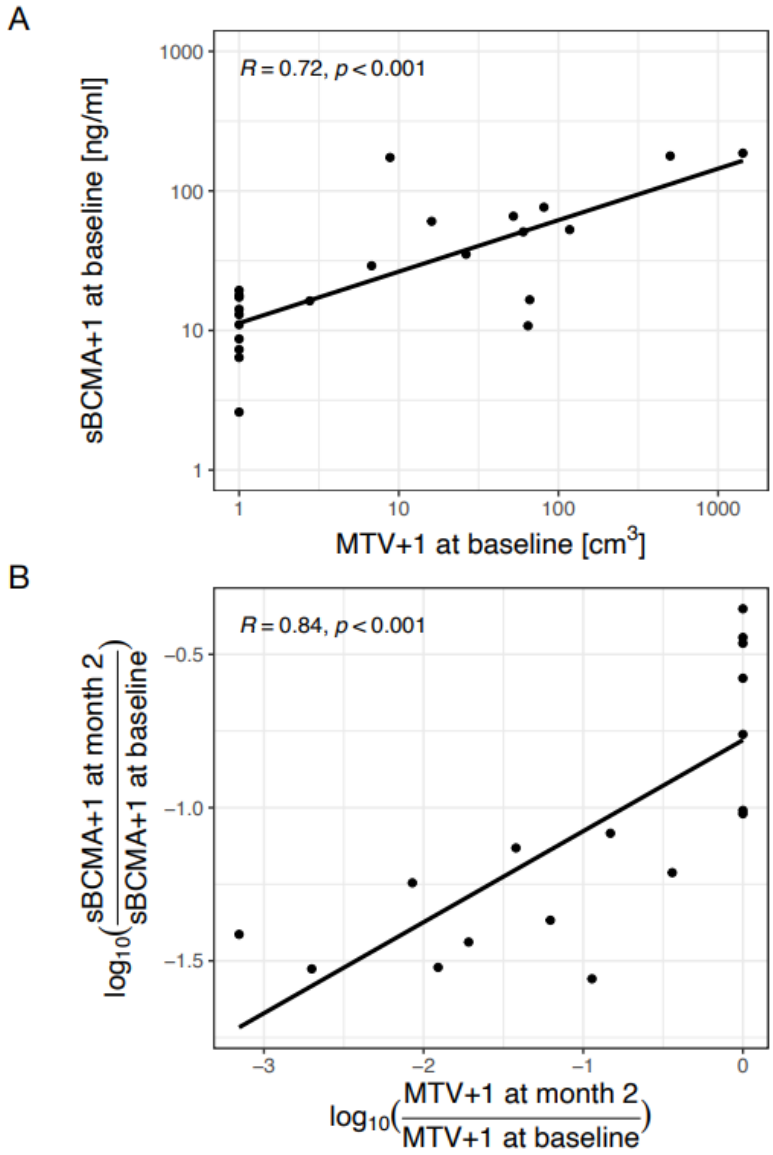


Number at risk

EMD	9	5	4	0	0
noEMD	17	10	4	4	0

Supplementary data:

Supplementary figure 1

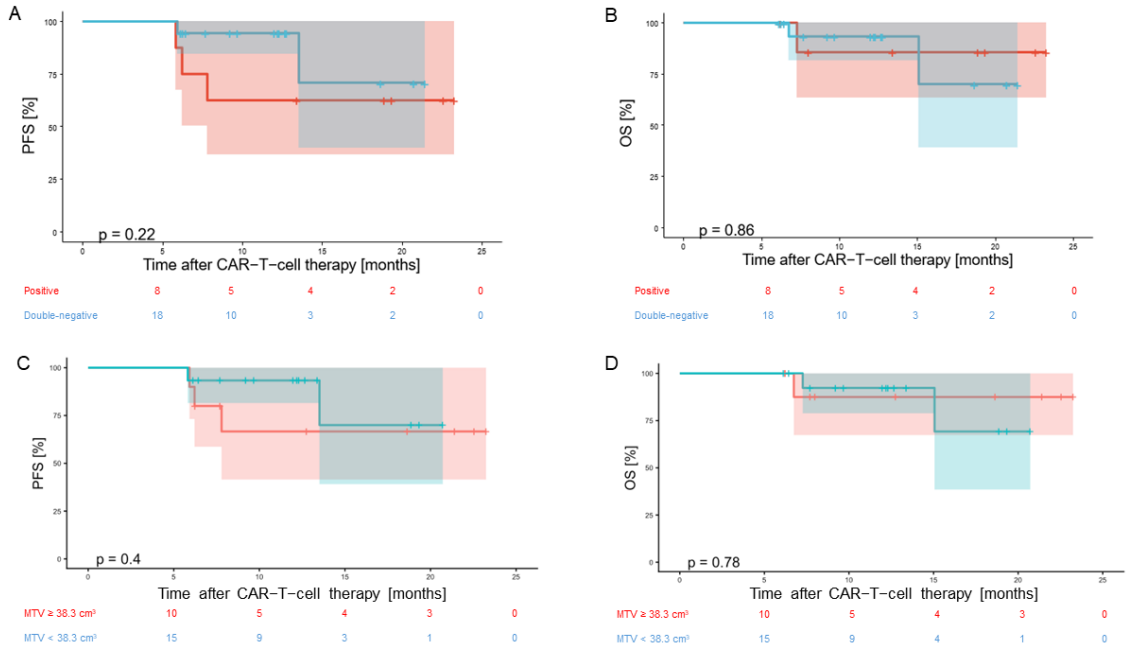


Legend to supplementary figure 1: Correlation of sBCMA and MTV before and after cilta-cel (A) Correlation of sBCMA and MTV at baseline (before cilta-cel). (B) Correlation of the change in sBCMA-levels and MTV-levels from baseline to month +2 after cilta-cel. At month +2, MTV levels were undetectable (0) in 25 out of 26 patients.

Spearman's rank correlation coefficient was used to assess associations. To enable visualization of log-transformed data, a value of 1 was added to both sBCMA and MTV

prior to $\log_{10}$ transformation. This adjustment does not affect the Spearman correlation results.

Supplementary figure 2



Legend to supplementary figure 2: PFS and OS dependent on MRD-status and baseline MTV (A) Progression-free survival in patients with MRDneg (bone marrow) and CMR (PET) at month 2 (blue curve) vs. patients with either MRDpos (bone marrow) and/or Non-CMR (PET) at month 2 (red curve). (B) Overall survival in patients with MRDneg (bone marrow) and CMR (PET) at month 2 (blue curve) vs. patients with either MRDpos (bone marrow) and/or Non-CMR (PET) at month 2 (red curve). (C) Progression-free survival in patients with baseline MTV ≥ 38.3 cm^3 (red curve) vs. baseline MTV < 38.3 cm^3 (blue curve). (D) Overall survival in patients with baseline MTV ≥ 38.3 cm^3 (red curve) vs. baseline MTV < 38.3 cm^3 (blue curve). Survival analyses were performed using the Kaplan-Meier estimator for survival curve estimation. The cut-off for MTV (38.3 cm^3; median specificity: 70%; median sensitivity: 90.3 %) was calculated by ROC-analysis.

Supplementary table 1

Sites and response patterns of EMD lesions

Patient	EMD sites at baseline	Metabolic response of EMD lesions at month 2	Morphologic response of EMD lesions at month 2	EMD sites at relapse after CAR-T
#2	pleura	CMR	PR	
#4	pancreas	CMR	PR	pancreas
#11	mediastinum	Non-CMR	SD	
#12	liver, muscle, spleen	CMR	CR	
#17	peritoneum, corpus cavernosus penis	CMR	PR	
#22	pleura	CMR	CR	pleura
#23	lymphnodes, peritoneum, pleura, liver, kidney, adrenal glands	CMR	PR	pleura
#25	muscle	CMR	PR	
#26	retrocrural space	CMR	CR	retrocrural space

Abbr.: EMD – extramedullary disease; CMR – complete metabolic response; Non-CMR – Non complete metabolic response; CR – complete response; PR – partial response; SD – stable disease