Glofitamab and epcoritamab for large B cell lymphoma: a real-world retrospective UK analysis of efficacy, tolerability, and impact of treatment sequencing

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Abstract

Glofitamab and epcoritamab are CD3xCD20 bispecific antibodies licensed for the treatment of relapsed/refractory large B-cell lymphoma (RR LBCL), yet real-world data are limited. We, therefore, analyzed data collected from 332 patients (219 prescribed glofitamab, 113 prescribed epcoritamab) across 34 UK centers (November 2023-May 2025). This high-risk cohort had received a median of two prior lines of treatment, 179 (55%) had primary refractory disease, 81 (25%) had Eastern Co-operative Oncology Group performance status (ECOG PS) ≥2, 152 (50%) had received prior chimeric antigen receptor T-cell

therapy, and 232 (78%) were ineligible for pivotal trials. Seven patients died before treatment initiation, one patient was yet to start treatment. Of 324 treated patients, 28% had cytokine release syndrome, predominantly grade 1/2 (82/90). Overall and complete response rates were 43% and 24%, respectively, while for trial-eligible patients the complete response rate was 43%. At a median follow-up of 10.0 months (interquartile range, 5.3-15.0), median progression-free survival (PFS) was 3.1 months (95% confidence interval [CI]: 2.5-4.2), and median overall survival (OS) was 6.9 months (95% CI: 4.9-10.8). For patients not completing cycle 2, 6-month OS was 4% (95% CI: 1-11%). The median duration of complete response was not reached. Refractoriness to a prior line of treatment (hazard ratio [HR]=2.89, 95% CI: 1.73-4.81, *P*=0.007), elevated lactate dehydrogenase (HR=2.62, 95% CI: 1.74-3.93. *P*=0.001), bendamustine exposure within 6 months (HR=1.62, 95% CI: 1.14-2.30, *P*=0.007) and ECOG PS 1 (HR=2.70, 95% CI: 1.52-4.79, *P*=0.001) or 2 (HR=6.49, 95% CI: 3.47-12.01, *P*<0.001) were associated with inferior PFS. This analysis shows that both glofitamab and epcoritamab are effective treatment options in RR LBCL, with durable complete responses achieved, while underscoring the importance of improved patient selection.

Introduction

The treatment of relapsed/refractory large B-cell lymphoma (RR LBCL) has been transformed by chimeric antigen receptor T-cell therapy (CAR-T).[1-5] However, patients with RR LBCL who are unsuitable for, unable to access, or who relapse after CAR-T have poor outcomes.[6,7] In recent years, following positive results from single-arm phase I/II trials, the CD3xCD20 bispecific antibodies (BsAb) glofitamab and epcoritamab have been rapidly integrated into clinical practice in the UK for patients who have received two or more prior lines of therapy, as permitted by regulatory licensing and reimbursement frameworks.[8,9] Epcoritamab and glofitamab are both routinely commissioned in the UK following approval by the National Institute for Health and Care Excellence (NICE) and the Scottish Medicines Consortium (SMC) in the publicly funded National Health Service which is free at point of access for patients. Odronextamab is licensed but is not currently commissioned in the UK.[10,11]

In registration trials, both glofitamab and epcoritamab demonstrated complete response (CR) rates of approximately 40% in heavily treated patients, many of whom had refractory disease and of whom approximately one-third had previously received CAR-T.[8,9] Follow-up analyses show good durability of responses to both antibodies, with the median duration of complete response (DoCR) being 29.8 months and 32 months for glofitamab and epcoritamab, respectively.[12,13] Some real-world data on BsAb have been published already,[14,15] but additional large cohort real-world data are needed.

Several disease-specific and patient-related characteristics have been shown to be associated with poorer outcomes following CAR-T, including poor Eastern Cooperative Oncology Group performance status (ECOG PS), elevated pre-treatment lactate dehydrogenase (LDH) concentration, extranodal involvement and large tumor volume.[16-20] To date, there is a paucity of data to guide prognosis and identify risk factors for response to BsAb in third-line treatment of LBCL. A subanalysis of the glofitamab registration study demonstrated that tumor metabolic volume is predictive of progression-free survival (PFS)[13] and a real-world data series identified CD20 negativity as predictive of poor outcomes, but other pretreatment predictors of response have not been identified.[14]

The aim of this UK real-world study was to characterize patients treated with glofitamab and epcoritamab, to assess safety and efficacy in the current treatment landscape, and to investigate factors influencing outcomes so that we can better understand treatment sequencing and patient selection.

Methods

This is a retrospective analysis of anonymized data from consecutive patients treated in 34 UK centers. Patients received glofitamab or epcoritamab outside of clinical trials between November 2023 and May 2025. Follow-up data were collected until July 2025. Data were collected from electronic records at local centers as part of a national service evaluation with local audit approval at individual sites. As a service evaluation, all data collection and handling respected the ethical standards set out in the UK. UK BsAb delivery spans academic and community hospitals, and step-up dosing – initially concentrated in tertiary centers – is now routinely undertaken in community sites, consistent with the British Society for Haematology (BSH) BsAb guidance.[21]

Response assessments were performed locally, with positron emission tomography (PET)/computed tomography (CT) or with plain CT imaging as per local guidelines. Cytokine release syndrome (CRS) and immune cell-associated neurotoxicity syndrome (ICANS) were graded according to American Society for Transplantation and Cellular Therapy consensus criteria,[22] and treatment for CRS and ICANS followed local protocols. Other toxicities were retrospectively graded according to Common Terminology Criteria for Adverse Events v5.0. All patients were managed in accordance with the respective summary of product characteristics including at least one overnight admission for CRS monitoring during cycle 1.

Statistical considerations

All patients commencing BsAb are included in the safety and efficacy evaluable populations. Baseline characteristics,

safety data and response rates were compared between treatment groups using χ^2 and Wilcoxon rank-sum tests for categorical and continuous variables, respectively. Duration of response (DoR) and DoCR were measured from the date of best response. PFS and overall survival (OS) were measured from the date of first BsAB dose. DoR, DoCR, PFS and OS were assessed using Kaplan-Meier survival analysis and compared between treatment groups using Cox regression. Two separate multivariable analyses were performed to evaluate predictors of PFS and CR rate. PFS was analyzed using Cox regression and CR rate using logistic regression. To avoid overfitting, forward stepwise variable selection (*P* value for inclusion 0.05) was used. Missing data were handled using complete case analysis, whereby observations with missing values for a given variable were excluded from analyses involving that variable.

Results

Baseline characteristics

Three hundred thirty-two patients were intended to receive either epcoritamab (N=113) or glofitamab (N=219) in 34 hospitals in the UK between November 2023 and May 2025 (*Online Supplementary Table S1*). Most patients received treatment on a nationally commissioned basis but 96 (29%) received treatment via compassionate access schemes prior to routine commissioning.

The patients' baseline characteristics are described in Table 1. The median number of lines of prior therapy was two (range, 1-7) with 47% receiving BsAb in fourth-line treatment or beyond. Of all patients, 80% were refractory (defined as having disease that did not respond to, or that progressed within 6 months after completing a line of therapy) to their last line of therapy, while 55% were refractory to their first line of therapy. Sixty percent of patients had a histological diagnosis of diffuse LBCL not otherwise specified with transformed follicular lymphoma the second commonest diagnosis (18%); a wider range of other subtypes of non-Hodgkin lymphoma were treated than were included in the pivotal trials (Table 1). Baseline characteristics were compared to the inclusion criteria for the registrational glofitamab trial.[9]

Of the 299 patients for whom data allowed comparison, 26% (50/196) of glofitamab-treated patients and 17% (17/103) of epcoritamab-treated patients met eligibility criteria. Reasons for not meeting eligibility criteria included cytopenias (N=144), ECOG PS ≥2 (N=81), specific co-morbidities (N=51), active or recent infection requiring hospital admission (N=49), and histological diagnosis (N=38).

Important differences in the baseline characteristics of patients who received epcoritamab or glofitamab were identified. Patients treated with epcoritamab were more likely to have an ECOG PS ≥2 (40% *vs*. 17%, *P*<0.001) and were less likely to have previously received CAR-T (37% *vs*. 56%, *P*=0.002).

Treatment received

The median follow-up was 10.0 months (interquartile range [IQR], 5.3-15.0) and the data cut-off date was August 29, 2025. Of 332 patients intended to receive treatment, eight did not commence BsAb (Figure 1) and so are excluded from subsequent analyses; 212 and 112 patients given glofitamab and epcoritamab, respectively, were included in the treatment efficacy and safety analyses.

The median number of full cycles of glofitamab completed was three (range, 0-12). Thirty-one patients (16%) did not complete the second cycle of treatment. For patients completing the second cycle, the median number of cycles completed was four (range, 2-12). The commonest reason for terminating treatment was progression of disease (65%). Forty (21%) of 194 patients completed 12 cycles of fixed-duration treatment with 18 still receiving treatment at the time of analysis.

The median number of full cycles of epcoritamab completed was two (range, 0-25). Forty-six (47%) patients did not complete cycle 2 of treatment. For patients completing the second cycle, the median number of cycles completed was four (range, 2-25). Progression was the most common reason for termination (79%). Fifteen patients were still on treatment at the time of analysis; four (4%) received at least 12 cycles.

Safety

Safety data are presented in *Online Supplementary Table S2*. For patients receiving glofitamab, any grade CRS occurred in 59/212 (28%); the maximum grade was 1 in 30 (14%), grade 2 in 22 (10%), and grade ≥3 in seven patients (3%); no grade 4 events were reported, and one patient had a fatal grade 5 CRS event. In this patient, at the onset of CRS a clinical decision was taken not to escalate the care with admission to an intensive care unit (ICU) because of concurrent disease progression and poor performance status (see additional information in the *Online Supplementary Data*). Tocilizumab was administered to 32/195 (16%) patients; only one dose was required in 22/32 (69%) cases. Any grade ICANS occurred in 9/212 (4%) patients treated with glofitamab (grade 1, 3% [N=6], grade 2, <1% [N=1], grade ≥3, <1% [N=1]). ICU admission was required in 10/208 (5%) patients receiving glofitamab, primarily for CRS or sepsis management. Infections were reported in 102/212 (48%) patients (grade ≥3, 57/212, 27%), and grade ≥3 neutropenia was observed in 51/208 (25%). Treatment delays (most commonly due to slow blood count recovery or infections) were required in 61/204 (30%) patients.

For patients receiving epcoritamab, any grade CRS occurred in 31/112 (28%); the maximum grade was 1 in 21 (19%) patients, grade 2 in nine (8%) patients, and there was one (1%) grade 3 event. ICANS was reported in 6/112 (5%) patients treated with epcoritamab (grade 1, 3% [N=3], grade 2, 1% [N=1], grade 3, 2% [N=2]); tocilizumab was administered to 14/107 (13%) with 10/14 (71%) requiring only one dose and

Table 1. Patients' baseline characteristics.

Characteristics	All N=332	Glofitamab N=219	Epcoritamab N=113	*P*
Age, years, median (range) [IQR]	68 (18-93) [58-7]	68 (18-90) [57-76]	68 (34-93) [59-76]	0.8
Sex, N (%) Female Male	 119 (36) 213 (64)	 77 (35) 142 (65)	 42 (37) 71 (63)	0.7
ECOG PS, N (%) 0 1 2 3 4 Unknown (excluded from denominator)	 51 (15) 198 (60) 74 (22) 5 (2) 2 (1) 2	 41 (19) 140 (65) 34 (16) 2 (1) 0 2	 10 (9) 58 (51) 40 (35) 3 (3) 2 (2) 0	<0.001
Diagnosis, N (%) DLBCL Transformed follicular lymphoma HGBCL Transformed mantle cell lymphoma T-cell/histiocyte-rich large B-cell lymphoma Richter syndrome Follicular lymphoma T-LPL Primary mediastinal B-cell lymphoma PTLD Burkitt lymphoma Gray zone lymphoma Unknown (excluded from denominator)	 198 (60) 61 (18) 27 (8) 12 (4) 7 (2) 6 (2) 6 (2) 5 (2) 2 (1) 5 (2) 1 (<1) 1 (<1) 1	 129 (59) 43 (20) 15 (7) 11 (5) 6 (3) 5 (2) 2 (1) 2 (1) 2 (1) 1 (<1) 1 (<1) 1 (<1) 1	 69 (61) 18 (16) 12 (11) 1 (1) 1 (1) 1 (1) 4 (4) 3 (3) 0 4 (4) 0 0 0	0.07
International Prognostic Index, N (%) 0 1 2 3 4 5 Unknown (excluded from denominator)	 4 (1) 32 (10) 100 (31) 94 (29) 78 (24) 13 (4) 11	 4 (2) 20 (10) 65 (31) 62 (30) 51 (24) 8 (4) 9	 0 12 (11) 35 (32) 32 (29) 27 (24) 5 (5) 2	0.8
Bulky disease (>7.5 cm), N (%) Yes No Unknown (excluded from denominator)	 85 (26) 245 (74) 2	 51 (24) 166 (77) 2	 34 (30) 79 (70) 0	0.2
N of prior lines of treatment, median (range) [IQR]	2 (1-7) [2-3]	2 (1-7) [2-3]	2 (1-5) [2-3]	0.5
Refractory to first-line treatment, N (%) Yes No Unknown (excluded)	 179 (55) 148 (45) 5	 116 (54) 99 (46) 4	 63 (56) 49 (44) 1	0.7
Refractory to most recent line, N (%) Yes No Unknown (excluded from denominator)	 264 (80) 65 (20) 3	 177 (82) 39 (18) 3	 87 (77) 26 (23) 0	0.3
Prior CAR-T, N (%) Yes No Unknown (excluded from denominator)	 152 (50) 155 (50) 25	 115 (56) 92 (44) 12	 37 (37) 63 (63) 13	0.002
Prior bendamustine, N (%) Yes No	 160 (48) 172 (52)	 106 (48) 113 (52)	 54 (48) 59 (52)	0.9

Continued on following page.

Characteristics	All N=332	Glofitamab N=219	Epcoritamab N=113	*P*
Lymphocyte count, x10^9/L, median (range)	0.62 (<0.1-18.97)	0.61 (0.1-18.97)	0.62 (<0.1-16.7)	0.4
<0.5x10^9/L, N (%)	128 (39)	83 (41)	45 (43)	0.7
≥0.5x10^9/L, N (%)	180 (54)	121 (59)	59 (57)	
Unknown (excluded), N	24	15	9	
LDH, U/L, median (range)	323 (78-19,065)	325 (78-19,065)	311 (149-17,789)	0.6
≤ULN, N (%)	75 (27)	48 (26)	27 (28)	0.7
>ULN, N (%)	208 (74)	140 (74)	68 (72)	
Unknown (excluded), N	49	31	18	
Trial eligible, N (%)				
Yes	67 (22)	50 (26)	17 (17)	0.08
No	232 (78)	146 (75)	86 (84)	
Unknown (excluded from denominator)	33	23	10	

CAR-T: chimeric antigen receptor T-cell therapy; DLBCL: diffuse large B-cell lymphoma; ECOG PS: Eastern Cooperative Oncology Group performance status; HGBCL: high-grade B-cell lymphoma; IQR: interquartile range; LDH: lactate dehydrogenase; PTLD: post-transplant lymphoproliferative disorder; T-LPL: transformed lymphoplasmacytic lymphoma; ULN: upper limit of normal.

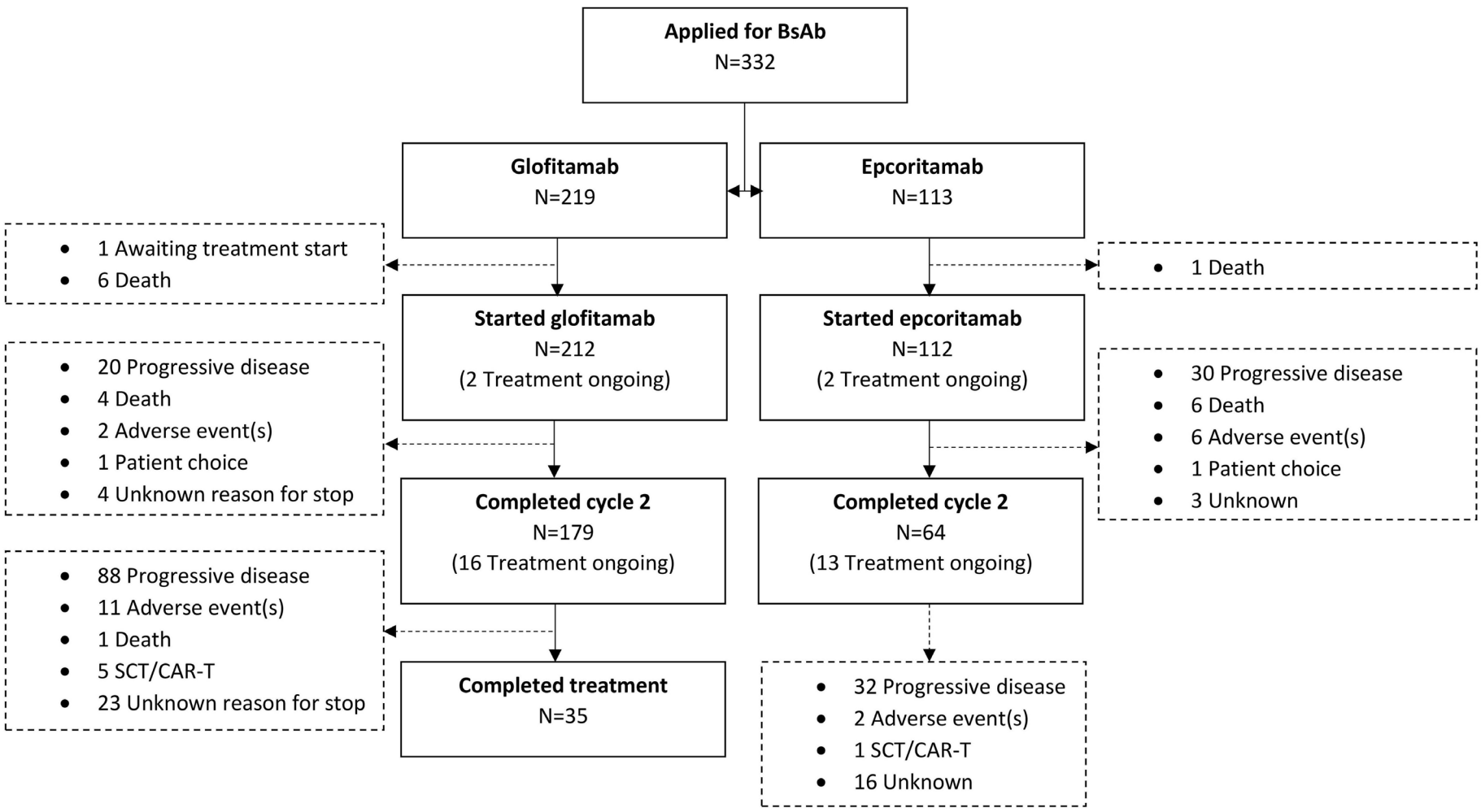

Figure 1. Flow chart of the patients' disposition in the study. CAR-T: chimeric antigren receptor T-cell therapy; SCT: stem cell transplantation.

5/106 (5%) needing ICU admission, most commonly with sepsis or for CRS management. Infections were reported in 62/112 (55%) patients (grade ≥3 36/112, 32%). Treatment delays were required in 47/110 (43%) patients, primarily due to infection or cytopenias.

Immunoglobulin replacement was administered to 29 patients (9%) in the whole cohort, predominantly in patients exposed to CAR-T (N=21). Antimicrobial prophylaxis was employed in 322/324 (99%) patients, predominately with co-trimoxazole and acyclovir as per BSH BsAb guidance.[21]

Efficacy

For glofitamab-treated patients the overall response rate was 48% and the CR rate was 27% (Table 2). Among patients who completed cycle 2, 31% achieved CR and for the subset of patients who would have met the trial inclusion criteria 47% achieved CR. The median DoR was 18.8 months (95% CI: 8.1-not evaluable [NE]), the median DoCR has not yet been reached, and the 12-month DoCR was 84% (95% CI: 66-93%) (Figure 2). The median PFS was 3.4 months (95% CI: 2.5-5.5) with a 12-month PFS of 31% (95% CI: 24-39); for those

completing cycle 2, the median PFS was 5.5 months (95% CI: 3.2-8.9), while for those who would have been eligible for the pivotal trials the PFS was 8.9 months (95% CI 4.3-21.2) (*Online Supplementary Table S3*, *Online Supplementary Figure S1*). The median OS was 10.0 months (95% CI: 6.2-12.9) with a 12-month OS of 46% (95% CI: 38-53%); for trial-eligible patients, the median OS was 20.3 months (95% CI: 13.3-NE) (*Online Supplementary Figure S2*).

For epcoritamab-treated patients the overall response rate was 33% and the CR rate was 17%; for patients who received ≥2 cycles of treatment the overall response and CR rates were 53% and 30%, respectively, and for those who would

Table 2. Response data.

Variables	**All N=324**	**Glofitamab N=212**	**Epcoritamab N=112**	**Glofitamab *vs.* epcoritamab**
Best response, N (%) CR/CMR PR/PMR Stable disease Progressive disease Died before assessment Unknown (excluded)	 73 (24) 58 (19) 8 (3) 157 (51) 10 (3) 18	 55 (27) 41 (20) 3 (1) 97 (48) 5 (2) 11	 18 (17) 17 (16) 5 (5) 60 (57) 5 (5) 7	-
Overall response rate, % (95% CI) CR rate, % (95% CI)	43 (37-49) 24 (19-29)	48 (41-55) 27 (21-34)	33 (24-43) 17 (10-26)	*P*=0.02* *P*=0.05*
DoR, months, median (95% CI) 12-month DoR rate, % (95% CI)	18.8 (18.3-NE) 63 (51-73)	18.8 (8.1-NE) 61 (47-72)	Not reached 71 (45-86)	HR=1.12 (95% CI: 0.46-2.72), *P*=0.8
DoCR, months, median (95% CI) 12-month DoCR, % (95% CI)	Not reached 88 (73-95)	Not reached 84 (66-93)	Not reached 100 (NE)	*P*=0.2**
Started cycle 2	**All N=243**	**Glofitamab N=179**	**Epcoritamab N=64**	**Glofitamab *vs.* epcoritamab**
Best response CR/CMR PR/PMR Stable disease Progressive disease Died before assessment Unknown (excluded)	 72 (31) 55 (24) 5 (2) 97 (42) 1 (1) 13	 54 (32) 41 (24) 1 (1) 73 (43) 1 (1) 9	 18 (30) 14 (23) 4 (7) 24 (40) 0 4	-
Overall response rate, % (95% CI) CR rate, % (95% CI)	55 (49-62) 31 (25-38)	56 (48-63) 32 (25-39)	53 (40-66) 30 (19-43)	*P*=0.7* *P*=0.8*
DoR, months, median (95% CI) 12-month DoR rate, % (95% CI)	18.8 (18.3-NE) 64 (52-74)	18.8 (8.7-NE) 62 (48-73)	Not reached 73 (46-88)	HR=1.27 (95% CI: 0.48-3.33), *P*=0.6
DoCR, months, median (95% CI) 12-month DoCR, % (95% CI)	Not reached 90 (74-96)	Not reached 86 (67-95)	Not reached 100 (NE)	*P*=0.3**
Trial eligible	**All N=67**	**Glofitamab N=50**	**Epcoritamab N=17**	**Glofitamab *vs.* epcoritamab**
Best response CR/CMR PR/PMR Stable disease Progressive disease Unknown (excluded)	 27 (43) 11 (17) 3 (5) 22 (35) 4	 22 (47) 9 (19) 1 (2) 15 (32) 3	 5 (31%) 2 (13%) 2 (13%) 7 (44%) 1	-
Overall response rate, % (95% CI) CR rate, % (95% CI)	60 (47-72) 43 (30-56)	66 (51-79) 47 (32-62)	44 (20-70) 31 (11-59)	*P*=0.1* *P*=0.3*
DoR, months, median (95% CI) 12-month DoR rate, % (95% CI)	18.3 (6.0-NE) 68 (48-82)	18.3 (6.0-NE) 71 (48-NE)	4.2 (2.4-NE) FU too limited	HR=0.49 (95% CI: 0.10-2.38), *P*=0.4
DoCR, months, median (95% CI) 12-month DoCR, % (95% CI)	18.8 (18.3-NE) 83 (56-94)	18.8 (18.3-NE) 81 (51-93)	Not reached FU too limited	*P*=0.5**

**P* value calculated using the χ^2 test; ***P* value calculated using a log-rank test. 95% CI: 95% confidence interval; CMR: complete molecular response; CR: complete response; DoCR: duration of complete response; DoR: duration of response; FU: follow-up; HR: hazard ratio; NE: not evaluable; PMR: partial molecular response; PR: partial response.

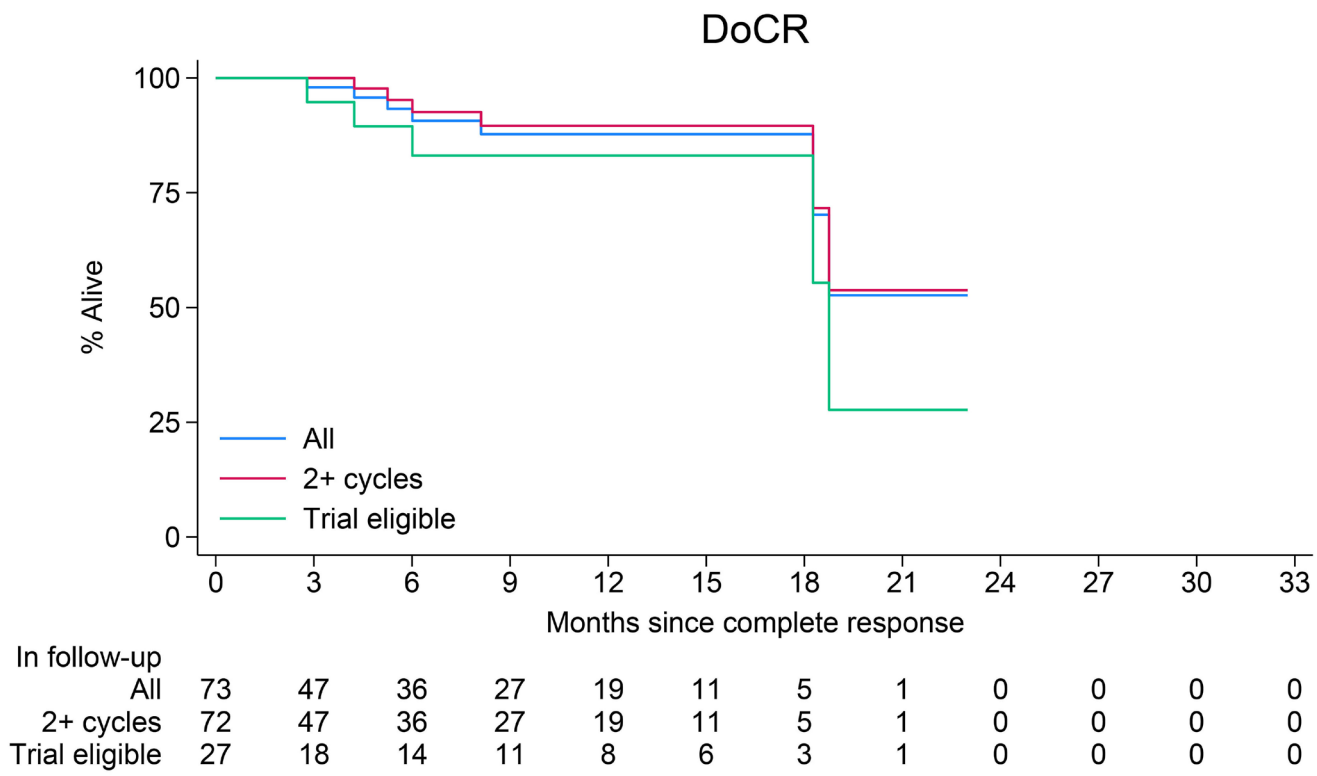

Figure 2. Duration of complete response (DoCR) of all patients.

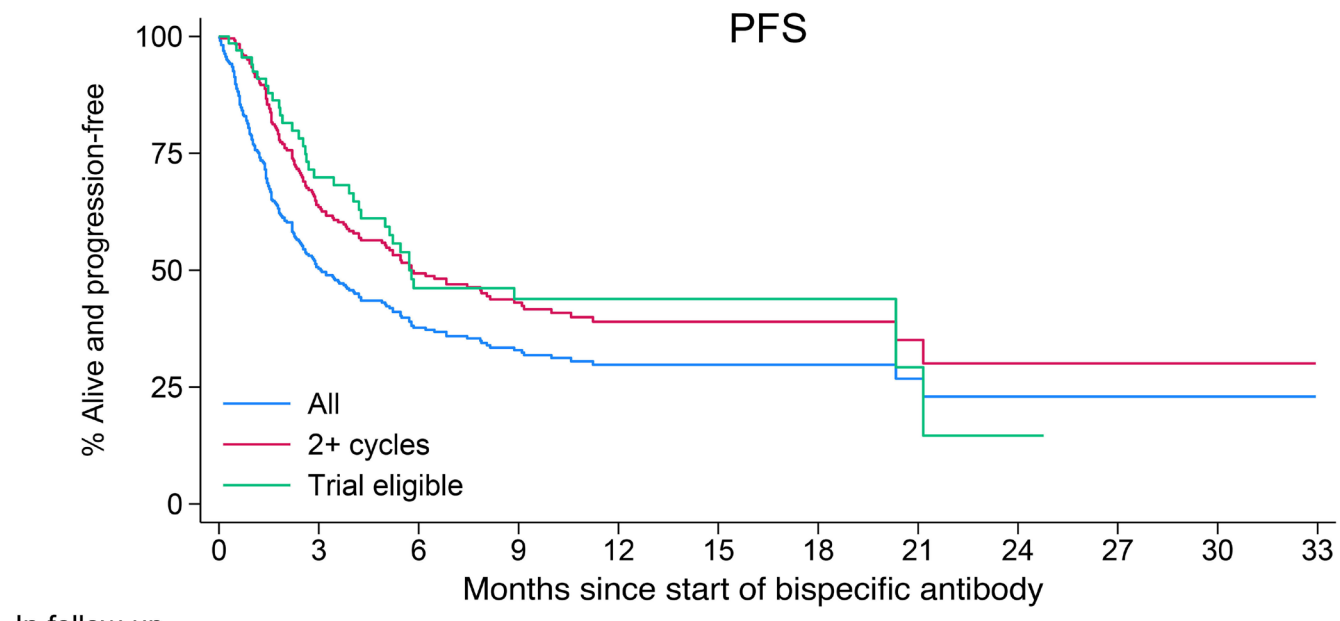

Figure 3. Progression-free survival (PFS) of all patients.

have been eligible for pivotal trials the overall response rate was 44% and the CR rate was 31%. The median DoR was not reached and the 12-month DoR was 71% (95% CI: 45-86%) with a 12-month DoCR of 100%. The median PFS was 2.9 months (95% CI: 1.7-4.3) (*Online Supplementary Table S3*, *Online Supplementary Figure S3*), and the median OS was 4.7 months (95% CI 2.9-6.8) (*Online Supplementary Figure S4*). For those starting cycle 2, the median PFS was 6.8 months (95% CI: 5.0-NE) and the median OS was not yet reached.

For all patients the median PFS was 3.1 months (95% CI: 2.5-4.2) (Figure 3) and the median OS was 6.9 months (95% CI: 4.9-10.8) (Figure 4).

For patients who did not complete cycle 2 with either BsAb, PFS and OS were particularly poor with the 3-month and 6-month OS, 13% (95% CI: 6-21) and 4% (95% CI: 1-11), respectively (Figure 5, *Online Supplementary Figure S5*).

In multivariable analysis of factors influencing CR, LDH above the upper limit of normal (>ULN) (odds ratio [OR]=0.35, 95% CI: 0.18-0.68, *P*=0.002), refractoriness to the prior line of therapy (OR=0.23, 95% CI: 0.11-0.46, *P*<0.001) and exposure to bendamustine within 6 months (OR=0.42, 95% CI: 0.19-0.94, *P*=0.03) were independently associated with a lower probability of achieving CR (*Online Supplementary Table S4*). Baseline characteristics by prior bendamustine exposure are detailed in *Online Supplementary Table S5*.

In multivariable analysis of factors associated with PFS (Table 3), patients with transformed follicular lymphoma had a significantly better outcome (HR=0.40, 95% CI: 0.24-0.68,

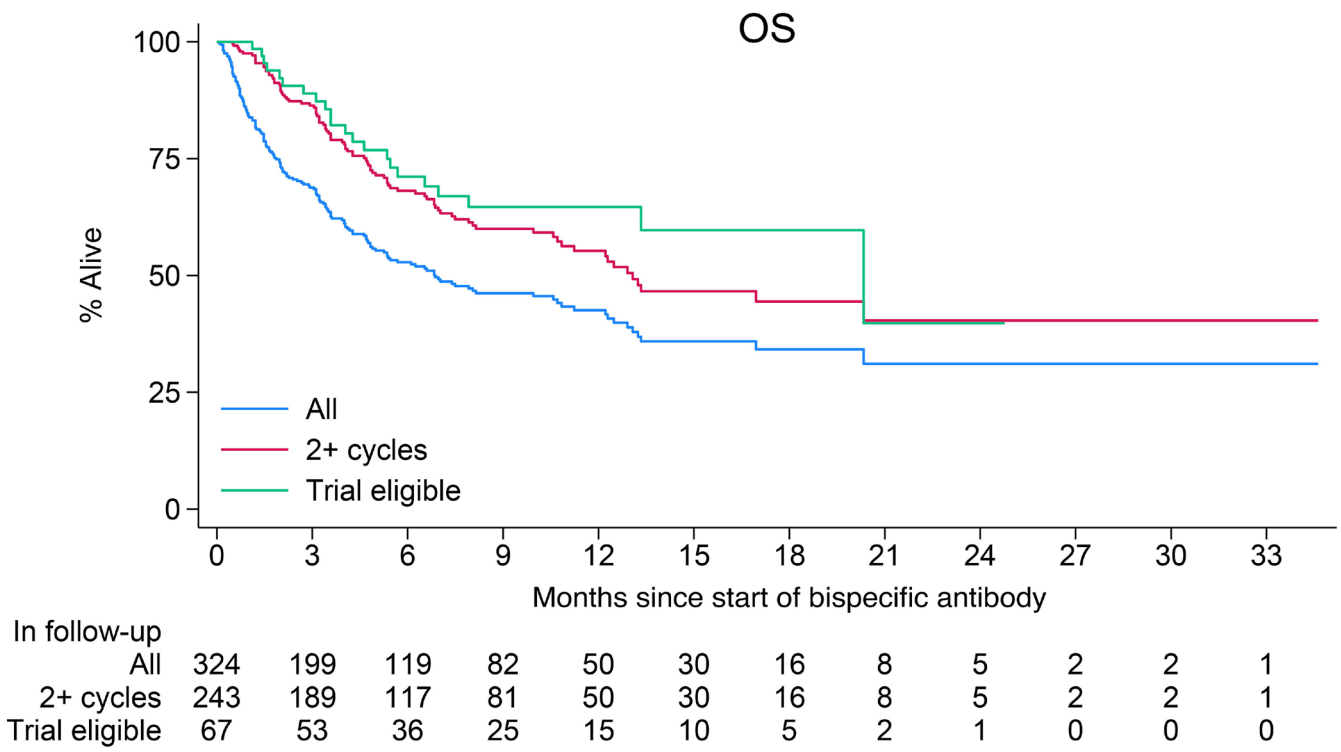

Figure 4. Overall survival (OS) of all patients.

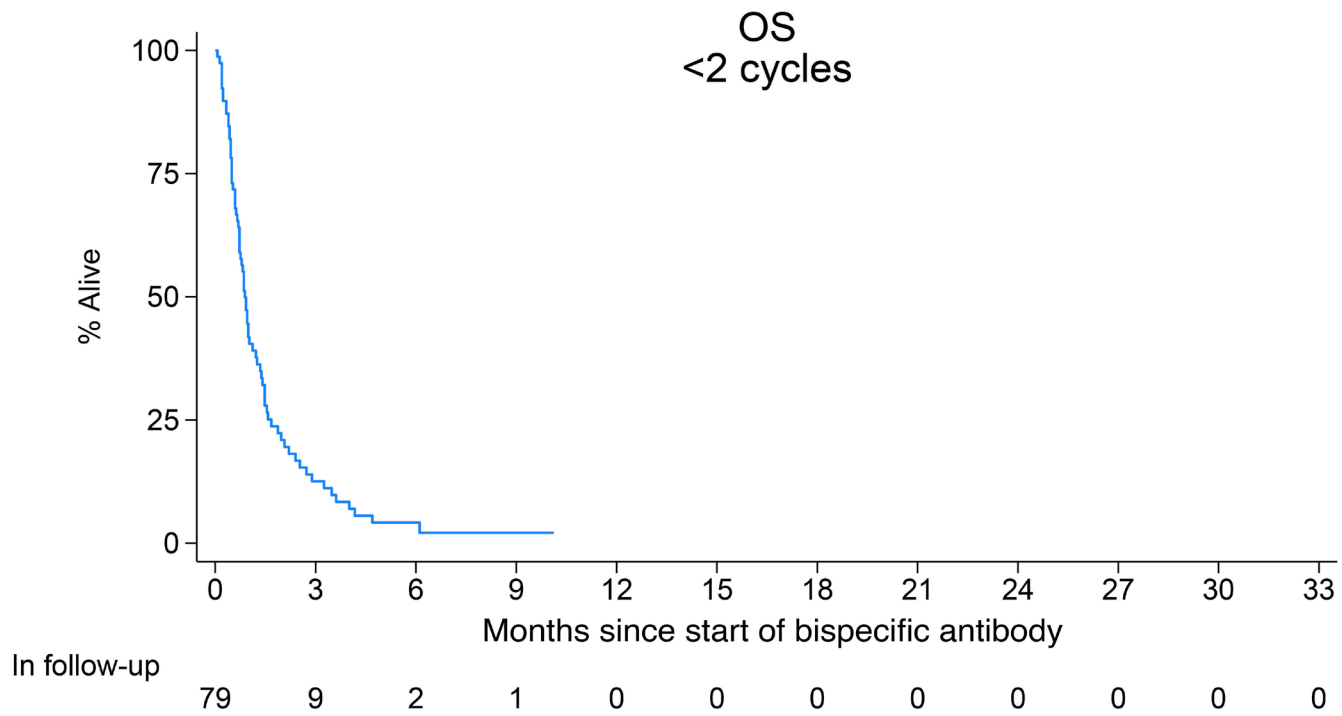

Figure 5. Overall survival (OS) of patients completing less than two cycles of treatment with a bispecific antibody.

P=0.001). Factors associated with inferior PFS were LDH >ULN (HR=2.62, 95% CI: 1.74-3.93, *P*<0.001), refractoriness to the prior line of therapy (HR=2.89, 95% CI: 1.73-4.81, *P*<0.001), ECOG PS 1 (HR=2.70, 95% CI: 1.52-4.79, *P*=0.001) or ECOG PS 2 (HR=6.49, 95% CI: 3.47-12.01, *P*<0.001), and exposure to bendamustine within 6 months (HR=1.62, 95% CI: 1.14-2.30, *P*=0.007). A univariate analysis restricted to patients receiving BsAb within 6 months of previous therapy (N=213) also found that patients who received bendamustine within 6 months of BsAb had poorer PFS (bendamustine received within 6 months *vs*. never received: HR=1.58, 95% CI: 1.12-2.23, *P*=0.01).

Prior CAR-T exposure was not independently associated with inferior PFS or CR rate on multivariate analysis. One-year PFS was 38% (95% CI: 29-47) in CAR-T-naïve patients and 22% (95% CI: 15-30) in patients who had been exposed to CAR-T (HR=1.31, 95% CI: 0.98-1.74, *P*=0.07) (*Online Supplementary Figure S6*). The CR rate was 27% (95% CI: 42-60) in CAR-T-naïve patients and 22% (95% CI: 16-30) in CAR-T-exposed patients (*P*=0.3). The 1-year OS was 50% (95% CI: 40-59) in CAR-T-naïve patients and 38% (95% CI: 29-47) in CAR-T-exposed patients (HR=1.15, 95% CI: 0.84-1.59, *P*=0.4) (*Online Supplementary Figure S7*).

Of the 107 patients (82 glofitamab, 25 epcoritamab) who had received CAR-T as the prior treatment line before BsAb, a shorter time from CAR-T infusion to BsAb start was associated with inferior PFS both as a continuous variable (HR for 1 month increase= 0.94, 95% CI: 0.89-1.00, *P*=0.05) and when early CAR-T failure occurred (<3 months from infusion) (HR=0.51, 95% CI: 0.32-0.83, *P*=0.005) (*Online Supplementary Figure S8*) which was also associated with inferior OS (HR=0.46, 95% CI: 0.26-0.79, *P*=0.005) (*Online Supplementary Figure S9*). Time to CAR-T failure was significantly associated with best response to BsAb both as a continuous variable for complete response (HR for 1 month increase=1.13, 95%

Table 3. Multivariate analysis of progression-free survival.

Variables	N	Univariable HR (95% CI), *P*	Multivariable (N=252*) HR (95% CI), *P*	Multivariable (N=256**) HR (95% CI), *P*
Treatment (glofitamab *vs.* epcoritamab)	324	0.85 (0.64-1.13), *P*=0.3	-	-
Age (5-year increase)	324	0.97 (0.92-1.02), *P*=0.2	-	-
Sex (male *vs.* female)	324	1.31 (0.98-1.76), *P*=0.07	-	-
Stage 3 *vs.* 1-2 4 *vs.* 1-2	323	 1.16 (0.70-1.94), *P*=0.6 1.60 (1.09-2.34), *P*=0.02	 - 1.59 (1.12-2.26), *P*=0.01	 0.94 (0.51-1.75), *P*=0.9 1.54 (0.99-2.38), *P*=0.05
Diagnosis (T-FL *vs.* all others)	323	0.55 (0.36-0.83), *P*=0.005	0.40 (0.24-0.67), *P*=0.001	0.40 (0.24-0.67), *P*=0.001
International Prognostic Index 2 *vs.* 0-1 3 *vs.* 0-1 4-5 *vs.* 0-1	314	 1.55 (0.88-2.73), *P*=0.1 1.85 (1.04-3.26), *P*=0.04 2.82 (1.61-4.93), *P*<0.001	-	-
Bulky disease (yes *vs.* no)	323	1.64 (1.21-2.22), *P*=0.001	-	-
N of prior lines of therapy 3 *vs.* <3 4 *vs.* <3 ≥5 *vs.* <3	324	 1.20 (0.88-1.64), *P*=0.2 1.03 (0.68-1.57), *P*=0.9 0.87 (0.44-1.72), *P*=0.7	-	-
ECOG PS 1 *vs.* 0 ≥2 *vs.* 0	323	 1.77 (1.12-2.79), *P*=0.02 3.78 (2.33-6.14), *P*<0.001	 2.72 (1.51-4.91), *P*=0.001 6.52 (3.47-12.25), *P*<0.001	 2.70 (1.52-4.79), *P*=0.001 6.46 (3.47-12.01), *P*<0.001
Refractory to most recent line of treatment	322	2.81 (1.83-4.31), *P*<0.001	2.58 (1.57-4.22), *P*<0.001	2.89 (1.73-4.81), *P*<0.001
Prior CAR-T	299	1.31 (0.98-1.74), *P*=0.07	1.40 (1.00-1.95), *P*=0.05	1.40 (1.00-1.97), *P*=0.05
Last bendamustine >6 months *vs.* never ≤6 months *vs.* never	318	 1.01 (0.67-1.52), *P*=0.9 1.93 (1.42-2.62), *P*<0.001	 - 1.53 (1.10-2.14), *P*=0.01	 1.23 (0.72-2.10), *P*=0.4 1.62 (1.14-2.30), *P*=0.007
Bendamustine cycles (1-cycle increase)	296	1.05 (0.98-1.11), *P*=0.2	-	-
LDH >ULN (yes *vs.* no)	279	2.32 (1.59-3.39), *P*<0.001	2.68 (1.78-4.03), *P*<0.001	2.62 (1.74-3.93), *P*<0.001
Lymphocytes <0.5x10^9/L (yes *vs.* no)	324	1.48 (1.13-1.96), *P*=0.005	-	-

*Only includes patients who had complete data for every variable in the table except 'Bendamustine cycles' which was excluded because of the strong correlation with timing of last bendamustine treatment. **Only includes patients who had complete data for the variables selected by forward stepwise selection. 95% CI: 95% confidence interval; CAR-T: chimeric antigen receptor T-cell therapy; ECOG PS: Eastern Cooperative Oncology Group performance status; HR: hazard ratio; LDH: lactate dehydrogenase; T-FL: transformed follicular lymphoma; ULN: upper limit of normal.

CI: 1.03-1.25, *P*=0.01) and for overall response (HR for 1 month increase=1.19, 95% CI: 1.06-1.35, *P*=0.005). Early CAR-T failure was also significantly associated with best response to BsAb for both complete response (HR=2.53, 95% CI: 1.01-6.33, *P*=<0.001), and for overall response (HR=2.93, 95% CI: 1.29-6.65, *P*=0.01).

CAR-T-naïve patients were more likely to be older, have a higher International Prognostic Index, have received fewer prior lines of therapy, not to have been refractory to their first-line treatment and less likely to have received prior bendamustine.

Predictive factors associated with not completing cycle 2 included ECOG PS >1 (OR=0.09, 95% CI: 0.02-0.42, *P*=0.002), bendamustine within 6 months (OR=0.48, 95% CI: 0.24-0.99, *P*=0.05), LDH >ULN (OR=0.15, 95% CI: 0.05-0.43, *P*<0.001), and epcoritamab treatment (OR=0.27, 95% CI: 0.14-0.53, *P*<0.001).

Of seven patients with a diagnosis of T-cell/histiocyte-rich LBCL (6 treated with glofitamab, 1 with epcoritamab) none responded to treatment with best responses being progressive disease (6/7) and stable disease (1/7).

Of 14 patients with active CNS involvement at the time of BsAb treatment initiation. CR was reached in 4/14 (29%) and PR in a further 5/14 (36%); the other 5/14 (36%) had progressive disease.

Discussion

We report here a retrospective real-world series of 324 patients treated with epcoritamab or glofitamab for RR

LBCL in the UK. This is the largest series of comprehensive, real-world data on patients treated with BsAb for RR LBCL published to date. We demonstrate a similar toxicity profile to those in the published trials, high efficacy with durable remissions, and identify factors influencing response and informing treatment decisions.

In this series, most patients had high-risk features with nearly half of the patients receiving treatment in fourth line or beyond, and 50% having received prior CAR-T, a high-risk population for whom life expectancy would be anticipated to be short in the absence of effective therapy. Furthermore, 78% would not have met trial eligibility criteria.

It is important to highlight that there were significant differences in baseline characteristics between those treated with the two BsAb. Patients treated with epcoritamab were more likely to have higher ECOG PS score, and less likely to have previously received CAR-T than patients treated with glofitamab. Reasons for these differences may include a more permissive ECOG PS for epcoritamab approval and reimbursement but other reasons influencing the choice of BsAb were not formally investigated. Both drugs are routinely commissioned and reimbursed in the UK for patients with RR LBCL who have received two or more lines of therapy. More patients were treated with glofitamab, possibly reflecting earlier reimbursement and availability. Commissioning in the UK permits use in patients with ECOG PS 0-2 for epcoritamab whereas glofitamab is restricted to patients with ECOG PS 0-1 as per the registration trials and this may have influenced the use of epcoritamab in a frailer cohort of patients who were less likely to have been suitable for CAR-T. The inherent differences in the populations treated in this study prevents any comparison of efficacy and safety between the two products.

The safety data presented here closely mirror those of the clinical trials despite being used in predominantly trial-ineligible patients. The rate of any grade CRS was 28% for both antibodies with this toxicity being grade ≥2 in about 13% of patients for both. ICANS events were rare. Only 5% of patients needed care in an ICU during treatment with either drug. Infections were the commonest reported toxicity in this heavily pre-treated population, highlighting the need to be vigilant in monitoring for infections and ensuring adequate vaccination and antimicrobial prophylaxis. Overall, these safety data confirm that BsAb can be delivered safely in a wide range of hospitals. The geographical spread of units and community hospitals offering BsAb emphasizes deliverability in hospitals close to the patients' homes. This is important for patient experience and is a key difference between delivery of BsAb and CAR-T, which remains restricted to larger hospitals.

Achieving CR is important in LBCL and the registration trials have focused on CR rate and the durability of the remissions. The overall CR rate in this study of 24% is inferior to the published trial data but when considering use as monotherapy in such a heavily pre-treated, refractory population for whom there are very few other treatment options, BsAB represents an effective treatment choice. When analysis is restricted to patients who would have met trial eligibility criteria, the CR rate is comparable to that seen in the registration trials (43%).

We identified that a proportion of patients did not complete the second cycle of BsAb, most commonly because of progression of disease and clinical deterioration during cycle 1. Numerically more patients treated with epcoritamab were unable to complete cycle 2 than those treated with glofitamab, again reflecting differences in these populations. Outcomes for these patients were very poor, with a 3-month OS of 13% (95% CI: 6-21). We performed an exploratory analysis of outcomes for patients completing cycle 2 and identified outcomes that were close to the published trial data for these patients. Our finding that up to 47% of patients do not complete cycle 2 indicates that we need to improve the selection of patients or investigate ways to improve responses for high-risk patients. Unlike the delivery of CAR-T, BsAb are available immediately with no requirement to wait for funding approvals, apheresis slots and manufacturing timelines. This difference in the patients' pathway may lead to differences in patients being selected for each approach and may explain why real-world efficacy data for CAR-T-infused patients is closer to the trial data than we see in this and other series of patients treated with BsAb in the real-world setting.[14,16,23]

In a multivariate analysis of factors impacting CR, refractoriness to the last line of therapy and elevated LDH were independently associated with reduced chance of attaining CR. Patients with a diagnosis of transformed follicular lymphoma were significantly more likely to achieve CR. These findings are similar to predictive factors identified in real-world CAR-T studies.[16,24]

Durability of remissions was very long in our cohort with the median DoCR not reached for patients treated with either antibody and a 12-month DoCR of 88% overall. This is an important finding and although the follow-up is too short at present to understand the curative potential of these drugs in the real-world setting, it is encouraging that few relapses have been observed to date in those who achieved CR.

Survival data in our series showed a median PFS of 3.4 and 2.9 months for glofitamab and epcoritamab, respectively, with median OS of 10 and 4.7 months, respectively. Given the differences in the patient populations we are unable to interpret any differences in survival data between the two antibodies. The median PFS and OS for patients who completed cycle 2 and those who were trial-eligible were superior to those of the whole population, again highlighting a need to improve selection of patients and explore alternative strategies for those most likely to progress prior to completing cycle 2.

In a multivariate analysis of risk factors for PFS, trans-

formed follicular lymphoma was associated with improved PFS; refractoriness to last line of therapy, higher ECOG PS, elevated LDH and prior bendamustine were independently associated with inferior PFS.

Whereas previous studies, including the epcoritamab NHL1 study, did not identify prior bendamustine as a risk factor for impaired response,[8,14,15,25,26] we did detect this in our larger cohort in which 160 (48%) patients had previously been exposed to bendamustine (HR=1.69, 95% CI: 1.18-2.41; *P*=0.004). Patients who had received bendamustine within 6 months of BsAb had an inferior PFS and reduced chance of attaining CR. This novel finding should be interpreted with caution and we were unable to explore reasons for poor response in bendamustine-exposed patients, but they may include impaired T-cell fitness or confounding factors.

The PFS for patients previously treated with CAR-T was numerically lower than for CAR-naïve patients but the difference did not reach statistical significance (HR=1.40, 95% CI: 1.00-1.97, *P*=0.051). Previous trial data have indicated similar outcomes for CAR-T-exposed patients.[9,27,28] As in other series, timing of relapse after CAR-T appears to be prognostic, with shorter time to CAR-T failure being associated with inferior response rates,[29] PFS and OS. Better treatments are needed for patients with early relapse after CAR-T.

The strengths of our study are that it involved a large cohort that captured most patients treated in the UK in the early years of BsAb availability including data from both larger academic hospitals and smaller community hospitals. As with all retrospective series, the main weaknesses are that it was hard to capture complete and detailed safety data, we were unable to conduct a central review of histology or collect data on CD20 status which has previously been identified as an important risk factor for response, and responses could not be verified by central imaging review.

In conclusion, our data further confirm that epcoritamab and glofitamab monotherapies are effective treatments in relapsed LBCL, with long durability of remissions in those who attain CR even in this high-risk population. Unlike other published data on efficacy, in this study efficacy was impacted by prior treatment with bendamustine which may have implications for treatment sequencing decisions. Safety data closely mirrored trial data. Outcomes were very poor for those unable to complete cycle 2 of treatment. It is important to improve identification of those at high-risk of failure so that these patients can be offered other management options which may include clinical trials or no further treatment.

CD3xCD20 BsAb are being explored in numerous trials in earlier lines of therapy and in novel combinations with data demonstrating that higher response rates can be achieved. It is hoped that such strategies will lead to better options for patients with RR LBCL in the near future.[30-32]

Disclosures

WO reports advisory board services for and honoraria from Roche, Takeda, Kite Gilead, MSD, Novartis, Beigene, AZ, Kyowa Kirin, Autolus, AbbVie, Incyte, BMS, Sobi, and Johnson and Johnson. EH has received honoraria from Novartis. TM has been a member of advisory boards for and received honoraria from Kite/Gilead, Novartis, Pfizer, Amgen, Takeda, Autolus, BMS, and Janssen. DE-S has received honoraria from AbbVie, Adaptive, AstraZeneca, Beigene/BeOne, Gilead, Janssen, Nurix, Roche, and Takeda; has received travel support from AbbVie, Novartis, and Roche; and has participated in advisory boards for AbbVie, ASTEX, AstraZeneca, Beigene/BeOne, Janssen, Kyowa Kirin, Lilly, Roche, and Sobi. LYC has received travel support from AbbVie. AJD has received honoraria from Kite/Gilead, Roche, AbbVie, BMS, JW Therapeutics, and Sobi; has received research funding from Kite/Gilead, BMS, Roche, AstraZeneca, MSD, and Cellcentric; has been a member of advisory boards for Roche, Incyte, BMS, Kite/Gilead, AbbVie, Sobi, and Serb; and has received conference/travel support from Roche and BMS. AS has received travel support from Takeda and BeiGene/BeOne. SLa has participated in advisory boards for Pfizer, AbbVie, BeiGene/BeOne, and BMS/Celgene. MWi has provided consultancy for AbbVie, AstraZeneca, Sobi, Veriton Pharma; has received honoraria from Roche, AbbVie, Kite/Gilead, Incyte, AstraZeneca, and Takeda; and has received travel support from Kite/Gilead, Takeda, AstraZeneca, and Janssen. PM has been a consultant for Roche and AbbVie. DT has received honoraria from Roche, AbbVie, and BeiGene/BeOne and has participated in advisory boards for Roche, Gilead, and Regeneron. CPF has provided consultancy services for AbbVie, Arvinas, BMS, GenMab, Gilead/Kite, Incyte, Morphosys, Ono, Roche, SERB, and Sobi; has received honoraria from AbbVie, Kite/Gilead, Incyte, Roche, and SERB; and has received research funding from BeiGene/BeOne, Incyte, and AbbVie/Genmab. MG has received honoraria from Incyte and Sobi and a travel grant from AbbVie. JS has received honoraria from AstraZeneca, AbbVie, Roche, and Gilead and travel grants from Gilead and Roche. AK has participated in advisory boards for and received honoraria from Kite/Gilead, Novartis, AbbVie, Roche, and BMS. GC has held employment/leadership positions in GenesisCare; has provided consultancy services for Roche, Takeda, AstraZeneca, Sobi, Beigene, Johnson and Johnson, BMS, Secura Bio, and Sobi; and has received travel grants from Roche and Takeda. NM has provided consultancy services for AbbVie, Roche, and AstraZeneca; and received travel grants from Janssen and Takeda. RMc has participated in advisory boards for and received honoraria from AstraZeneca, Johnson and Johnson, SOBI, and Gilead, and served as a consultant for BeOne. KZDM has received honoraria from AstraZeneca and travel grants from Gilead and Novartis. JN has received honoraria from Kite/Gilead. GP has provided consultancy services for AbbVie and Janssen and has received honoraria from AbbVie, Janssen, AstraZeneca, and BeiGene. DC has participated in

advisory boards for AbbVie, BMS, Jazz Pharma, and Roche; and received a travel grant from Servier. MA has received honoraria from Sobi and Takeda. PK has received honoraria from Roche and AbbVie and travel grants from Kite/Gilead. WT has provided consultancy services for and received honoraria from Roche, AbbVie, Genmab, Takeda, and Sobi, and has received a travel grant from Roche. All other listed authors have no conflicts of interest to disclose.

Contributions

WT, WO, WW, and EH designed and conducted the study, analyzed and interpreted the data, reviewed and approved the manuscript, and agreed to be accountable for its contents. All authors collected data.

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Data-sharing statement

The data that support the findings of this article are available from the corresponding author upon reasonable request. A data-sharing agreement will be required, and data will be provided in de-identified form to protect patients' confidentiality.

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