haematologica
Journal of the Ferrata Storti Foundation

Anti-programmed cell death protein 1-based salvage therapy for relapsed/refractory Hodgkin lymphoma: a multicenter real-world analysis

by Hishan Tharmaseelan, Lisa M. Sgonina, Ina Bühnen, Ulf Schnetzke, Julia Meißner, Markus Sellmayr, David Beverungen, Johannes C. Hellmuth, Joe Puthenparambil, Tanja Zamrik, Justin Ferdinandus, Evgenii Shumilov, Andrea Kerkhoff, Thomas Melchardt, Paul J. Bröckelmann, Carsten Kobe, Peter Borchmann and Bastian von Tresckow

Received: August 18, 2025.
Accepted: February 18, 2026.

Citation: Hishan Tharmaseelan, Lisa M. Sgonina, Ina Bühnen, Ulf Schnetzke, Julia Meißner, Markus Sellmayr, David Beverungen, Johannes C. Hellmuth, Joe Puthenparambil, Tanja Zamrik, Justin Ferdinandus, Evgenii Shumilov, Andrea Kerkhoff, Thomas Melchardt, Paul J. Bröckelmann, Carsten Kobe, Peter Borchmann and Bastian von Tresckow. Anti-programmed cell death protein 1-based salvage therapy for relapsed/refractory Hodgkin lymphoma: a multicenter real-world analysis. Haematologica. 2026 Feb 26. doi: 10.3324/haematol.2025.288996 [Epub ahead of print]

Anti–programmed cell death protein 1–based salvage therapy for relapsed/refractory Hodgkin lymphoma: a multicenter real-world analysis

Hishan Tharmaseelan[1], Lisa M. Sgonina[2], Ina Bühnen[1], Ulf Schnetzke[3,4], Julia Meißner[5], Markus Sellmayr[6], David Beverungen[7], Johannes C. Hellmuth[6], Joe Puthenparambil[8], Tanja Zamrik[8], Justin Ferdinandus[1], Evgenii Shumilov[2], Andrea Kerkhoff[2], Thomas Melchardt[9], Paul J. Bröckelmann[1], Carsten Kobe[10], Peter Borchmann[1], Bastian von Tresckow[1,8]*

[1] *University of Cologne, Faculty of Medicine and University Hospital of Cologne, Department I of Internal Medicine; Center for Integrated Oncology Aachen Bonn Cologne Duesseldorf (CIO ABCD); and German Hodgkin Study Group (GHSG), Cologne, Germany*

[2] *Department of Medicine A, Hematology, Oncology and Pneumology, University Hospital Muenster (UKM), Muenster, Germany*

[3] *Klinik fuer Innere Medizin II, Abteilung fuer Haematologie und Internistische Onkologie, Universitaetsklinikum Jena, Jena, Germany*

[4] *Comprehensive Cancer Center Central Germany, Campus Jena, Jena, Germany*

[5] *Department of Hematology and Oncology, University of Heidelberg, Heidelberg, Germany*

[6] *Department of Medicine III, LMU University Hospital, Ludwig-Maximilians-Universität (LMU) Munich, Munich, Germany*

[7] *Department of Hematology, Cellular Therapy and Hemostaseology, University Hospital Leipzig, Leipzig, Germany*

[8] *Department of Hematology and Stem Cell Transplantation, West German Cancer Center and German Cancer Consortium (DKTK partner site Essen), University Duisburg-Essen, University Hospital Essen, Essen, Germany*

[9] *Department of Internal Medicine III with Haematology, Medical Oncology, Haemostaseology, Infectiology and Rheumatology, Oncologic Center, Salzburg Cancer Research Institute, Center for Clinical Cancer and Immunology Trials (SCRI-CCCIT), Paracelsus Medical University Salzburg, Salzburg, Austria*

[10] *Department of Nuclear Medicine, University Hospital Cologne, Cologne, Germany*

**: Corresponding Author; Bastian von Tresckow Contact: Department of Hematology and Stem Cell Transplantation, University Hospital Essen, Hufelandstraße 55, 45147 Essen, Germany, email: Bastian.vonTresckow@uk-essen.de*

Short Title: *Real-World Analysis of PD-1-based salvage in r/r cHL*

Data availability statement: *The datasets analyzed during the current study are available from the corresponding author on reasonable request.*

Ethics approval and patient consent statement: *The requirement for written informed consent was waived due to the anonymous and retrospective fashion of analysis according to local institutional IRB-guidelines.*

Competing interests: US has received honoraria and travel support from AbbVie, AstraZeneca, Beigene, BMS, Gilead Kite, J&J, Novartis, SOBI and Takeda; **TM** has received honoraria from Takeda, BMS and MSD; **PJB** is an advisor or consultant for Hexal, Merck Sharp & Dohme (MSD), Need Inc., Stemline and Takeda; holds stock options in Need Inc., has received honoraria from AstraZeneca, BeOne, Bristol-Myers Squibb/Celgene (BMS), Eli Lilly, MSD, Need Inc., Stemline and Takeda and reports research funding from BeOne (institution), BMS (institution), MSD (institution) and Takeda (institution); **BvT** is an advisor or consultant for Allogene, Amgen, BMS/Celgene, Cerus, Gilead Kite, Incyte, IQVIA, Janssen-Cilag, Lilly, Merck Sharp & Dohme, Miltenyi, Novartis, Noscendo, Pentixapharm, Pfizer, Pierre Fabre, Qualworld, Regeneron, Roche, Sobi and Takeda; has received honoraria from AbbVie, AstraZeneca, BMS/Celgene, Gilead Kite, Incyte, Janssen-Cilag, Lilly, Merck Sharp & Dohme, Novartis, Roche, Serb and Takeda; reports research funding from Esteve (Inst), Merck Sharp & Dohme (Inst), Novartis (Inst), and Takeda (Inst); and reports travel support from AbbVie, AstraZeneca, Gilead Kite, Janssen-Cilag, Lilly, Merck Sharp & Dohme, Pierre Fabre, Roche, Takeda, and Novartis; is member of steering committees for Regeneron (Inst) and Takeda.

Author contributions: HT was responsible for conceptualization, methodology, validation, visualization, writing – review & editing, writing – original draft, funding acquisition, investigation, and software. LMS contributed to conceptualization and data curation, while IB was responsible for formal analysis and software. US, JM, MS, DB, and JP contributed to data curation. JCH was involved in data curation and writing – review & editing. TZ contributed to investigation, visualization, and data curation. JF worked on methodology, investigation, and conceptualization. ES was responsible for data curation, supervision, and methodology, while AK contributed to data curation. TM was responsible for visualization, writing – review & editing, and data curation. PJB contributed to visualization, writing – review & editing, and validation. CK supported writing – review & editing. PB was responsible for supervision, resources, and investigation. BvT contributed to supervision, resources, data curation, formal analysis, and project administration.

Abstract:

In relapsed/refractory classic Hodgkin lymphoma (r/r cHL), salvage followed by high-dose chemotherapy and autologous stem cell transplantation (HD-ASCT) yields suboptimal outcomes. PD-1 inhibitor-based salvage regimens have shown superior complete response (CR) rates of up to 95% (for P-GVD) with unprecedented PFS after HD-ASCT. However, immune checkpoint inhibitors are not EMA-approved in 2nd line, and European data remain sparse. This retrospective, multicentric analysis included r/r cHL patients who received salvage with intent to consolidation. Response before and after SCT consolidation, and PFS and OS were assessed. 47 patients were included (median prior lines: 2). Salvage regimens were PD-1 monotherapy (n=10), PD-1 + chemotherapy [P-ICE/ N-ICE /P-GVD] (n=34), or PD-1 + BV (brentuximab vedotin) (n=3). Overall response (OR)/CR were 90.9%/47.7%. OR/CR rates by salvage regimen were: PD-1 monotherapy 80/10%; PD-1 + chemotherapy, 93.6/61.3%; PD-1 + BV, 100/33.3%. With median follow-up of 16 months, 1-year PFS was 83.9% and OS was 95.6%. In the subgroup of patients with one prior line (n=22), 1-year PFS was 100%. In this real-world cohort, previously reported high CR rates were not reached. However, with the restriction of a limited follow-up, outcomes after HD-ASCT were excellent, supporting the role of PD-1 inhibitor-based salvage followed by consolidative HD-ASCT for r/r cHL.

Introduction

Despite classic Hodgkin lymphoma (cHL) showing high cure rates of up to 90% in first line[1], patients with relapsed/refractory classic Hodgkin lymphoma (r/r cHL) face an unfavorable prognosis with PFS rates below 50% with conventional chemotherapy[2].

The treatment of this subgroup of patients in the past mainly consisted of two cycles of traditional polychemotherapy-based regimens, such as ICE[3] or DHAP[4], followed by high-dose chemotherapy (HD) and autologous stem cell transplant (ASCT)[5]. In selected cases with localized disease, radiotherapy is applied. As the prognosis of patients with r/r cHL undergoing HD-ASCT is highly dependent on the response prior to HD, the most effective salvage regimens should be used in this setting.

In the last decade, immunotherapy using immune checkpoint inhibitors (ICI) has been investigated for several indications[6]. Classic Hodgkin lymphoma shows high expression of programmed cell death 1 (PD-1) receptor ligands, caused, among other factors, by EBV infection[7] and amplification of chromosome 9p24.1[8]. There have been several multicentric trials incorporating ICI, with the pivotal monotherapy trials Keynote-087 (NCT02453594)[9] and Checkmate 205 (NCT02181738)[10] showing high response rates in patients with second or higher r/r cHL [Keynote-087: ORR (overall response rate) / CR (complete response) in 69/22.4%; Checkmate 205: ORR/CR in 69/16%]. These results have established pembrolizumab and nivolumab as effective third-line treatments in otherwise dire cases, leading to FDA/EMA approval. While Keynote-204

expanded the FDA indication of pembrolizumab to first relapse, this was not adopted by the EMA[11].

ICI are powerful agents, resulting in sensitization to chemotherapy[12] and to radiotherapy[13]. Thus, combination regimens of ICI and chemotherapy have been investigated not only as part of first line therapy[14,15] but also incorporated in salvage regimens such as P-GVD (pembrolizumab, gemcitabine, vinorelbine, liposomal doxorubicin), N-ICE (nivolumab, ifosfamide, carboplatin, etoposide) or P-ICE (pembrolizumab, ifosfamide, carboplatin, etoposide) and tested in single-arm phase II trials. Moskowitz et al. included 39 patients in a phase II study with P-GVD, achieving an ORR/CR rate of 100/95% after 2-4 cycles of treatment[16]. In two other phase II salvage trials, combining ICI with chemotherapy with intent to undergo HD and ASCT achieved similarly high ORR and CR rates (P-ICE: Bryan et al., n=42 patients, ORR/CR rates of 97.3%/ 86.5%[17]; N-ICE: Mei et al., n= 34, ORR/CR rate 100%/ 89%[18]). In addition, combination trials of the antibody-drug conjugate brentuximab vedotin with nivolumab were able to show ORR/CR of 85%/67%, with a 3-year PFS of 77% in N=91 patients[19].

More recently, a US real-world cohort study performed by Desai et al. confirmed these phase II results in an analysis of 936 patients, from which 65 patients receiving ICI-based regimens prior to HD-ASCT had superior 2-year PFS [98%, compared to 68.8% with conventional chemotherapy (n=728)][20]. Moreover, Merryman et al. had evaluated the role of ICI-based salvage followed by HD in patients with multiply r/r cHL (defined as insufficient response to proceed to ASCT after two or more treatment lines) and showed

highly favorable outcomes (18-months PFS of 81% in 78 patients evaluated) in comparison to historical data with conventional salvage therapy[21]. An overview of different anti-PD-1-based salvage treatment regimens is provided in Table 1.

All of the studies mentioned were conducted in a US cohort. However, ICIs are not EMA-approved for second line treatment of cHL and published European trials in this setting are thus far lacking, leading to the absence of data in a European setting. Therefore, we aimed to investigate the efficacy of ICI-based salvage therapy in a European multicenter real-world cohort.

Methods

Data collection and inclusion criteria

This retrospective, international, multicenter cohort study included patients from eight large academic centers across Europe. All patients treated with anti-PD-1-based salvage for a r/r cHL regardless of line of therapy (incl. PD-1-monotherapy or the combination with other drugs) and intent to undergo consolidation by HD-ASCT or allogeneic stem cell transplant (SCT), were included in this analysis. This study was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective nature of the study and the use of anonymized data, the requirement for written informed consent was waived according to local IRB regulations.

Anonymized patient data, incl. baseline characteristics, e.g., age, sex, risk factors, and Ann Arbor stage, as well as information on prior treatment lines — including salvage

and high-dose regimens — any immune-related adverse events (irAEs) and the status at last follow-up were collected based on a chart review by locally treating physicians.

Statistical analysis

The statistical analysis was conducted in SAS (version 9.4). Patient characteristics were described including mean and min/max. Treatments were assigned to three groups: ICI monotherapy (I); ICI + chemotherapy, including any polychemotherapy (II); and ICI + BV (III). To evaluate the metabolic response, best overall response in 18F-FDG PET-CT was documented by the local physicians as partial response (PR), complete response (CR), stable disease (SD), and progressive disease (PD) and Deauville-Score assessment was collected[23]. PFS and OS were estimated using the Kaplan-Meier method including 95% confidence intervals (CIs). PFS was defined as the interval from date of start of the treatment to the date of clinical or radiographic disease progression or death from any cause, whichever occurred first; patients without an event were censored at the date of last follow-up. OS was defined as the interval from the start of treatment to death of any cause. To enhance the comparability to previous phase-II trials, in a subgroup-analysis patients with only one prior line of treatment were evaluated.

(Immune related) adverse events and deaths

All immune related adverse effects (irAE), incl. specific subtypes, management and engraftment syndrome as reported by the local investigators were collected. Deaths and the cause of death were retrieved from the medical reports.

Results

In total N=47 patients treated between 2017 and 2024 were included in the analysis, from which 36.2% were female, had a median age of 31 (range 19-69) at start of ICI-treatment and received a median of 2 prior lines of treatment (range 1-4). Patients received mostly eBEACOPP (+ABVD) -based first-line treatment (n=32; 68%).

Pembrolizumab/nivolumab was applied as a monotherapy in 21.3% of cases (n=10) (I). From the group receiving salvage with ICI + chemotherapy (II) (75.6%, n=34), the majority n=30 (88.2%) received P-GVD. ICI in combination with BV (III) was administered to 3 patients (6.4%) (Table 2). 91.5% (n=43) of patients underwent consolidation, from which the majority received HD-ASCT (78.7%, n=37) and a smaller fraction allo-SCT (12.8%, n=6). Four patients (8.5%) did not undergo consolidation with HD-ASCT/allo-SCT due to patients' decision (4.2%, n=2), toxicity (2.1%, n=1) or death prior to consolidation (2.1%, n=1). Of the six patients undergoing allo-SCT (12.8%), one (16.7%) had no prior HD-ASCT. All 25 patients with more than one prior treatment line received chemotherapy-based salvage prior to reaching the anti-PD-1-based salvage line. From all patients included, no one received a second HD-ASCT during the available follow-up. A cumulative listing of the number of cycles and regimens is provided in supplemental table 1.

Treatment response pre- /post HD-ASCT

Overall response to anti-PD-1-based salvage was observed in 90.9% of patients (from n=44 of 47 evaluable patients). 47.7% of patients had a complete response to salvage. In the ICI + chemotherapy group (II), CR rate after salvage was the highest, with 61.3% achieving a complete remission. In contrast, the ICI monotherapy group (Group I) showed a notably lower CR rate, with only 1 of 10 patients achieving a CR. All patients post consolidation therapy (n=33 evaluable) showed a response to treatment, of which 97% (n=32) had a complete response (Figure 1).

Progression-free and overall survival

With a median follow-up time of 16 months, the 1-year (1y) PFS was 83.9% in the whole cohort (95% CI 71.9 - 95.9%) and 1-year OS was 95.6% (95% CI 89.6 - 100.0%). ICI + BV-based salvage (III) achieved the highest 1y- PFS (100% [95% CI 100.0 - 100.0%]), ICI + chemotherapy (II) reached a 1y- PFS of 90.2% (n=34, [95% CI 79.5 - 100.0%]. Patients receiving consolidation SCT had a higher 1y- PFS (87.7 [95% CI 76.3 - 99.1%] vs. 37.5% [95% CI 0.0 – 93.6%] without SCT) (Figure 2).

Subgroup-analysis of the second line patients

In the cohort of patients receiving ICI in second line (n=22), there was a higher share of male patients (59.1%) and eBEACOPP (+ ABVD) based regimens in the first line (72.8%). 95% of the subjects in this cohort received P-GVD (Table 3).

The response to treatment in this subgroup was comparable to the overall cohort, with an ORR prior to HD-ASCT of 95.5%, and a CR-rate of 59.1% (a detailed description of response rates for all patients and subgroup analysis can be found in supplemental table 2) (Figure 3). The 1y-PFS in this subgroup was excellent [100% (95% CI 100.0 - 100.0%)] (Figure 4).

Immune related adverse events and deaths

In our cohort irAE were documented in 10.6% of cases (n=5), 2 patients had an ICI-associated pneumonitis (4.3%). In one patient each a pancolitis (2.1%), thyroiditis (2.1%), and hepatitis (2.1%) were observed. All irAE except 1/5 cases (thyroiditis) resolved. The patient with a pancolitis was re-exposed to ICI. Two cases of engraftment syndrome (5.4%) were described in the cohort. Four deaths (8.5%) were observed (two were related to cHL, one secondary AML, one with unknown cause; supplemental table 3).

Discussion

Herein, we report favorable outcomes in patients treated with ICI-based salvage for relapsed/refractory cHL with intent to undergo HD-ASCT/allo-SCT in a real-world European setting. In a cohort of N=47 patients the 1-year PFS was 83.9%, with patients receiving anti-PD-1-based salvage in second line achieving a 1-year PFS of 100%.

Previously reported high and unprecedented ORR/CR rates with anti-PD-1-chemotherapy combinations, e.g., 100/95% with 2-4 cycles P-GVD by Moskowitz et al., could not be confirmed in the European cohort. This observation holds true in both intensively pre-treated patients and in those with only one prior line of treatment (2nd line treated patients in our analysis had ORR/CR rate of 95.5/59.1% with anti-PD-1 + chemotherapy). However, in the latter group an excellent PFS of 100% was reached, comparing favorably to what is known from historical data of r/r cHL patients treated with conventional chemotherapy salvage prior SCT in the second line (1-year PFS of 82.3% [95% CI 77.5–86.2%])[2].

Anti-PD-1 treatment altogether has a favorable toxicity profile. However, immune-related adverse events during treatment and associated effects such as increased risk of GvHD in patients subsequently undergoing allo-SCT are of concern[24,25]. Though limited by the retrospective nature of this study, irAE toxicities were reported in 10.6% of patients, a comparable rate to other publications[26]. In addition, a potentially elevated risk of engraftment syndrome was previously reported in patients receiving anti-PD-1-based salvage before ASCT[25]. In our study, engraftment syndrome was only observed in two cases of 43 patients undergoing SCT (4.6%). This was different from the trial by Moskowitz et al. investigating P-GVD, where engraftment syndrome was reported in 68% of patients[16]. This was with a relatively broad definition of any of the following symptoms occurring days 8–11 post-ASCT: high-grade fever (>38.5□°C), skin rash covering >25% of body surface area, or diarrhea (>2 watery bowel movements in 24 hours); or hepatitis, pulmonary infiltrates, acute kidney injury, or neurologic

dysfunction[16]. In the study by Mei et al, investigating N-ICE, 4/33 (12.1%) patients experienced engraftment syndrome (in median 8 days after transplant). In the Phase II trial testing P-ICE by Bryan et al., one patient died from respiratory failure, likely due to engraftment syndrome[17]. The different frequencies of engraftment syndrome may be explained by a lack of a widely recognized definition and reporting bias, clearly showing a need for standardized criteria for engraftment syndrome and the collection of comprehensive data in the context of ASCT after anti-PD-1-based salvage[25]. In summary of our and previously published data, the addition of anti-PD-1 therapy to salvage therapy followed by consolidation seems to only add limited toxicity to the treatment strategy further supporting its use.

The findings of this study must be interpreted within the context of its limitations. Chief among these is the retrospective study design, leading to a lower availability of structured data including grading of adverse events and a potential selection bias of patients. In addition, with a median follow-up of 16 months, the durability of the responses of anti-PD-1-based salvage followed by consolidation cannot be finally judged from our dataset. Another limitation is the small sample size, probably caused by the use of more intensive eBEACOPP based regimens in first line and thus less frequent relapses and by missing approval of PD-1-inhibitors for second line treatment in Europe. Thus, a smaller share of relapses and less frequent use of ICI as part of salvage is seen in the European treatment context and intra-cohort comparisons must be interpreted with caution due to selection bias. This may result in the application of PD-1-based salvage therapy in patients with higher-risk disease (such as those

experiencing stage IV relapse or failure following prior polychemotherapy-only salvage), thereby contributing to the inclusion of a poorer-prognosis cohort and thus inferior response rates. However, in addition to the actual treatment efficacy of a regimen in a specific patient group, differences in response rates may stem from various factors, such as scanner manufacturer, scanner quality improvement over time and response assessment methodology. While the original Lugano classification relies primarily on visual assessment via Deauville scores (1–5) to determine treatment response in FDG-PET[27], this introduces inter-observer variability, especially in borderline cases. Thus, to allow for more objective and comparable PET results, some trials use modified Lugano criteria by integrating semi-quantitative measures — specifically standardized uptake value (SUV) thresholds — into the Deauville scoring system creating a heterogeneity in reported response data[28].

Moreover, the predictive role of PET in relapsed cHL in patients treated with anti-PD-1 antibodies has been questioned[21]. The above mentioned phase II studies combining checkpoint-inhibition and chemotherapy in relapsed cHL have reported comparably high CR rates, with assessments generally based on the Cheson 2014 criteria[27]. Different data collection time periods, heterogeneity in PET assessments and technical equipment might have contributed to differences between the CR rates in the above-mentioned phase II trials and our analysis. The comparably high ORR-rates but lower CR rates in our analysis as well as the similar PFS results reported in phase II trials and our cohort point in this direction. Certainly, the reporting of quantitative biomarkers such as metabolic tumor volume (MTV) and minimal residual disease by assessment of

circulating tumor DNA (ctDNA) is warranted in future analyses concerning anti-PD-1-therapy in r/r cHL.

Nevertheless, the 1-year PFS of 83.9% is remarkable given the heterogeneity of this cohort with six patients undergoing allo-SCT, others receiving ICI monotherapy or combination with polychemotherapy/BV as salvage or being in third or fourth line of disease. ICI monotherapy in particular was predominantly used in patients who had undergone more intensive prior treatment; the inferior PFS is therefore likely to reflect primarily patient selection and prior treatment intensity rather than necessarily inferior biological efficacy (only 1/10 patients treated with ICI monotherapy in second line). In addition, due to this selection the high CR rates after ICI-monotherapy after consolidative HD-ASCT have to be interpreted with caution.

Our findings show that even after more intense prior therapy and lower CR rates to anti-PD-1-based salvage as compared to US data, PFS outcome after consolidation in our dataset is still excellent (87.7% after one year) and in the range of published US registry data[20]. However, longer follow-up is needed to confirm the durability of the responses.

Immune checkpoint inhibitors introduced a fundamental shift in oncology, with particularly high impact in cHL, where they achieved durable tumor control in a subset of patients who previously lacked effective treatment options. In retrospective evaluations since the era of the incorporation of novel agents in the salvage prior to ASCT, improvements in outcomes were observed compared to historic data, especially

when PD-1-inhibitors were used as part of salvage[29]. Currently there are still trials evaluating regimens in this setting, e.g. the Canadian HD11 trial is recruiting in a phase II trial (NCT05180097), where patients are randomized to GDP (Gemcitabine, Dexamethasone, and Cisplatin) or Pembrolizumab + BV prior to ASCT.

Subject to longer follow-up of phase II data, real-world data and the results reported here confirming the durability of the high early PFS rates, anti-PD-1-based therapy seems to be the preferred salvage option before HD-ASCT. Registrational clinical trials leading to approval of anti-PD-1-based treatment in the first salvage setting in r/r cHL in Europe are highly warranted to increase patient access of the probably most effective therapy in this difficult situation. However, if the high PFS rates with anti-PD-1-based therapy in the consolidation setting in r/r cHL hold true after longer follow-up, one might question if a standard treatment group consisting of conventional chemotherapy was still ethically justified.

In summary, our results support the use of ICI and polychemotherapy-based salvage combinations in r/r cHL and supports the role of HD and ASCT in the treatment of relapsed or refractory classic Hodgkin lymphoma.

References

1. Ansell SM. Hodgkin lymphoma: 2025 update on diagnosis, risk-stratification, and management. Am J Hematol. 2024;99(12):2367-2378.

2. Bröckelmann PJ, Müller H, Gillessen S, et al. Clinical outcomes of relapsed and refractory Hodgkin lymphoma patients after contemporary first-line treatment: a German Hodgkin Study Group analysis. Leukemia. 2022;36(3):772-780.

3. Hertzberg MS, Crombie C, Benson W, Taper J, Gottlieb D, Bradstock KF. Outpatient-based ifosfamide, carboplatin and etoposide (ICE) chemotherapy in transplant-eligible patients with non-Hodgkin's lymphoma and Hodgkin's disease. Ann Oncol. 2003;14 Suppl 1:i11-i16.

4. Velasquez WS, Cabanillas F, Salvador P, et al. Effective Salvage Therapy for Lymphoma With Cisplatin in Combination With High-Dose Ara-C and Dexamethasone (DHAP). Blood. 1988;71(1):117-122.

5. Josting A, Müller H, Borchmann P, et al. Dose Intensity of Chemotherapy in Patients With Relapsed Hodgkin's Lymphoma. J Clin Oncol. 2010;28(34):5074-5080.

6. Robert C, Schachter J, Long GV, et al. Pembrolizumab versus Ipilimumab in Advanced Melanoma. N Engl J Med. 2015;372(26):2521-2532.

7. Green MR, Rodig S, Juszczynski P, et al. Constitutive AP-1 Activity and EBV Infection Induce PD-L1 in Hodgkin Lymphomas and Posttransplant Lymphoproliferative Disorders: Implications for Targeted Therapy. Clin Cancer Res. 2012;18(6):1611-1618.

8. Green MR, Monti S, Rodig SJ, et al. Integrative analysis reveals selective 9p24.1 amplification, increased PD-1 ligand expression, and further induction via JAK2 in nodular sclerosing Hodgkin lymphoma and primary mediastinal large B-cell lymphoma. Blood. 2010;116(17):3268-3277.

9. Armand P, Zinzani PL, Lee HJ, et al. Five-year follow-up of KEYNOTE-087: pembrolizumab monotherapy for relapsed/refractory classical Hodgkin lymphoma. Blood. 2023;142(10):878-886.

10. Armand P, Engert A, Younes A, et al. Nivolumab for Relapsed/Refractory Classic Hodgkin Lymphoma After Failure of Autologous Hematopoietic Cell Transplantation: Extended Follow-Up of the Multicohort Single-Arm Phase II CheckMate 205 Trial. J Clin Oncol. 2018;36(14):1428-1439.

11. FDA. FDA extends approval of pembrolizumab for classical Hodgkin lymphoma. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-extends-approval-pembrolizumab-classical-hodgkin-lymphoma (2020, accessed June 15, 2025).

12. Galluzzi L, Humeau J, Buqué A, Zitvogel L, Kroemer G. Immunostimulation with chemotherapy in the era of immune checkpoint inhibitors. Nat Rev Clin Oncol. 2020;17(12):725-741.

13. Chen J, Levy A, Tian A-L, et al. Low-dose irradiation of the gut improves the efficacy of PD-L1 blockade in metastatic cancer patients. Cancer Cell. 2025;43(3):361-379.e10.

14. Bröckelmann PJ, Goergen H, Keller U, et al. Efficacy of Nivolumab and AVD in Early-Stage Unfavorable Classic Hodgkin Lymphoma: The Randomized Phase 2 German Hodgkin Study Group NIVAHL Trial. JAMA Oncol. 2020;6(6):872-880.

15. Herrera AF, LeBlanc M, Castellino SM, et al. Nivolumab+AVD in Advanced-Stage Classic Hodgkin's Lymphoma. N Engl J Med. 2024;391(15):1379-1389.

16. Moskowitz AJ, Shah G, Schöder H, et al. Phase II Trial of Pembrolizumab Plus Gemcitabine, Vinorelbine, and Liposomal Doxorubicin as Second-Line Therapy for Relapsed or Refractory Classical Hodgkin Lymphoma. J Clin Oncol. 2021;39(28):3109-3117.

17. Bryan LJ, Casulo C, Allen PB, et al. Pembrolizumab Added to Ifosfamide, Carboplatin, and Etoposide Chemotherapy for Relapsed or Refractory Classic Hodgkin Lymphoma: A Multi-institutional Phase 2 Investigator-Initiated Nonrandomized Clinical Trial. JAMA Oncol. 2023;9(5):683-691.

18. Mei MG, Lee HJ, Palmer JM, et al. Response-adapted anti-PD-1–based salvage therapy for Hodgkin lymphoma with nivolumab alone or in combination with ICE. Blood. 2022;139(25):3605-3616.

19. Advani RH, Moskowitz AJ, Bartlett NL, et al. Brentuximab vedotin in combination with nivolumab in relapsed or refractory Hodgkin lymphoma: 3-year study results. Blood. 2021;138(6):427-438.

20. Desai SH, Spinner MA, David K, et al. Checkpoint inhibitor-based salvage regimens prior to autologous stem cell transplant improve event-free survival in relapsed/refractory classic Hodgkin lymphoma. Am J Hematol. 2023;98(3):464-471.

21. Merryman RW, Redd RA, Nishihori T, et al. Autologous stem cell transplantation after anti-PD-1 therapy for multiply relapsed or refractory Hodgkin lymphoma. Blood Adv. 2021;5(6):1648-1659.

22. Bröckelmann PJ, Müller H, Kücüksarioglan E, et al. Outcomes of patients with the third or higher relapsed classical Hodgkin lymphoma: results from the German Hodgkin Study Group. Ann Oncol. 2019;30(3):490-491.

23. Barrington SF, Kluge R. FDG PET for therapy monitoring in Hodgkin and non-Hodgkin lymphomas. Eur J Nucl Med Mol Imaging. 2017;44(Suppl 1):97-110.

24. Tabbara N, Zahurak M, Sterling CH, et al. Improved overall survival with checkpoint inhibition and allogeneic transplantation in relapsed Hodgkin lymphoma. Blood Adv. 2025;9(5):1202-1209.

25. Bai B, Wang X-X, Gao Y, et al. Prior anti-PD-1 therapy as a risk factor for life-threatening peri-engraftment respiratory distress syndrome in patients undergoing autologous stem cell transplantation. Bone Marrow Transplant. 2021;56(5):1151-1158.

26. Eun Y, Kim IY, Sun J-M, et al. Risk factors for immune-related adverse events associated with anti-PD-1 pembrolizumab. Sci Rep. 2019;9(1):14039.

27. Cheson BD, Fisher RI, Barrington SF, et al. Recommendations for Initial Evaluation, Staging, and Response Assessment of Hodgkin and Non-Hodgkin Lymphoma: The Lugano Classification. J Clin Oncol. 2014;32(27):3059-3067.

28. Casasnovas R-O, Bouabdallah R, Brice P, et al. PET-adapted treatment for newly diagnosed advanced Hodgkin lymphoma (AHL2011): a randomised, multicentre, non-inferiority, phase 3 study. Lancet Oncol. 2019;20(2):202-215.

29. Spinner MA, Sica RA, Tamaresis JS, et al. Improved outcomes for relapsed/refractory Hodgkin lymphoma after autologous transplantation in the era of novel agents. Blood. 2023;141(22):2727-2737.

30. Moskowitz A, Shah G, Ganesan N, et al. Pembrolizumab maintenance instead of autologous hematopoietic cell transplantation for patients with relapsed or refractory Hodgkin lymphoma in complete response after pembrolizumab. Hemasphere. 2024;8(S2):e70012.

31. Ding K, Liu H, Ma J, et al. Tislelizumab with gemcitabine and oxaliplatin in patients with relapsed or refractory classic Hodgkin lymphoma: a multicenter phase II trial. Haematologica. 2023;108(8):2146-2154.

32. Liu P, Li Z, Cen H, et al. Sintilimab (anti-PD-1) plus ifosfamide, carboplatin, and etoposide (ICE) in second-line classical Hodgkin lymphoma (cHL): Results of a multicenter, randomized, controlled, double-blind phase 3 study (ORIENT-21). J Clin Oncol. 2025;43(16_suppl):7007.

Trial	Regimen	n	ORR (%)	CRR (%)	PFS (%)
Mei et al.[18]	N-ICE	34	100	89	2-year PFS 72% / 94% after aSCT
Bryan et al.[17]	P-ICE	42	97.3	86.5	2-year PFS: 87.2%
Moskowitz et al.[16,30]	P-GVD	39	100	95	5-year PFS: 91% (95% CI: 82-100)
Ding et al.[31,*]	Tislelizumab + GemOx	30	100	96.7	1-year PFS rate 96% (95% CI: 74.8-99.4%)
Liu et al.[32]	Sintilimab + ICE	44		61.4	median PFS not reached
Advani et al.[19]	Bv-Nivo	91	85	67	3-year PFS 77%

Table 1: Overview of the most important PD-1-based salvage incorporating trials. ORR: Overall response rate; CRR: Complete remission rate; PFS: Progression-free survival; N-ICE: Nivolumab + Ifosfamide, Carboplatin, Etoposide; P-ICE: Pembrolizumab + Ifosfamide, Carboplatin, Etoposide; P-GVD: Pembrolizumab + Gemcitabine, Vinorelbine, Doxil; Tislelizumab + GemOx: Tislelizumab + Gemcitabine, Oxaliplatin; Sintilimab + ICE: Sintilimab + Ifosfamide, Carboplatin, Etoposide; BV-Nivo: Brentuximab Vedotin + Nivolumab; *Ding et al. did not apply high-dose chemotherapy consolidation.

			N	%
Age at initial diagnosis	years, median (range)		28 (17-68)	
Age at start of PD-1 salvage therapy	years, median (range)		31 (19-69)	
Sex	male		30	63.8
	female		17	36.2
First line treatment	eBEACOPP - based		19	40.4
	eBEACOPP + ABVD - based		13	27.7
	ABVD - based		9	19.1
	BrECADD - based		2	4.3
	COPP/COPDAC		2	4.3
	BV-AVD		1	2.1
	N-AVD		1	2.1
Number of prior therapy lines	median (range)	2 (1-4)		
		1	22	46.8
		2	20	42.6
		3	3	6.4
		4	2	4.3
Salvage regimen	**(I)** ICI mono		**10**	21.3
	Nivolumab		4/10	40
	Pembrolizumab		6/10	60
	(II) ICI + chemotherapy		**34**	75.6
	P-GVD		30/34	88.2
	P-ICE		3/34	8.8
	N-ICE		1/34	2.9
	(III) ICI + BV		**3**	6.4
	Pembro + BV		1/3	33.3
	Nivo + BV		2/3	66.7
Consolidation	allo-SCT		6	12.8
	prior auto-SCT		5/6	83.3
	auto-SCT		37	78.7
	no SCT		4	8.5
Consolidating regimen	BEAM		36	83.7
	TEAM		1	2.3
(of auto/allo pts.)	TBI (12 Gy) + Cyclo ATG		2	4.7
	Flu/Treo(/ATG)		4	9.3

Ann Arbor stage at relapse	II	12/34	35.3
	III	8/34	23.5
	IV	14/34	41.2
B-Symptoms at relapse	yes	13/32	40.6
	no	19/32	59.4
ECOG at relapse	0	21/33	63.6
	1	12/33	36.4
Bulky disease at relapse	yes	11/34	32.4
	no	23/34	67.6

Table 2: Patient characteristics of the whole cohort. eBEACOPP: escalated BEACOPP (Bleomycin, Etoposide, Adriamycin, Cyclophosphamide, Vincristine, Procarbazine, Prednisone); ABVD: Adriamycin, Bleomycin, Vinblastine, Dacarbazine; BrECADD: Brentuximab Vedotin, Etoposide, Cyclophosphamide, Doxorubicin, Dacarbazine, Dexamethasone; COPP/COPDAC: Cyclophosphamide, Vincristine, Procarbazine, Prednisone/Cyclophosphamide, Vincristine, Prednisone, Dacarbazine; BV-AVD: Brentuximab Vedotin + Adriamycin, Vinblastine, Dacarbazine; N-AVD: Nivolumab + Adriamycin, Vinblastine, Dacarbazine; ICI: Immune checkpoint inhibitor; BV: Brentuximab Vedotin; P-GVD: Pembrolizumab + Gemcitabine, Vinorelbine, liposomal Doxorubicin; P-ICE: Pembrolizumab + Ifosfamide, Carboplatin, Etoposide; N-ICE: Nivolumab + Ifosfamide, Carboplatin, Etoposide; auto-SCT: Autologous stem cell transplantation; allo-SCT: Allogeneic stem cell transplantation; BEAM: BCNU (Carmustine), Etoposide, Cytarabine, Melphalan; TEAM: Thiotepa, Etoposide, Cytarabine, Melphalan; TBI: Total body irradiation; Cyclo: Cyclophosphamide; ATG: Anti-thymocyte globulin; Flu/Treo(/ATG): Fludarabine, Treosulfan (± ATG); ECOG: Eastern Cooperative Oncology Group performance status.

			N	%
Age at initial diagnosis	years, median (range)		29 (21-67)	
Age at start of PD-1 salvage therapy	years, median (range)		31 (21-68)	
Sex	male		13	59.1
	female		9	40.9
First line treatment	eBEACOPP - based	10/22		45.5
	eBEACOPP + ABVD - based	6/22		27.3
	BrECADD	2/22		9.1
	ABVD - based	2/22		9.1
	BV-AVD	1/22		4.5
	N-AVD	1/22		4.5
Salvage regimen	**(I)** ICI mono		**1**	4.5
	Pembrolizumab		1/1	100
	(II) ICI + chemotherapy		**20**	90.9
	P-ICE		1/20	5
	P-GVD		19/20	95
	(III) ICI + BV		**1**	4.5
	Nivo + BV		1/1	100
Consolidation	allogeneic SCT		0	0
	autologous SCT		21	95.5
	no SCT		1	4.5
Consolidating regimen *(of auto/allo pts.)*	BEAM		21	100
Ann Arbor stage at relapse	II		7/19	36.8
	III		5/19	26.3
	IV		7/19	36.8
B-Symptoms at relapse	yes		10/19	52.6
	no		9/19	47.4
ECOG at relapse	0		12/17	70.6

	1	5/17	29.4
Bulky disease at relapse	yes	3/18	16.7
	no	15/18	83.3

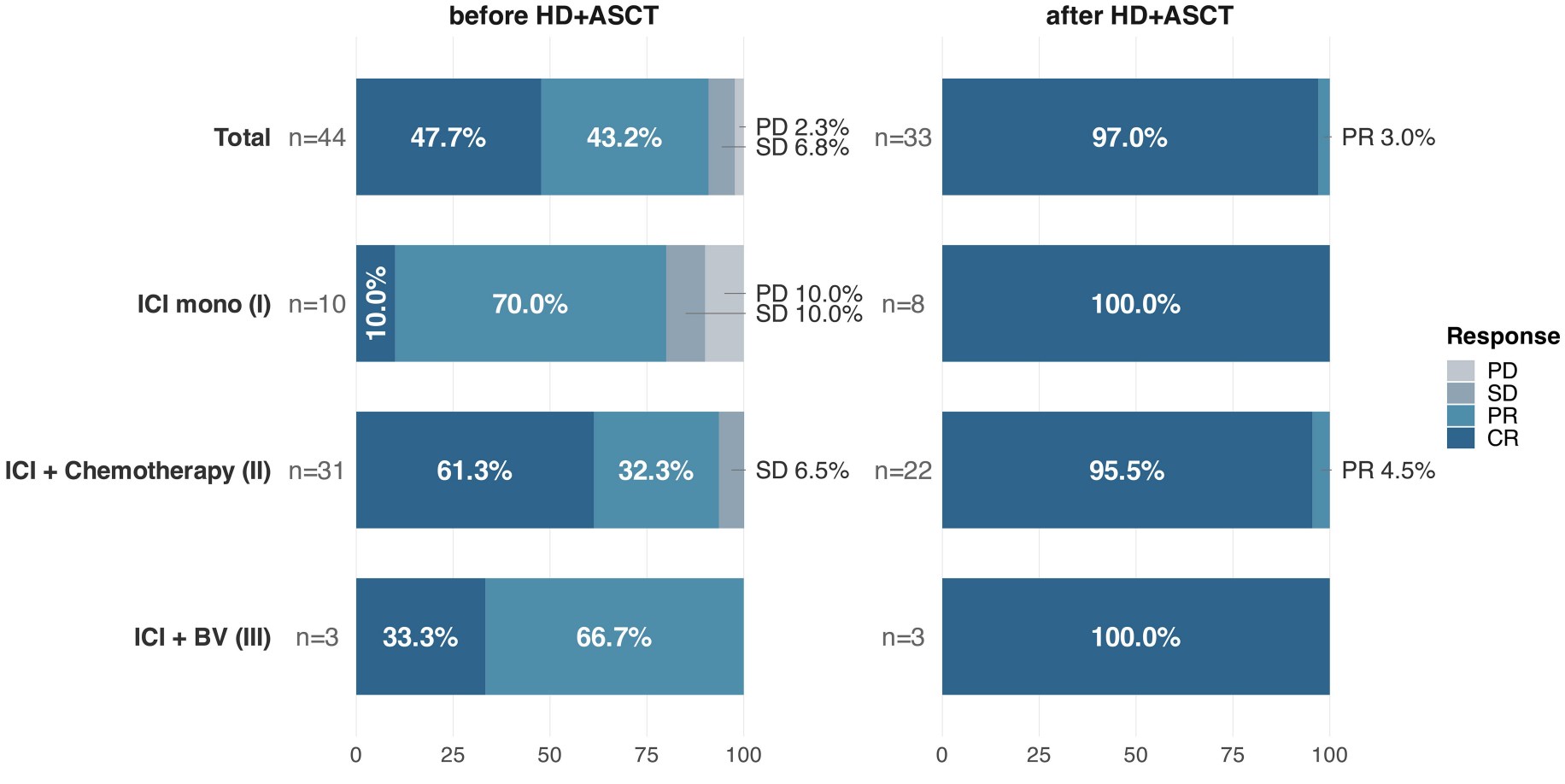
Table 3: Patient characteristics of the patients receiving one prior line of treatment. BEACOPP: escalated BEACOPP (Bleomycin, Etoposide, Adriamycin, Cyclophosphamide, Vincristine, Procarbazine, Prednisone); ABVD: Adriamycin, Bleomycin, Vinblastine, Dacarbazine; BrECADD: Brentuximab Vedotin, Etoposide, Cyclophosphamide, Doxorubicin, Dacarbazine, Dexamethasone; BV-AVD: Brentuximab Vedotin + Adriamycin, Vinblastine, Dacarbazine; N-AVD: Nivolumab + Adriamycin, Vinblastine, Dacarbazine; ICI: Immune checkpoint inhibitor; BV: Brentuximab Vedotin; P-ICE: Pembrolizumab + Ifosfamide, Carboplatin, Etoposide; P-GVD: Pembrolizumab + Gemcitabine, Vinorelbine, Doxil; Nivo + BV: Nivolumab + Brentuximab Vedotin; SCT: Stem cell transplantation; BEAM: BCNU (Carmustine), Etoposide, Cytarabine, Melphalan; ECOG: Eastern Cooperative Oncology Group performance status.

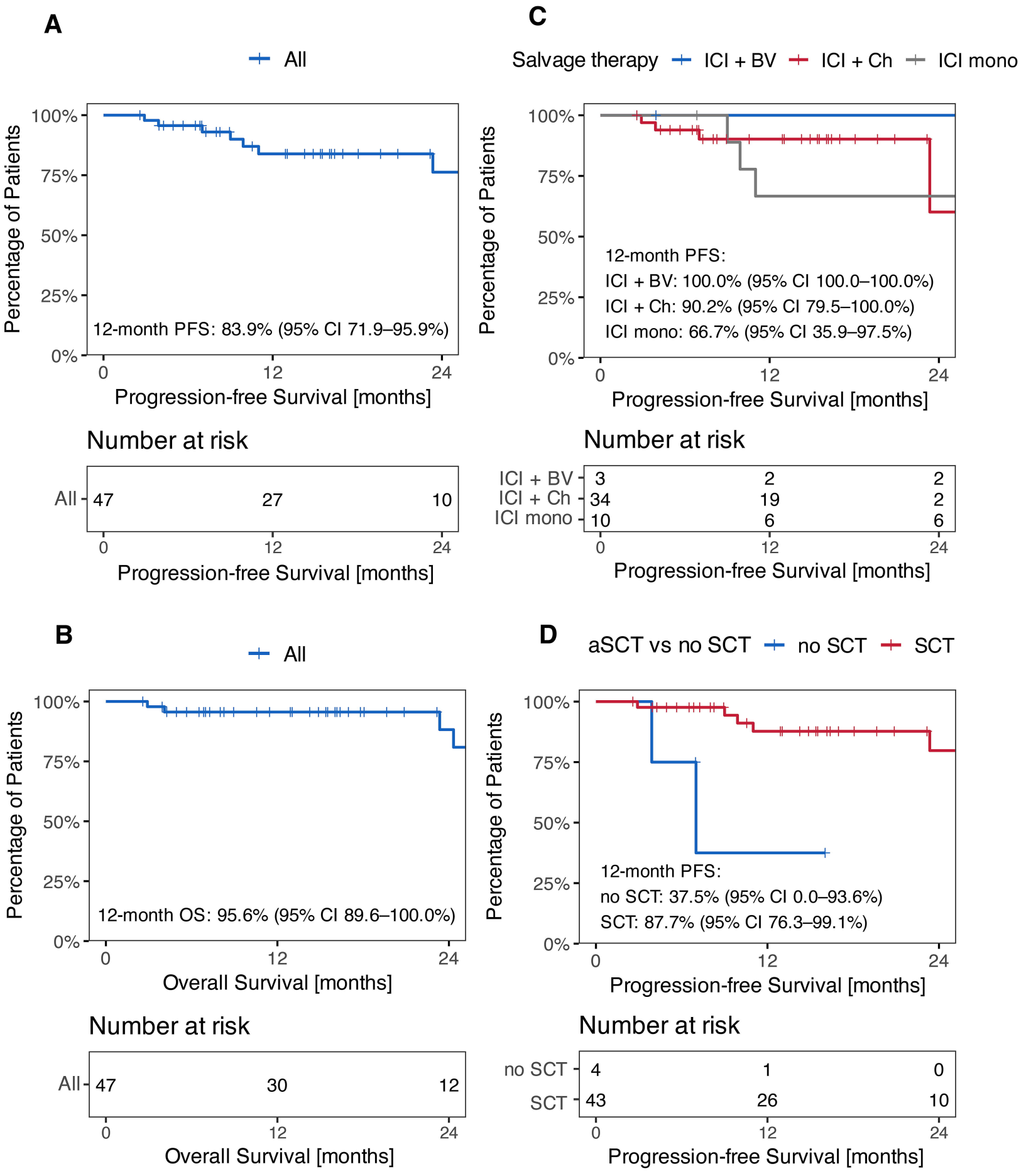
Figure 1: Best overall response to salvage therapy and subsequent consolidation, stratified by treatment subgroup. Left: response after salvage prior to high-dose chemotherapy with autologous stem cell transplantation (HD-ASCT) or allogeneic stem cell transplantation (allo). Right: response after consolidation in patients who underwent HD-ASCT/allo. Treatment subgroups: immune checkpoint inhibitor (ICI) monotherapy, ICI plus chemotherapy, and ICI plus brentuximab vedotin (BV).

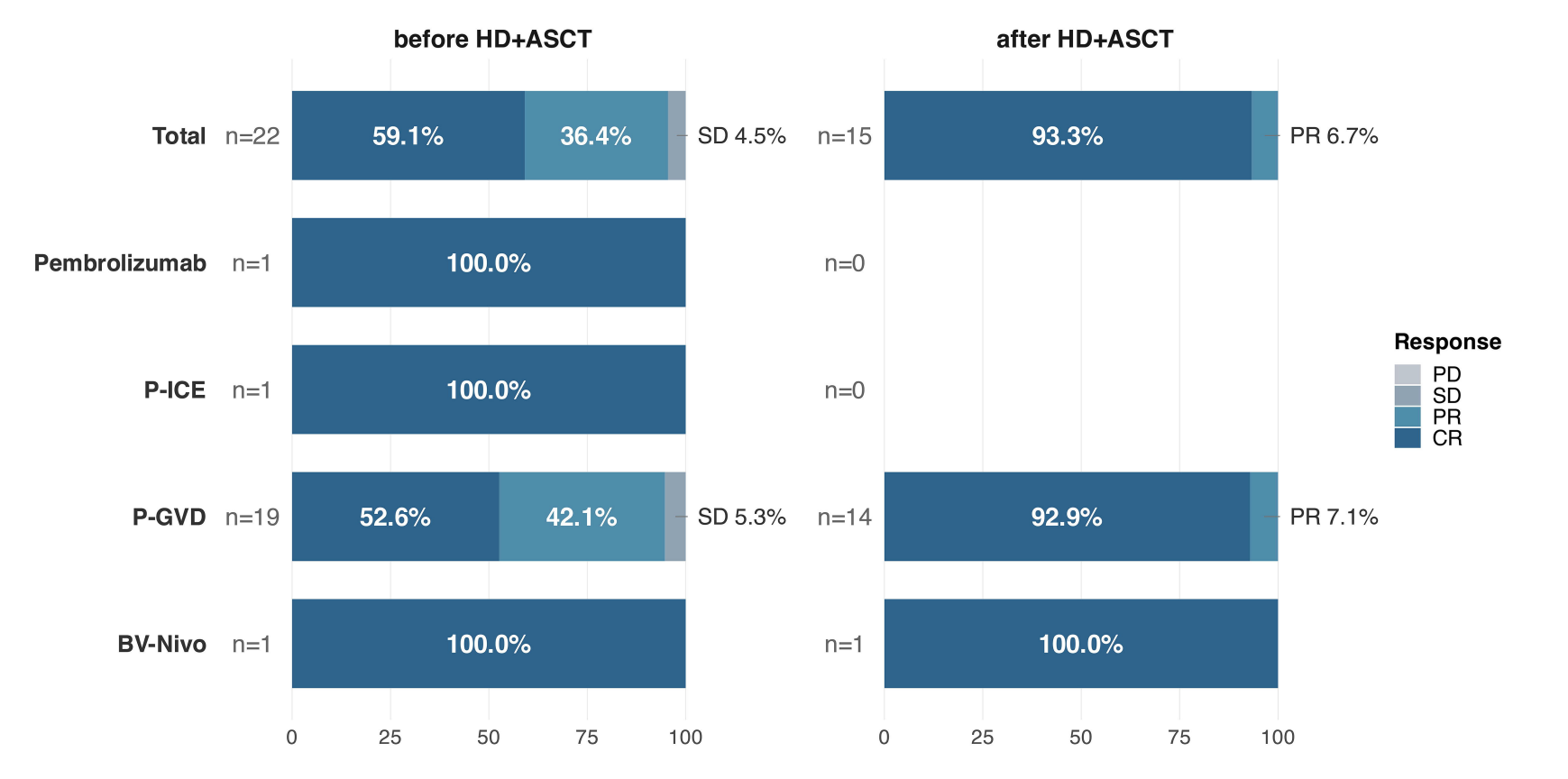
Figure 2: Kaplan–Meier plots of the entire cohort and stratified by treatment subgroups. (A) progression-free (PFS) and (B) overall survival for the entire cohort; (C) PFS stratified by salvage regimen subtype [immune checkpoint inhibitor (ICI) monotherapy, ICI plus chemotherapy (ICI+Ch), and ICI plus brentuximab vedotin (ICI+BV)]; (D) PFS stratified by consolidation with autologous/ allogeneic stem cell transplant (SCT vs. no SCT).

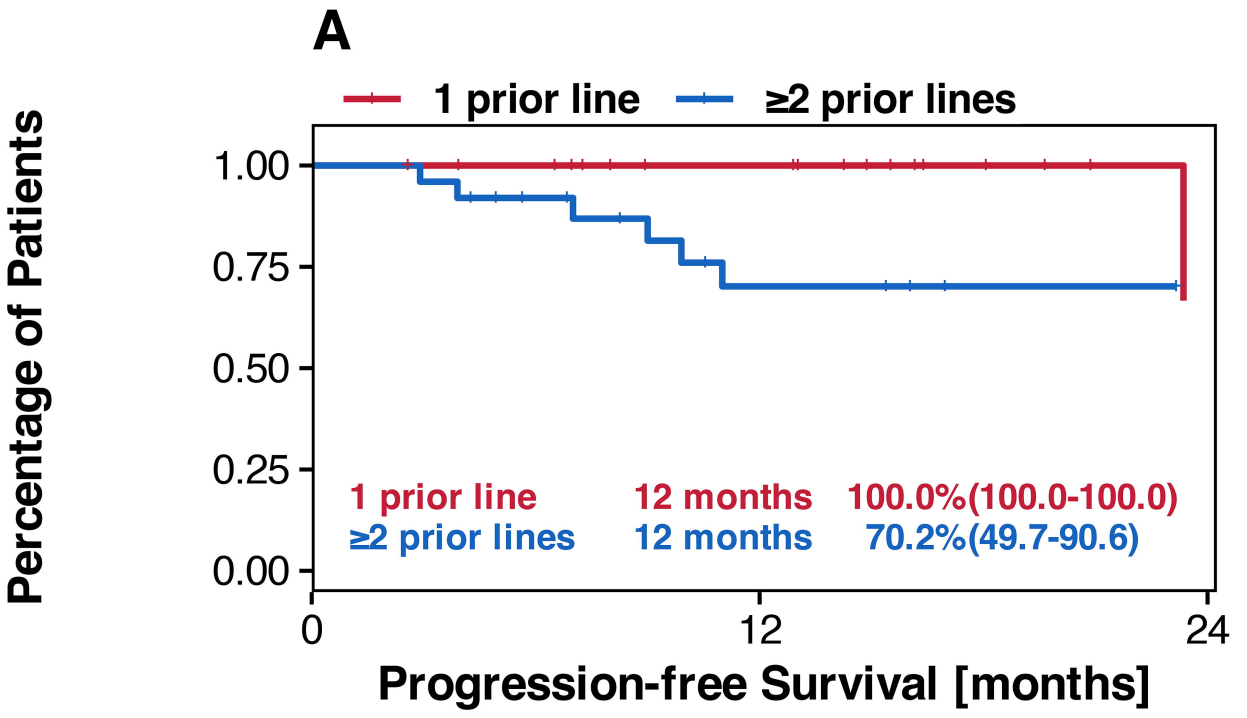
Figure 3: Best overall response to salvage therapy by exact regimen in patients who received one prior line of therapy. [P-GVD: pembrolizumab, gemcitabine, vinorelbine, liposomal doxorubicin; P-ICE: pembrolizumab, ifosfamide, carboplatin, etoposide and BV-Nivo = Brentuximab vedotin + Nivolumab]

Figure 4: Kaplan–Meier survival by number of prior treatment lines. Kaplan–Meier plots of (A) progression-free survival and (B) overall survival stratified by number of prior treatment lines (1 vs. >1)

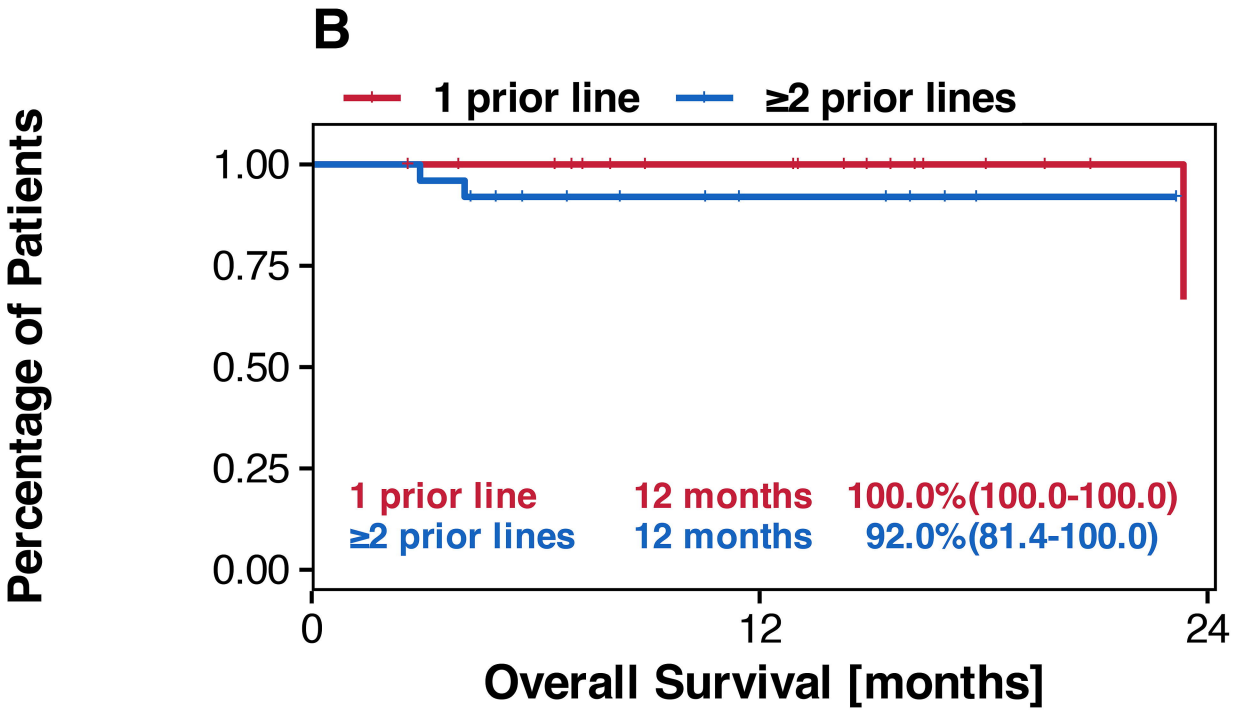

before HD+ASCT
after HD+ASCT
Total
n=44
47.7%
43.2%
PD 2.3%
SD 6.8%
n=33
97.0%
PR 3.0%
ICI mono (I)
n=10
10.0%
70.0%
PD 10.0%
SD 10.0%
n=8
100.0%
ICI + Chemotherapy (II)
n=31
61.3%
32.3%
SD 6.5%
n=22
95.5%
PR 4.5%
ICI + BV (III)
n=3
33.3%
66.7%
n=3
100.0%
0
25
50
75
100
Response
PD
SD
PR
CR


A
All
100%
75%
50%
25%
0%
Percentage of Patients
12-month PFS: 83.9% (95% CI 71.9–95.9%)
0
12
24
Progression-free Survival [months]
Number at risk
All
47
27
10
0
12
24
Progression-free Survival [months]
C
Salvage therapy
ICI + BV
ICI + Ch
ICI mono
100%
75%
50%
25%
0%
Percentage of Patients
12-month PFS:
ICI + BV: 100.0% (95% CI 100.0–100.0%)
ICI + Ch: 90.2% (95% CI 79.5–100.0%)
ICI mono: 66.7% (95% CI 35.9–97.5%)
0
12
24
Progression-free Survival [months]
Number at risk
ICI + BV
ICI + Ch
ICI mono
3
2
2
34
19
2
10
6
6
0
12
24
Progression-free Survival [months]
B
All
100%
75%
50%
25%
0%
Percentage of Patients
12-month OS: 95.6% (95% CI 89.6–100.0%)
0
12
24
Overall Survival [months]
Number at risk
All
47
30
12
0
12
24
Overall Survival [months]
D
aSCT vs no SCT
no SCT
SCT
100%
75%
50%
25%
0%
Percentage of Patients
12-month PFS:
no SCT: 37.5% (95% CI 0.0–93.6%)
SCT: 87.7% (95% CI 76.3–99.1%)
0
12
24
Progression-free Survival [months]
Number at risk
no SCT
SCT
4
1
0
43
26
10
0
12
24
Progression-free Survival [months]


before HD+ASCT
after HD+ASCT
Total
n=22
59.1%
36.4%
SD 4.5%
n=15
93.3%
PR 6.7%
Pembrolizumab
n=1
100.0%
n=0
P-ICE
n=1
100.0%
n=0
P-GVD
n=19
52.6%
42.1%
SD 5.3%
n=14
92.9%
PR 7.1%
BV-Nivo
n=1
100.0%
n=1
100.0%
0
25
50
75
100
Response
PD
SD
PR
CR



1 prior line	22	15	2
≥2 prior lines	25	12	8



1 prior line	22	15	2
≥2 prior lines	25	15	10

Supplemental Material

Supplemental table 1: Detailed description of the number of cycles and salvage regimen

No of cycles x regimen	n
4x P-GVD	5
2x P-GVD	15
4x BV-Nivo	1
16x BV-Nivo	1
4x Pembro	2
8x Nivolumab	1
7x BV-Pembro	1
3x P-GVD	10
2x Pembrolizumab	1
2x P-ICE	3
3x N-ICE	1
3x Nivolumab	1
5x Pembrolizumab	1
6x Pembrolizumab	2
24x Nivolumab	1
4x Nivolumab	1

Supplemental table 2: Detailed description of the response to treatment

Response to salvage and HD-ASCT/allo, split by three subgroups including ICI mono, combined with BV or chemotherapy

		ICI + BV		ICI + Chemotherapy		ICI + mono		Total	
		N	%	N	%	N	%	N	%
Best overall response to HD-ASCT	CR	3	100	21	95.5	8	100	32/33	97
	PR	0	0	1	4.5	0	0	1/33	3
Best overall response pre HD-ASCT	CR	1	33.3	19	61.3	1	10	21/44	47.7
	PR	2	66.7	10	32.3	7	70	19/44	43.2
	SD	0	0	2	6.5	1	10	3/44	6.8
	PD	0	0	0	0	1	10	1/44	2.3

Response to salvage and HD-ASCT/allo, split by exact regimen

		BV + Nivo		BV + Pembro		N-ICE		P-ICE		P-GVD		Nivo		Pembro		Total	
		N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
Best overall response to HD-ASCT	CR	2	100	1	100	1	100	2*	100	18	94.7	4	100	4	100	32/33	97
	PR	0	0	0	0	0	0	0	0	1	5.3	0	0	0	0	1/33	3
Best overall response pre HD-ASCT	CR	1	50	1	100	0	0	3	100	16	59.3	0	0	0	0	21/44	47.7
	PR	1	50	0	0	1	100	0	0	9	33.3	3	75	4	100	19/44	43.2
	SD	0	0	0	0	0	0	0	0	2	0	1	25	0	0	3/44	6.8
	PD	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1/44	2.3
Total		2	100	1	100	1	100	3	100	27	100	4	100	4	100	44	100

Response to salvage and HD-ASCT/allo in patients with one prior line of treatment, split by three subgroups including ICI mono, combined with BV or chemotherapy

		ICI + mono		ICI + BV		ICI + Chemotherapy		Total	
		N	%	N	%	N	%	N	%
Best overall response to HD-ASCT	CR	0	0	1	100	13	92.9	14/15	93.3
	PR	0	0	0	0	1	7.1	1/15	6.7
Best overall response pre HD-ASCT	CR	1*	100	1	100	11	55	13/22	59.1
	PR	0	0	0	0	8	40	8/22	36.4
	SD	0	0	0	0	1	5	1/22	4.5
	PD	0	0	0	0	0	0	0	0

Response to salvage and HD-ASCT/allo in patients with one prior line, exact regimens

		BV-Nivo		P-GVD		P-ICE		Pembrolizumab		Total	
		N	%	N	%	N	%	N	%	N	%
Best overall response to HD-ASCT	CR	1	100	13	92.9	0	0	0	0	14/15	93.3
	PR	0	0	1	7.1	0	0	0	0	1/15	6.7
Best overall response pre HD-ASCT	CR	1	100	10	52.6	1	100	1	100	13/22	59.1
	PR	0	0	8	42.1	0	0	0	0	8/22	36.4
	SD	0	0	1	5.3	0	0	0	0	1/22	4.5
	PD	0	0	0	0	0	0	0	0	0	0

Supplemental table 3: Immune related adverse events and deaths

Overview of immune related adverse events

Sex	Age at initial diagnosis	ASCT protocol	ASCT	irAE	PFS (m)	CTCAE	after cycle no (PD1-containing cycle)	Management	Outcome	PD1-reexposition
F	29	BEAM	Auto SCT	Thyreoiditis	6.8	°II	1	Carbimazole (intial hyperthyreosis), then switch to L-Thyoxin substitution (during hypothyreosis)	not resolved	No
F	21	BEAM	Auto SCT	Hepatitis	8	°III	2	140 mg Prednisolone (6d) / 100 mg (2d), no PD1-reexposition	resolved	No
F	68		No SCT	Pneumonitis	7	°III	4	60 mg Prednisolon (7 d), oxygen mask	resolved	No
M	59	Flu/Treo/ATG	Allo SCT	Pancolitis	75.5	°III	3	30d Prednisolone (start 100 mg/d).	resolved	Yes
M	44		No SCT	Pneumonitis	3.4	°III	1	34d Prednisolon (starting dose 1 mg/kg), oxygen mask	resolved	No

Overview of all deaths

Sex	**Age at initial diagnosis** (years)	**Age at death** (years)	**ASCT protocol**	**ASCT**	**Cause of death**	**Date of death**	**SV** (months)	**Progression**	**PFS** (months)
M	24	27	BEAM	Auto SCT	PD	18.11.2022	24.3	Yes	9.9
M	60	66	BEAM	Auto SCT	PD	08.03.2024	2.9	No	2.9
M	62	69	BEAM	Auto SCT	AML	26.06.2024	23.4	No	23.4
M	44	50		No SCT	unknown	24.12.2023	4.1	Yes	3.9