Article

Not peer-reviewed version

Global Prevalence of Post-Transplantation Lymphoproliferative Disease (PTLD) in Hematopoietic Stem Cell Transplantation (HSCT): A Systematic Review and Meta-Analysis

Shahad Saif Khandker , Alif Hasan Pranto , Afrin Rahman Juthy , Mariam Zaman , Argha Sarkar , Druphadi Sen , Dewan Zubaer Islam , Ehsan Suez , Md Asiful Islam * , Rahima Begum * , A. N. M. Mamun-Or-Rashid *

Posted Date: 5 March 2026

doi: 10.20944/preprints202603.0439.v1

Keywords: PTLD; HSCT; transplant; malignant; frequency; incident

Article

Global Prevalence of Post-Transplantation Lymphoproliferative Disease (PTLD) in Hematopoietic Stem Cell Transplantation (HSCT): A Systematic Review and Meta-Analysis

Shahad Saif Khandker [1], Alif Hasan Pranto [2], Afrin Rahman Juthy [3], Mariam Zaman [2], Argha Sarkar [2], Druphadi Sen [4], Dewan Zubaer Islam [5], Ehsan Suez [6], Md Asiful Islam [7,*], Rahima Begum [8] and A. N. M. Mamun-Or-Rashid [9]

[1] Department of Biochemistry and Molecular Biology, Gono Bishwabidyalay, Dhaka 1344, Bangladesh
[2] School of Pharmacy, BRAC University, Dhaka 1212, Bangladesh
[3] Faculty of Engineering and Science, University of Greenwich, Central Ave, Gillingham, Chatham ME4 4TB, UK
[4] Department of Cell and Systems Biology, University of Toronto, 27 King's College Cir, Toronto, ON M5S 1A1, Canada
[5] Department of Microbiology, Jahangirnagar University, Dhaka 1342, Bangladesh
[6] Institute of Bioinformatics, University of Georgia, Athens, GA 30602, USA
[7] Department of Biomedical Science and Physiology, School of Pharmacy and Life Sciences, Faculty of Science and Engineering, University of Wolverhampton, Wolverhampton, WV1 1LY, UK
[8] Department of Microbiology, Gono Bishwabidalaly, Bangladesh
[9] Department of Environmental and Occupational Health, University of Pittsburgh, Pittsburgh, PA 15261, USA
* Correspondence: asiful.islam@wlv.ac.uk

Abstract

Background: Hematopoietic stem cell transplantation (HSCT) is a widely utilized subtype of transplantation employed in various malignant and non-malignant diseases, particularly when conventional treatments or therapeutics prove ineffective. Despite the frequent occurrence of post-transplantation lymphoproliferative disease (PTLD) in patients undergoing HSCT, no comprehensive global prevalence rate has been established to date. Methodology: In this study, we selected 39 studies from 941 studies from three databases (i.e., PubMed, ScienceDirect, and Google Scholar) to identify the global prevalence rate of PTLD in HSCT patients. Results: The pooled prevalence was determined as 5.6% (95% CI: 5.0 to 6.3) and increased to 12.4% (95% CI: 10.2 to 14.7) after excluding outlier studies. The quality of the studies was also high. The prevalence of death cases among HSCT patients was determined as 0.6% (95% CI: 0.4 to 0.9). PTLD was most prevalent in allogenic HSCT (i.e., 5.6% (95% CI: 4.9 to 6.3)) and within the European region (i.e., 27.1% (95% CI: 21.4 to 32.8)). Among risk factors, HLA mismatch was reported in most of the studies. Conclusions: This study assessed and discussed the overall global prevalence of PTLD in HSCT patients, continent-based prevalence, and risk factors that can be helpful in finding the possible prevention mechanism of PTLD and implementing individualized treatment approaches based on the treatment availability during HSCT.

Keywords: PTLD; HSCT; transplant; malignant; frequency; incident

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 5 March 2026 doi:10.20944/preprints202603.0439.v1

1. Introduction

Transplantation is a life-saving medical procedure that involves the transplantation of cells, tissues, or organs to replace or repair damaged ones. This procedure significantly improves the quality of life in cases of organ failure [1]. Hematopoietic stem cell transplantation (HSCT), a subset of transplantation therapies, exemplifies this potential, offering hope for patients with malignant and non-malignant diseases [2]. Stem cells, particularly hematopoietic stem cells (HSCs), play a crucial role in reconstructing the immune system, which also helps to foster immune tolerance and improve post-transplant outcomes [3]. While advancements in transplant techniques have significantly increased survival rates, they also come with risks. Among the most serious complications is post-transplant lymphoproliferative disorder (PTLD) [4].

PTLD refers to a heterogeneous group of lymphoid proliferative conditions ranging from benign, self-limiting infectious mononucleosis to aggressive lymphomas [5]. Usually, after transplantation, patients who experience fever, adenopathy, weight loss, mass lesions, indescribable pain, or dysfunction of the transplanted organ can be recognized as a suspect for PTLD [6]. Although PTLD in the solid-organ transplant population initiates after 6 months and in HST recipients, 70–90 days, some reports claim that the onset of PTLD can occur even within 1 week or even after 9 years after the transplantation. Symptoms are diverse and may be related to organ dysfunction, mass effect, viremia, or lymphoma-related B symptoms. The reasons for these differences in incidence were not clearly reported; however, depending on the amount of lymphoid tissue present in each organ, immunological reaction, virus, and the immunosuppression treatment, it plausibly may vary [6,7].

Despite studies identifying PTLD in various solid organ transplant recipients, the association and prevalence of PTLD in HSCT patients remain unclear. Therefore, in this systematic review and meta-analysis, we determined the incidence of PTLD in HSCT patients along with subgroup analyses of the prevalence of PTLD among different types of HSC transplantation (i.e. allogeneic, autologous, and syngeneic), the prevalence of death cases in HSCT, continent-based analyses of PTLD prevalence in HSCT, and the possible risk factors.

2. Methodology

2.1. Study Design and Selection of Inclusion-Exclusion Criteria

This study followed the PRISMA study design (registration link: https://osf.io/mp72n), with minor adjustments, and employed a systematic approach to identify relevant studies and conduct a meta-analysis to assess the global prevalence of PTLD among HSCT patients, adhering to established PRISMA guidelines obtained from previous studies [8–10]. The study included peer-reviewed articles focusing on the prevalence, incidence, occurrence, or frequency of PTLD in HSCT patients. Excluded materials encompassed narrative or systematic reviews, meta-analyses, book chapters, case reports, editorials, correspondence, conference papers, press releases, and non-original journal articles. Only English-language publications were considered. The study did not involve patients, the public, healthcare institutions, or any third parties in data collection, processing, study design, assessment, analysis, or interpretation of results.

2.2. Search Strategy and Study Inclusion

A comprehensive search strategy was developed to screen relevant studies from three online databases: PubMed, ScienceDirect, and Google Scholar. The search utilized multiple terms such as "prevalence," "post-transplant lymphoproliferative disorder," and "hematopoietic stem," along with their respective related terms or short forms like "incidence", "epidemiology", "frequency"; "PTLD", "bone marrow", and "transplant". Boolean operators (AND/OR) were used to refine the search. Specific filters, including "Title/Abstract" for PubMed, "Title, abstract or author-specified keywords" for ScienceDirect, and "allintitle" for Google Scholar, were applied in the advanced search options,

 doi:10.20944/preprints202603.0439.v1

following previous studies with slight modifications [11,12]. No publication date filters were used. Duplicate search results were carefully managed and excluded from the study.

2.3. Data Extraction, Meta-Analysis and Heterogeneity

The authors independently conducted the initial search and necessary data extraction from the included studies, subsequently validating their findings through discussion. Primarily, the data of PTLD cases and the number of HSCT patients were obtained from each of the individual studies. The extracted information encompassed basic study characteristics such as study ID, study type, HSCT participants number, male and female numbers, age, HSCT type, PTLD type, and study region. Any disagreements regarding data inclusion or exclusion were carefully resolved by the authors, and they addressed missing or unclear information by contacting the corresponding or first author of the study.

The authors employed a meta-analysis approach to ascertain the pooled prevalence of PTLD in HSCT patients, utilizing the collected data. A random-effects model with a 95% confidence interval (CI) was employed to analyze the pooled prevalence. The I2 statistics method was utilized to estimate the pooled heterogeneity of the included studies. The I2 values indicated the level of heterogeneity among the studies, with 25-50% suggesting low heterogeneity, 51-75% indicating moderate heterogeneity, and values exceeding 75% signifying high heterogeneity. Furthermore, outlier studies and sensitivity analysis were implemented, based on previously established methodologies to explore potential outliers and their influence on the overall effect size [13,14]. The outlier determination and sensitivity analyses were conducted sequentially through the primary exclusion of outlier studies, identified using funnel and Galbraith plots, and subsequently the reanalysis of the primary meta-plot employing a random-effects model. The meta-analysis, forest plot, funnel plot, and Galbraith plot construction were executed using RStudio software (version 4.3.0) and the "metafor" package (version 4.2-0) of R.

2.4. Evaluation of Study Quality and Analysis of Bias Risk

The researchers evaluated the quality of the included studies using a set of nine questions derived from the Study Quality Assessment Tools, NIH, and Systematic Reviews: Step 6: Assess Quality of Included Studies, UNC [15,16]. In this study, the selected questions could be answered with "Yes", "No", "Unclear", "Not Reported" (NR), or "Not Applicable" (NA), which were then converted to numerical scores (e.g., 1 for Yes, 0 for No and Unclear, no score for NR and NA). For each study, these scores were totaled and divided by the number of questions (9 in this case), then converted to a percentage. The resulting percentage indicated the study's quality and potential publication bias risk: <50% suggested low quality and high bias risk, 50-70% indicated moderate quality, and >80% signified high quality and low bias risk. The scoring method was adapted from previous research with minor modifications [17,18].

2.5. Subgroup Analyses and Risk Factors

To evaluate the PTLD prevalence among HSCT patients based on the type of PTLD, death cases, and continent separate subgroup analyses were conducted. Plausible risk factors of the PTLD were also assessed.

3. Results

3.1. Search Results and Study Inclusion

Initially, a total of 941 search results were identified from three electronic databases: PubMed (n=224), ScienceDirect (n=594), and Google Scholar (n=123). These results were obtained through our search strategies, applied keywords, and various filters utilized during the searches. During the study screening phase, 608 articles were directly excluded. These articles included reviews, systematic

 doi:10.20944/preprints202603.0439.v1

reviews, mini-reviews, meta-analyses, editorials, case reports, and correspondence, excluding only the full-text original research articles. From the remaining 333 studies, 15 were excluded due to study duplication.

Out of the 50 articles selected for evaluation, after rigorous screening, validation, and excluding studies (n=279) that were deemed irrelevant to the study's aims and objectives, 39 studies were selected for inclusion in the systematic review and meta-analysis. These studies met the eligibility and inclusion criteria (Figure 1).

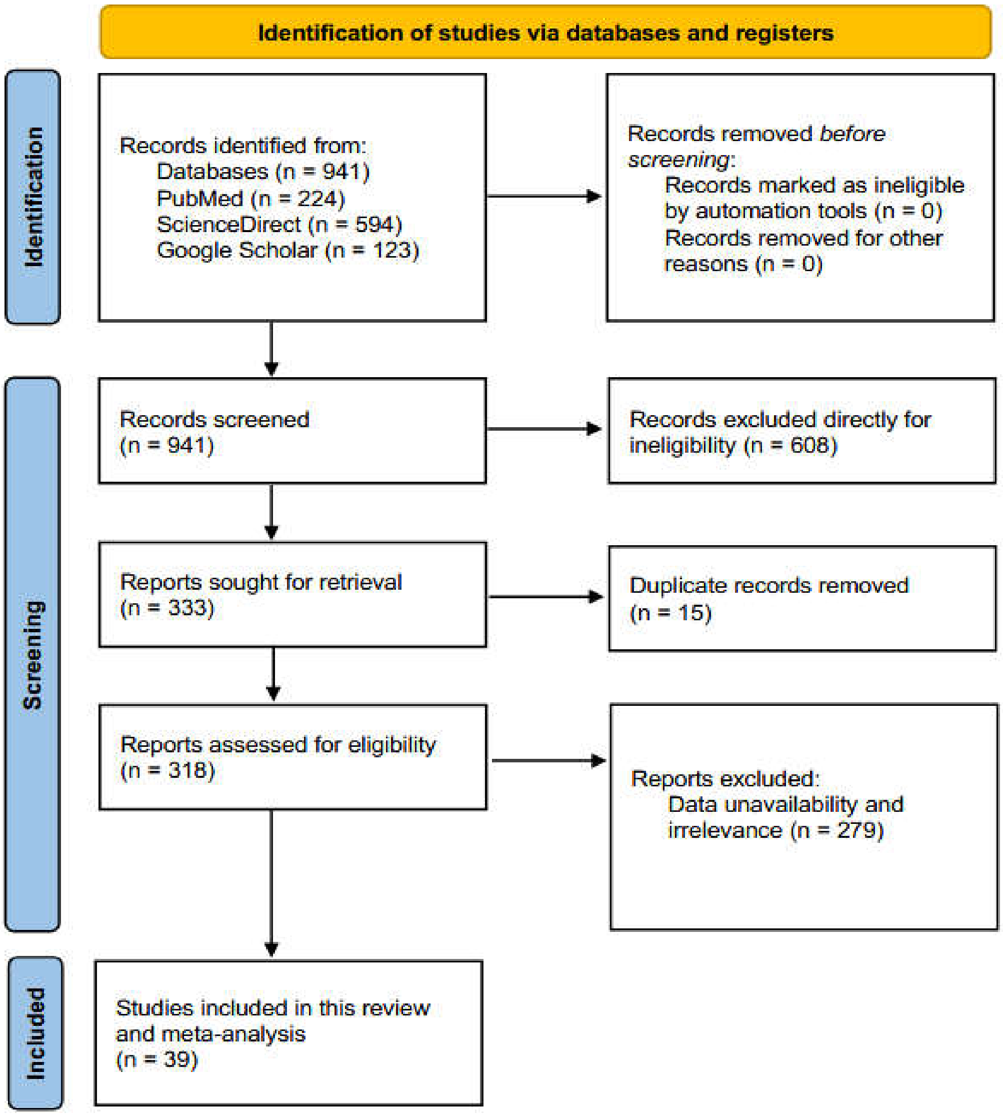


Figure 1. A simplified PRISMA flow diagram of methodology.

3.2. Major Characteristics of the Included Studies

The authors carefully extracted and validated major characteristic data of the included articles (n=39) that completely met the criteria of the current systematic review and meta-analysis [19–57]. The 39 included studies were conducted in different countries of different continents including Europe (i.e. Portugal, Italy, Germany, Switzerland, Sweden, Spain, France, Poland, and Denmark), Asia (i.e. India, Japan, China, Korea, and Turkey), and North America (i.e. Canada and USA). Among HSCT types allogenic, autologous, and syngeneic were reported. Among the types of PTLD, such as Monomorphic PTLD (M-PTLD), Polymorphic-PTLD (P-PTLD), Plasmacytic hyperplasia PTLD (PH-

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 5 March 2026 doi:10.20944/preprints202603.0439.v1

PTLD), Classic Hodgkin lymphoma PTLD (CHL-PTLD), Epstein–Barr virus PTLD (EBV-PTLD), and Early lesion PTLD (EL-PTLD). Besides, the type of the studies was cross-sectional or cohort. The number of total participants along with male, and female numbers and their age details were reported in detail in Table 1.

 doi:10.20944/preprints202603.0439.v1

Table 1. General characteristics of the included studies.

Study ID	Study Type	HSCT Participants	Male	Female	Patient Age (Y) (median range)	HSCT Types	PTLD Types	Region	Reference
Dias 2018	Cross-sectional study	15	7	8	NR	Allogeneic	M-PTLD, P-PTLD	Portugal	[19]
Fu 2017	Cross-sectional study	13	9	4	NR (18–46)	Allogeneic	M-PTLD	India	[20]
Fujimoto 2019	Cohort study	64539	NR	NR	NR	Autologous, Allogeneic, Syngeneic	NR	Japan	[21]
Chiereghin 2016	Cohort study	28	20	8	NR	Allogeneic	EBV-PTLD	Italy	[22]
Luo 2014	Cross-sectional study	343	260	83	NR	Allogeneic	NR	China	[23]
Kalra 2018	Cross-sectional study	306	NR	NR	NR	Allogeneic	NR	Canada	[24]
Overkamp 2020	Cohort study	26	NR	NR	NR	Allogeneic	M-PTLD, P-PTLD, CHL-PTLD,	Germany	[25]
Yoon 2014	Cohort study	2684	NR	NR	26(NR)	Allogeneic	M-PTLD, P-PTLD, PH-PTLD	South Korea	[26]

 doi:10.20944/preprints202603.0439.v1

Lückemeier 2023	Cohort study	15	11	4	NR (32-67)	Allogeneic	M-PTLD, P-PTLD, CHL-PTLD	Switzerland	[27]
Kinzel 2022	Cohort study	1192	674	518	45 (NR)	Allogeneic	M-PTLD, P-PTLD,	Canada	[28]
Sundin 2006	Cohort Study	553	NR	NR	NR	Allogeneic	EBV-PTLD	Sweden	[29]
Fu 2016	Cross sectional Study	30	19	11	23 (14-52)	Allogeneic	M-PTLD, P-PTLD	China	[30]
Park 2018	Cross sectional Study	22	11	11	9 (1.6–16.9)	Allogeneic	EBV-PTLD	Korea	[31]
Rosello 2021	Cohort Study	1009	597	412	41 (14-70)	Allogeneic	M-PTLD, P-PTLD	Spain	[32]
Choi 2010	Cross sectional Study	5817	11	3	42.6 (24-60)	Allogeneic	M-PTLD, P-PTLD	Korea	[33]
Liu 2018	Cross sectional Study	244	NR	NR	NR	Allogeneic	NR	China	[34]
Pagliuca 2019	Cross sectional Study	1024	123	84	42.5 (8.3-74.7)	Allogeneic	EBV-PTLD, M-PTLD, P-PTLD	France	[35]
Watson 2020	Cohort Study	92	52	0	47.1 (17-75)	Autologous, Allogeneic	NR	USA	[36]
Chen 2012	Cross sectional Study	15	12	3	31 (9-60)	Allogeneic	M-PTLD, P-PTLD, PH-PTLD, EL-PTLD	China	[37]
Gunduz 2017	Cohort Study	979	59	41	33 (5-71)	Allogeneic	CHL-PTLD	Turkey	[38]

doi:10.20944/preprints202603.0439.v1

Fujimoto 2020	Cohort Study	5270	3193	2077	47 (16-88)	Allogeneic	NR	Japan	[39]
Czyzewski 2012	Cross sectional Study	8	NR	NR	16 (5 – 19)	Allogeneic	EBV- PTLD, M-PTLD, P-PTLD	Poland	[40]
Ali 2018	Cross sectional Study	408	NR	NR	5.9 (2.3-17.3)	Allogeneic	EBV- PTLD, M-PTLD, P-PTLD	Canada	[41]
Wareham 2018	Cohort Study	2642	1581	1061	42.9 (17–35)	Allogeneic	EBV-PTLD	Denmark	[42]
Courville 2016	Cross sectional Study	32	19	13	44 (3–72)	Allogeneic	M-PTLD, P-PTLD	USA	[43]
Hahn 2023	Cohort Study	47518	29528	17990	5.32 (10-60)	Allogeneic	NR	Korea	[44]
Styczynski 2013	Cross sectional Study	4466	adult 90	adult 90	30 (10–68)	Allogeneic	EBV- PTLD	Poland	[45]
Xu 2015	Cohort Study	1184	NR	NR	27 (3-49)	Allogeneic	NR	China	[46]
Gilman 2017	Cross sectional Study	10	NR	NR	47 (19–71)	Allogeneic	EBV- PTLD, "M-PTLD, P-PTLD, PH-PTLD	Netherlands	[47]
Gonzalez-Farre 2014	Cross sectional Study	35	26	9	54 (26–77)	Allogeneic	P-PTLD, PH-PTLD, CHL-PTLD	Spain	[48]
Kroll 2011	Cross sectional Study	181	5	2	55 (37–73)	Autologous	EBV-PTLD	USA	[49]
Zhou 2022	Cohort Study	300	184	116	28 (NR)	Allogeneic	EBV-PTD	China	[50]

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 5 March 2026 doi:10.20944/preprints202603.0439.v1

D'Aveni 2014	Cohort Study	40	20	20	30 (NR)	Allogeneic	EBV-PTD	France	[51]
A Nash 2003	Cross sectional Study	56	21	35	42 (NR)	Autologous	EBV-PTD	USA	[52]
Comoli 2007	Cross sectional Study	27	15	12	8 (NR)	Allogeneic	EBV-PTD	Italy	[53]
Blaes 2009	Cohort Study	30	NR	NR	55 (NR)	Allogeneic	EBV-PTD	USA	[54]
Comoli 2007	Cross sectional Study	46	NR	NR	NR	Allogeneic	EBV-PTD	Italy	[55]
Park 2006	Cohort Study	1,116	NR	NR	NR (17-45)	Allogeneic	EBV-PTD	Korea	[56]
Althubaiti 2019	Cohort Study	266	NR	NR	NR	Allogeneic	EBV-PTD	Canada	[57]

Here, NR= Not reported, Y= years, PTLD= post-transplant lymphoproliferative disorder, HSCT= hematopoietic stem cell transplantation, M-PTLD= Monomorphic PTLD, P-PTLD= Polymorphic PTLD, PH-PTLD= Plasmacytic hyperplasia PTLD, CHL-PTLD= Classic Hodgkin lymphoma PTLD, EBV= Epstein–Barr virus, EBV-PTLD=EBV-related PTLD, EL-PTLD= Early lesion PTLD.

3.3. Primary Meta-Analysis, Heterogeneity, outliers and Sensitivity

The primary objective of our meta-analysis was to determine the pooled prevalence of PTLD in HSCT patients globally. As a result, 1822 PTLD cases were reported out of 142581 HSCT patients. Based on data extracted from the included studies, an overall pooled prevalence of 5.6% (95% CI: 5.0 to 6.3) with a substantial heterogeneity of 99% (p=0) was found. The highest prevalence was found in Overkamp 2020 (100%, (95% CI: 86.8 to 100.0)) and Czyzewski 2012 (100%, (95% CI: 63.1 to 100.0)) whereas the lowest prevalence was found in Choi 2010 (0.2% (95%CI: 0.1 to 0.4)) (Figure 2).

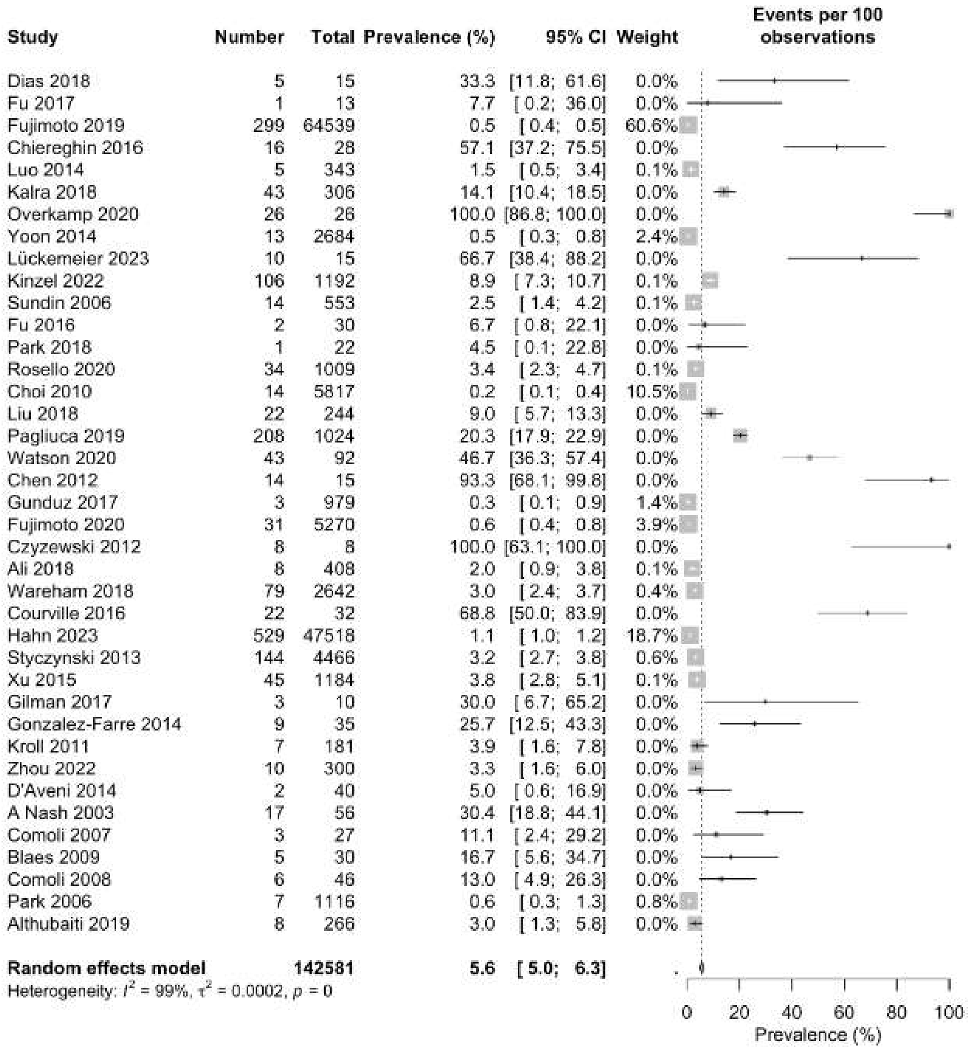


Figure 2. Forest plot of the pooled prevalence of PTLD in patients after hematopoietic stem cell transplantation.

A funnel plot constructed for visualization of the possible sources of publication bias indicated multiple potential outlier studies with high risks of bias (Figure 3). The Galbraith plot which was constructed for visual investigation and confirmation of the potential sources of bias indicated 11 outlier studies (i.e. Chen 2012, Courville 2016, Luckemeier 2023, Chiereghin 2016, Watson 2020, Park 2006, Fujimoto 2020, Yoon 2014, Fujimoto 2019, Gunduz 2017, and Choi 2010) as shown in Figure 6 (Figure 4).

After excluding the outliers, the forest plot was reconstructed using a random effect model to identify the sensitivity. As a result, the prevalence was enhanced to 12.4% (95%CI: 10.2 to 14.7) confirming the primary outcome to be accurate (Figure 5).

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 5 March 2026 doi:10.20944/preprints202603.0439.v1

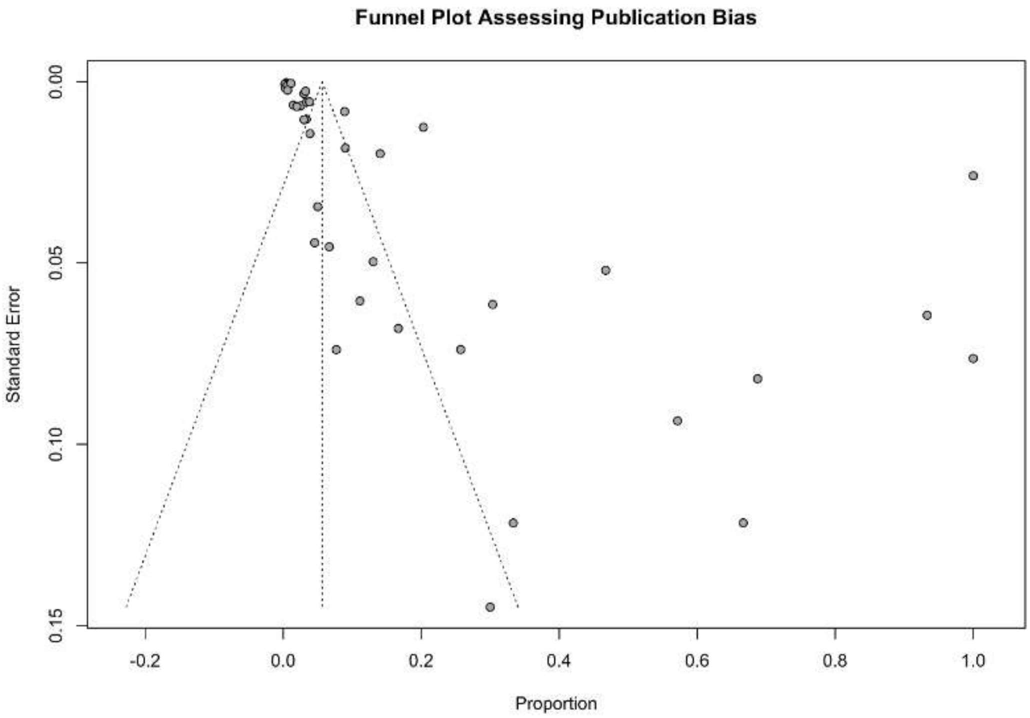


Figure 3. Funnel plot assessing the plausible publication biases of included studies for the prevalence of PTLD in patients after hematopoietic stem cell transplantation.

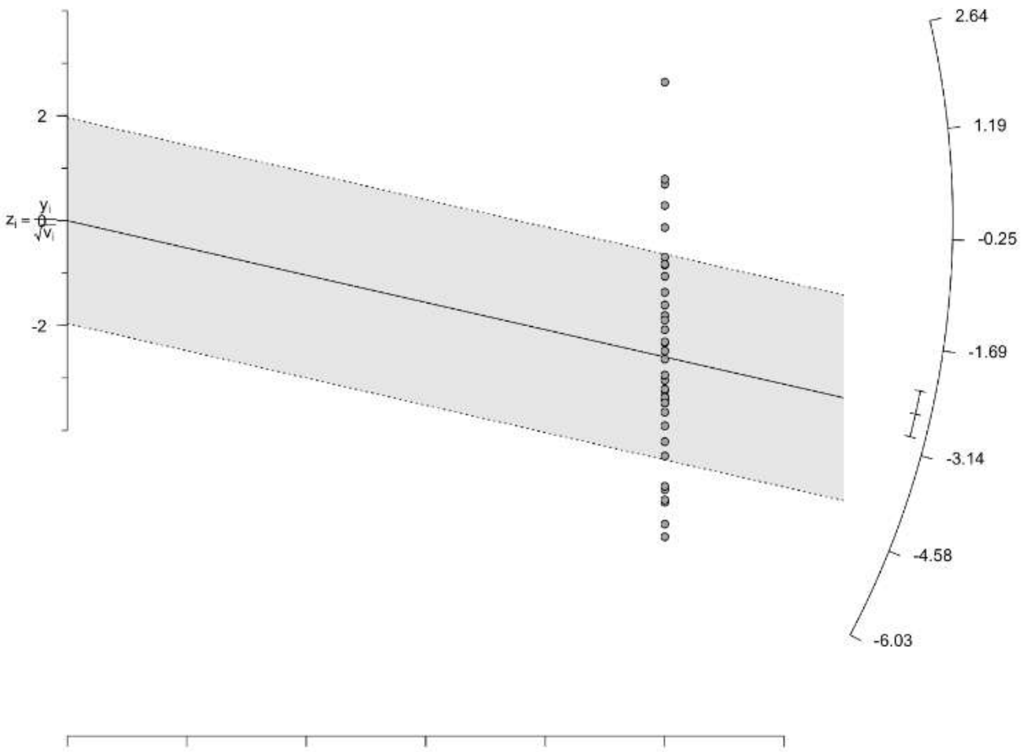


Figure 4. Galbraith's plot assessing the outlier studies for the prevalence of PTLD in patients after hematopoietic stem cell transplantation.

Study	Number	Total	Prevalence (%)	95% CI	Weight
Dias 2018	5	15	33.3	[11.8; 61.6]	0.0%
Fu 2017	1	13	7.7	[0.2; 36.0]	0.0%
Luo 2014	5	343	1.5	[0.5; 3.4]	0.5%
Kalra 2018	43	306	14.1	[10.4; 18.5]	0.1%
Overkamp 2020	26	26	100.0	[86.8; 100.0]	0.0%
Kinzel 2022	106	1192	8.9	[7.3; 10.7]	0.3%
Sundin 2006	14	553	2.5	[1.4; 4.2]	0.5%
Fu 2016	2	30	6.7	[0.8; 22.1]	0.0%
Park 2018	1	22	4.5	[0.1; 22.8]	0.0%
Rosello 2020	34	1009	3.4	[2.3; 4.7]	0.7%
Liu 2018	22	244	9.0	[5.7; 13.3]	0.1%
Pagliuca 2019	208	1024	20.3	[17.9; 22.9]	0.1%
Czyzewski 2012	8	8	100.0	[63.1; 100.0]	0.0%
Ali 2018	8	408	2.0	[0.9; 3.8]	0.4%
Wareham 2018	79	2642	3.0	[2.4; 3.7]	1.9%
Hahn 2023	529	47518	1.1	[1.0; 1.2]	91.1%
Styczynski 2013	144	4466	3.2	[2.7; 3.8]	3.0%
Xu 2015	45	1184	3.8	[2.8; 5.1]	0.7%
Gilman 2017	3	10	30.0	[6.7; 65.2]	0.0%
Gonzalez-Farre 2014	9	35	25.7	[12.5; 43.3]	0.0%
Kroll 2011	7	181	3.9	[1.6; 7.8]	0.1%
Zhou 2022	10	300	3.3	[1.6; 6.0]	0.2%
D'Aveni 2014	2	40	5.0	[0.6; 16.9]	0.0%
A Nash 2003	17	56	30.4	[18.8; 44.1]	0.0%
Comoli 2007	3	27	11.1	[2.4; 29.2]	0.0%
Blaes 2009	5	30	16.7	[5.6; 34.7]	0.0%
Comoli 2008	6	46	13.0	[4.9; 26.3]	0.0%
Althubaiti 2019	8	266	3.0	[1.3; 5.8]	0.2%
Random effects model		**61994**	**12.4**	**[10.2; 14.7]**	

Heterogeneity: $I^2 = 99\%$, $\tau^2 = 0.0027$, $p = 0$

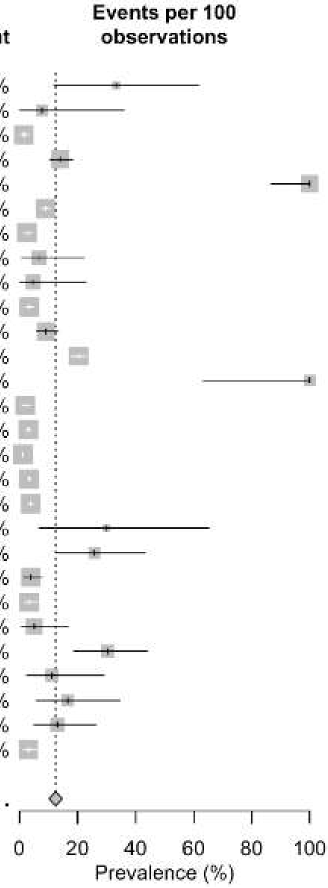
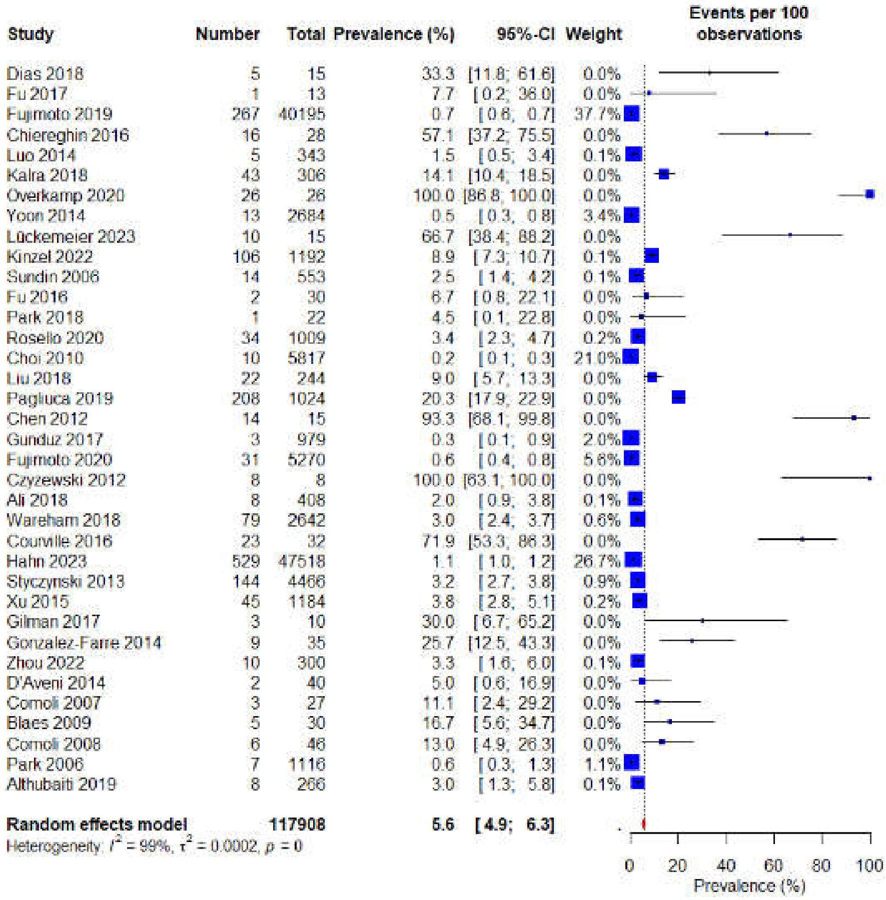
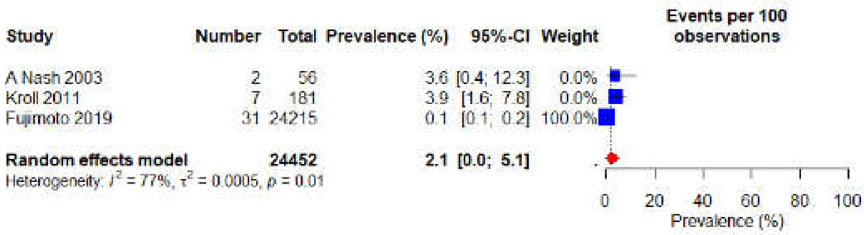


Figure 5. Forest plot excluding the outlier studies of the prevalence of PTLD in patients after hematopoietic stem cell transplantation.

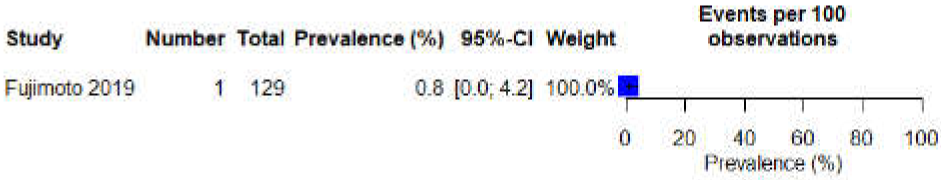
doi:10.20944/preprints202603.0439.v1



(a)



(b)



(c)

Figure 6. Forest plot of the prevalence of PTLD in patients after (a) allogeneic (b) autologous and (c) syngeneic hematopoietic stem cell transplantation.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 5 March 2026 doi:10.20944/preprints202603.0439.v1

3.4. Assessment of Study Quality and Risk of Bias

Based on the answers to our specified set of quality assessment questions and subsequent scoring strategy, 33 studies scored more than 80% indicating high-quality studies and 6 studies scored more than 50% resembling moderate-quality studies. Interestingly, 17 studies obtained a 100% score. No study was determined to be scored less than 50%, hence there were no low-quality studies with a high-risk of bias (Table 2).

Table 2. Quality investigation of the included studies.

SL	Study ID	1	2	3	4	5	6	7	8	9	Overall Score (%)
1	Dias 2018	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
2	Fu 2017	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
3	Fujimoto 2019	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
4	Chiereghin 2016	Y	Y	Y	Y	Y	Y	N	N	Y	77.78%
5	Luo 2014	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
6	Kalra 2018	Y	Y	Y	N	Y	Y	Y	Y	N	77.78%
7	Overkamp 2020	Y	Y	Y	N	Y	Y	Y	Y	Y	88.89%
8	Yoon 2014	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
9	Lückemeier 2023	Y	Y	Y	N	Y	Y	Y	Y	N	77.78%
10	Kinzel 2022	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
11	Sundin 2006	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
12	Fu 2016	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
13	Park 2018	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
14	Rosello 2021	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
15	Choi 2010	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
16	Liu 2018	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
17	Pagliuca 2019	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
18	Watson 2020	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
19	Chen 2012	Y	Y	Y	N	Y	Y	N	N	Y	66.67%
20	Gunduz 2017	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
21	Fujimoto 2020	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
22	Czyzewski 2012	Y	Y	Y	N	Y	Y	N	N	N	55.55%
23	Ali 2018	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
24	Wareham 2018	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
25	Courville 2016	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
26	Hahn 2023	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
27	Styczynski 2013	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
28	Xu 2015	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
29	Gilman 2017	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
30	Gonzalez-Farre 2014	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
31	Kroll 2011	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
32	Zhou 2022	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
33	D'Aveni 2014	Y	Y	Y	Y	Y	Y	N	N	N	66.67%
34	A Nash 2003	Y	Y	Y	Y	Y	Y	Y	Y	Y	100%
35	Comoli 2007	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
36	Blaes 2009	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
37	Comoli 2007	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
38	Park 2006	Y	Y	Y	Y	Y	Y	Y	Y	N	88.89%
39	Althubaiti 2019	Y	Y	Y	N	Y	Y	Y	Y	Y	88.89%

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 5 March 2026 doi:10.20944/preprints202603.0439.v1

Here, 1. Was the research question appropriate? 2. Is the target/study population clearly defined? 3. Were any inclusion and/or exclusion criteria mentioned? 4. Was any time frame mentioned? 5. Are non-responders clearly described? 6. does the sample represent the target population? 7. Were data collection methods standardized? 8.Was the timeframe sufficient so that one could reasonably expect to see an association between exposure and outcome if it existed? 9. Did the authors use statistical analyses?, Y=yes, N=no.

3.5. Subgroup Analysis and Risk Factors

Subgroup analysis was aimed to investigate the pooled prevalence of PTLD in HSCT patients based on the type of HSCT, overall death cases, and patients across different continents. For HSCT type, the highest pooled prevalence was found in allogeneic HSCT (5.6% (95% CI: 4.9 to 6.3)) followed by the autologous (2.1% (95%CI: 0.0 to 5.1)) and syngeneic (0.8% (95%CI: 0.0 to 4.2)) HSCT (Figure 6). The prevalence of death cases among the overall HSCT patients was found 0.6% (95%CI: 0.4 to 0.9) (Figure 7). Regarding the continent-based subgroup analysis, Asia was found to have the lowest prevalence rate (1.1% (95%I: 0.7 to 1.5)), followed by North America (10.1% (95%I: 5.8 to 14.4)); whereas, Europe had the highest prevalence rate of PTLD among HSCT patients (27.1% (95%I: 21.4 to 32.8)) (Figure 8). The geographical locations of the countries within different continents are demonstrated in detail in Figure 9.

Plausible reported risk factors were also obtained from the included studies. As a result, we observed that HLA mismatch was the highest reported risk factor of PTLD in HSCT patients, followed by the T-cell depletion, Graft-versus-host disease, and EBV seroprevalence or serostatus. All the plausible risk factors are presented in Figure 10.

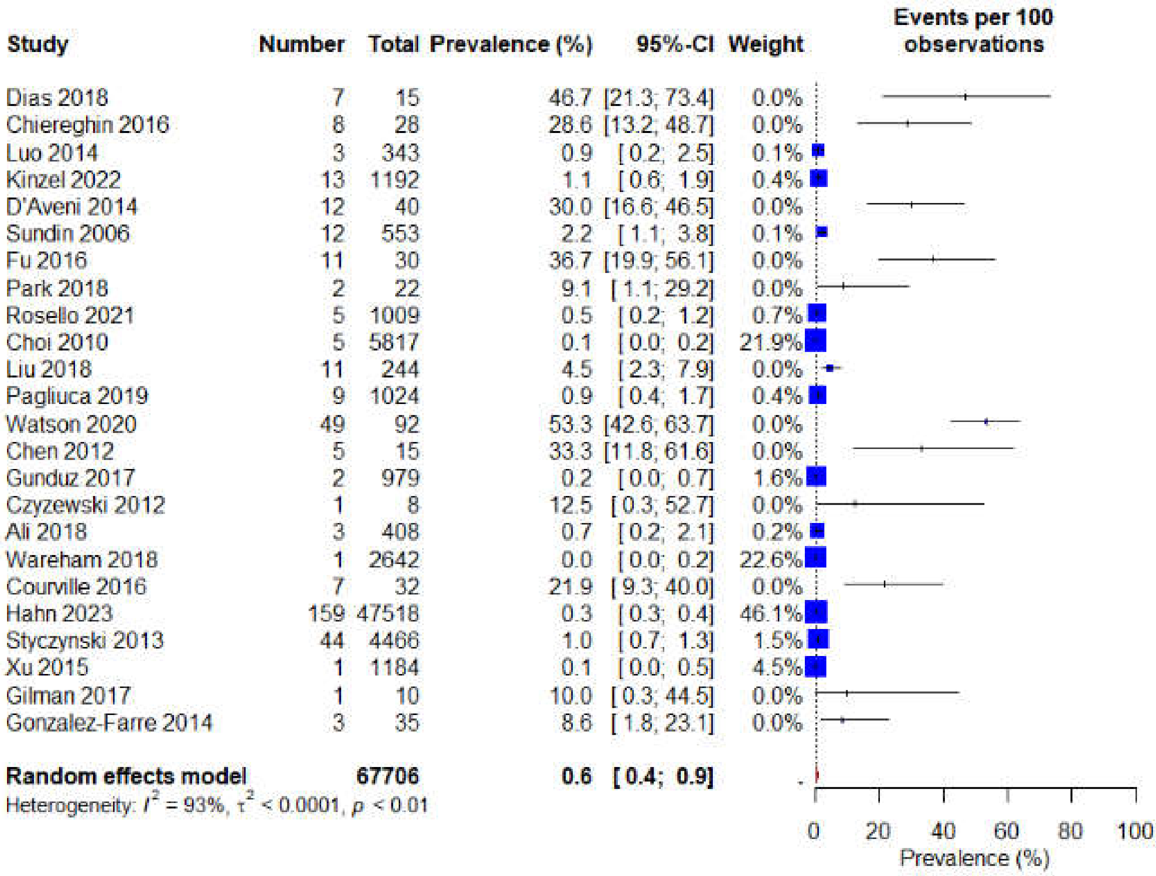


Figure 7. Forest plot of the prevalence of reported death cases in HSCT patients.

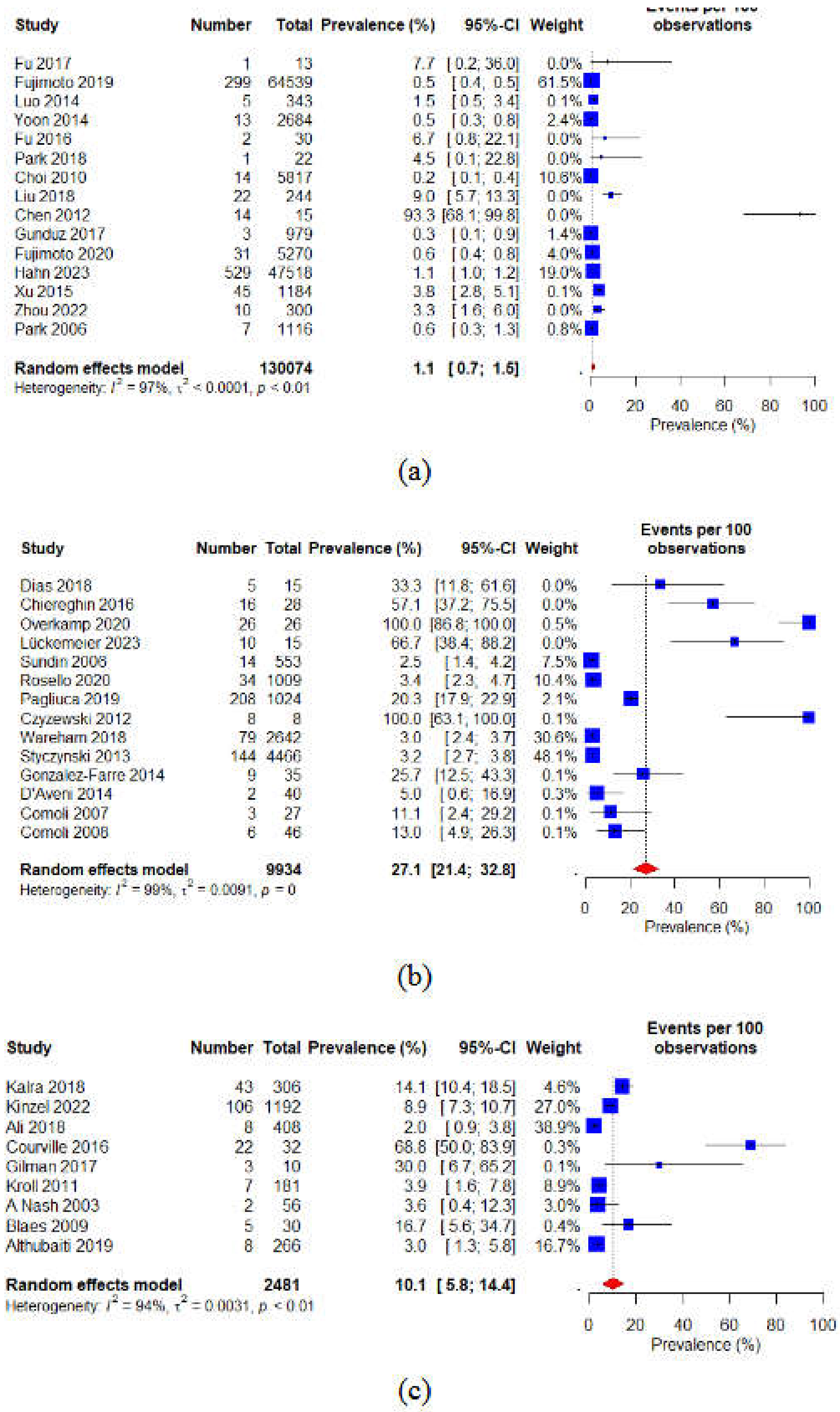


Figure 8. Forest plot of the pooled prevalence of PTLD in patients after HSTC in (a) Asia (b) Europe and (c) North America.

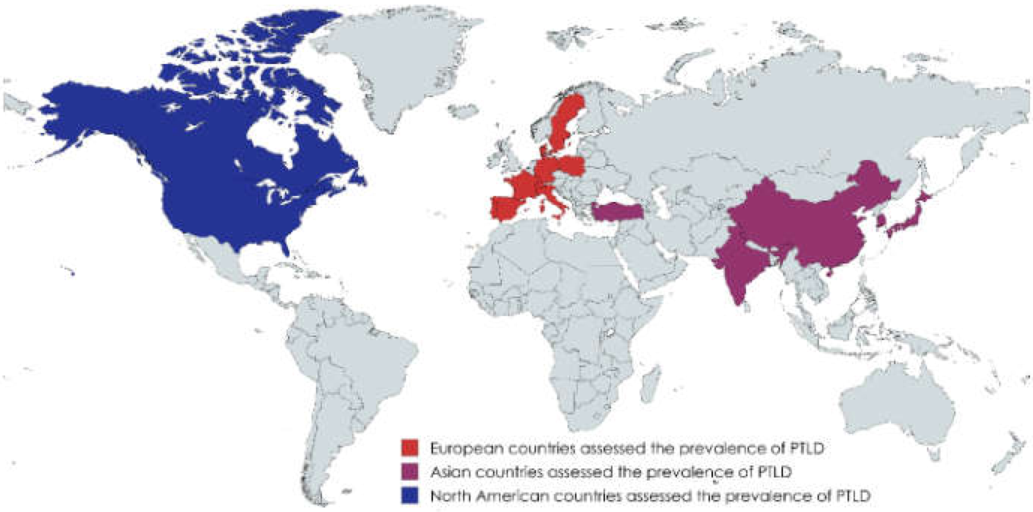


Figure 9. Geographical locations of the studies reported the prevalence of PTLD in HSTC patients.

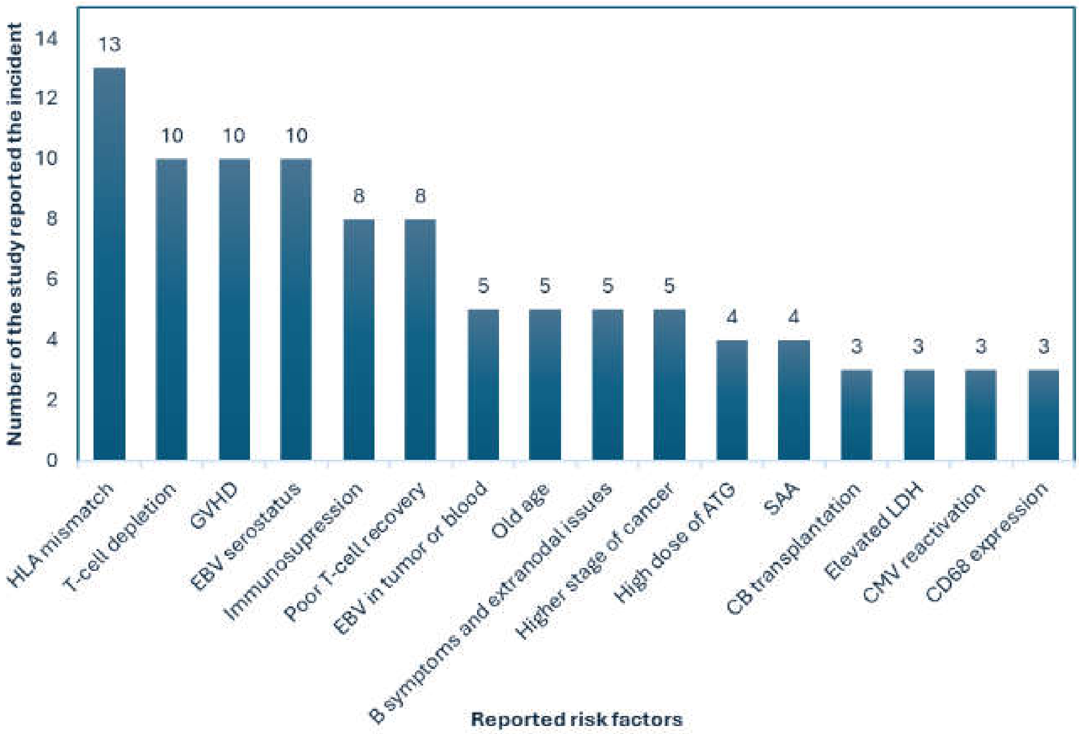


Figure 10. Plausible risk factors of PTLD reported in different included studies. Here, GVHD= Graft-versus-host disease, EBV= Epstein-Barr virus, ATG= Antithymocyte Globulin, SAA= severe aplastic anemia, CB= cord blood, LDH= lactate dehydrogenase, CMV= Cytomegalovirus.

4. Discussion

To the best of our knowledge, this is the individual meta-analysis to investigate the global prevalence of PTLD in patients undergoing HSCT. By analyzing data from diverse regions, we provide new insights into the prevalence of PTLD with HSCT, highlighting significant

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 5 March 2026 doi:10.20944/preprints202603.0439.v1

epidemiological and clinical trends. Our findings determined that the pooled prevalence rate of PTLD among HSCT was 5.6% (95% CI: 5.0 to 6.3), which indicates that it is a critical concern for HSCT patients, and they need to be carefully handled after the transplantation to restrict PTLD as the prevalence rate is quite high (Figure 2). Interestingly, after excluding the plausible outlier studies the prevalence rate of PTLD rose to 12.4% (95%CI: 10.2 to 14.7) which further proves it is to be even more serious issue for HSCT patients (Figure 5). According to previous reports, the incidence of PTLD varies depending on the type of transplantation such as 19% for intestinal transplants, 2-10% for heart transplants, 5%–9% for heart-lung transplants, and 2%–8% for liver transplants [7]. Therefore, other than PTLD in intestinal transplant patients, every other reported transplantation was investigated to have a lower prevalence of PTLD.

In subgroup analyses, we initially investigated that allogenic HSCT was the most prevalent transplantation type as compared to autologous and syngeneic HSCT. However, the prevalence of PTLD was also higher in the allogenic HSCT (5.6% (95%CI: 4.9 to 6.3)) as compared to autologous (2.1% (95%CI: 0.0 to 5.1)) and syngeneic (0.8% (95%CI: 0.0 to 4.2)) HSCT (Figure 6). These differences in PTLD among autologous, syngeneic, and allogeneic HSCT highlight the need for tailored transplant approaches. Autologous HSCT, which uses the patient's own stem cells, eliminates concerns about donor compatibility and reduces the risk of graft-versus-host disease (GVHD) [58]. Our findings indicate that autologous HSCT recipients have substantially lower rates of PTLD, with near-zero post-transplant mortality (Figure 6 and 7). Syngeneic HSCT on the other hand, performed between identical twins, shows promise but remains underexplored, with limited data available on its efficacy and long-term outcomes [59,60]. The prevalence of death cases was less than 1% (i.e. 0.6% (95%CI: 0.4 to 0.9)) among the overall HSCT patients, where not all the patients had PTLD (Figure 7). This phenomenon showed that besides PTLD other risk factors can create critical conditions for patients and can lead to death. Physiological conditions such as age, weakness, comorbidity such as diabetes, cardiovascular diseases, and psychological complications such as anxiety, and depression were some factors that may lead to death [61–63].

Depending on the study region, we did a separate subgroup analysis, observing that the studies were from three different continents: Asia, Europe, and North America. Interestingly, we found that the Asian region had the lowest PTLD rate (1.1% (95%I: 0.7 to 1.5)) compared to the European (27.1% (95%I: 21.4 to 32.8)) and North American (10.1% (95%I: 5.8 to 14.4)) regions, respectively (Figure 8 and 9). Plausible risk factors or other physiological conditions and comorbidities may impact these changes in the prevalence rate. Besides, we observed that no autologous HSCT was done in Europe, which may also had an impact on its higher prevalence rate. On the other hand, among the risk factors we determined from our included articles, HLA mismatch was the most prevalent, followed by T-cell depletion, GVHD, and EBV infection. Besides, immunosuppression, poor T-cell recovery, presence of EBV in tumor/blood, old age, B symptoms (i.e. sweats, pyrexia, and weight loss), and extranodal involvements (i.e. lung, bone marrow, gastrointestinal tract (GIT), skin, and central nervous system (CNS)), presence of high stage of cancer, high dose of Antithymocyte Globulin (ATG), severe aplastic anemia (SAA), cord blood (CB) transplantation, elevated level of lactate dehydrogenase (LDH), cytomegalovirus (CMV) reactivation, and CD68 expression are some other reported risk factors. Previous studies also support these risk factors to be crucial [64,65]. It was also reported that the development of PTLD is closely linked to immunosuppression, particularly the use of ATG, ex vivo T-cell depletion, and mismatched or unrelated donors [66]. These factors weaken the immune system's ability to control EBV-driven B-cell proliferation, significantly increasing the risk of PTLD. Umbilical cord blood grafts further elevate PTLD risk due to their lower T-cell maturity and count [67].

Advances in transplant techniques, such as improved T-cell depletion protocols, have reduced traditional complications but introduced new challenges, including EBV-related PTLD. Reduction of immunosuppression (RI) remains the first-line treatment for PTLD, aiming to restore CTL function while minimizing allograft rejection. For cases with CD20-positive PTLD, rituximab, and anti-CD20 monoclonal antibodies have shown promising results, achieving remission rates of 40–60% when

 doi:10.20944/preprints202603.0439.v1

combined with chemotherapy. However, treatment outcomes vary widely, reflecting the heterogeneity of PTLD. The increasing incidence of PTLD in recent decades highlights the evolving landscape of transplantation. Novel therapies, including chimeric antigen receptor (CAR) T cells targeting CD19 and EBV-specific adoptive T-cell therapies, are being explored as potential game-changers in PTLD management. Factors such as expanded use of HSCT, potent immunosuppressive drugs, and improved diagnostic techniques have contributed to this trend. However, these advances also present opportunities for early detection and intervention, which are critical for improving survival rates [64,68–70]. Nevertheless, routine checks and follow-ups are crucial for diagnosing and managing PTLD, enabling clinicians to initiate preemptive therapies before the onset of clinical symptoms.

5. Limitations

Data from general search engines, news portals, and blogs were excluded from this analysis. Only data from published articles was included.

6. Conclusions

This study represents a significant advancement in the understanding of the prevalence and association of PTLD in HSCT patients. Our findings underscore the imperative of early diagnosis, personalized treatment approaches, and sustained research into the mechanisms underlying PTLD. By addressing these challenges, favorable outcomes for HSCT recipients and the advancement of transplantation medicine can be realized.

Author Contributions: Conceptualization, S.S.K., and D.Z.I.; methodology, S.S.K., A.H.P., A.R.J., M.Z., A.S., and D.S.; software, E.S., S.S.K., R.B. and A.N.M.M-O-R; writing—original draft preparation, A.H.P., A.R.J., M.Z., A.S., D.S., D.Z.I., E.S. and S.S.K.; writing—review and editing, S.S.K., M.A.I., R.B. and A.N.M.M-O-R; visualization, E.S., S.S.K. and M.A.I.; supervision, S.S.K., M.A.I, R.B. and A.N.M.M-O-R. All authors have agreed on the journal to which the article has been submitted and have read and agreed to the published version of the manuscript.

Funding: There are no relevant financial or non-financial competing interests to report.

Data Availability Statement: All data analyzed in this study were obtained from previously published studies and publicly available sources. No new data were generated for this meta-analysis. The datasets supporting the conclusions of this article are available from the corresponding author upon reasonable request.

Conflicts of Interest: No potential competing interest was reported by the author(s). There are no relevant financial or non-financial competing interests to report.

Declaration: This study is not a clinical trial. It is a meta-analysis where secondary data analysis was done. Therefore, no registry, trial registration number, or data of registration was required.

Ethics, Consent to Participate, and Consent to Publish Declarations: Not applicable.

References

1. Ullah, A., et al., *Post-transplant Lymphoproliferative Disorder (PTLD) in the US Population: Demographics, Treatment Characteristics, and Survival Analysis.* Cureus, 2023. **15**(5).
2. Sullivan, K.M., R. Parkman, and M.C. Walters, *Bone marrow transplantation for non-malignant disease.* ASH Education Program Book, 2000. **2000**(1): p. 319-338.
3. Elahimehr, R., A.T. Scheinok, and D.B. McKay, *Hematopoietic stem cells and solid organ transplantation.* Transplantation reviews, 2016. **30**(4): p. 227-234.
4. Dierickx, D. and T.M. Habermann, *Post-transplantation lymphoproliferative disorders in adults.* New England Journal of Medicine, 2018. **378**(6): p. 549-562.
5. Zaffiri, L., et al., *Incidence and outcome of post-transplant lymphoproliferative disorders in lung transplant patients: analysis of ISHLT Registry.* The Journal of Heart and Lung Transplantation, 2020. **39**(10): p. 1089-1099.

6. Loren, A., et al., *Post-transplant lymphoproliferative disorder: a review.* Bone marrow transplantation, 2003. **31**(3): p. 145-155.
7. Shroff, R. and L. Rees, *The post-transplant lymphoproliferative disorder—a literature review.* Pediatric Nephrology, 2004. **19**: p. 369-377.
8. Khandker, S.S., et al., *Diagnostic accuracy of rapid antigen test kits for detecting SARS-CoV-2: a systematic review and meta-analysis of 17,171 suspected COVID-19 patients.* Journal of clinical medicine, 2021. **10**(16): p. 3493.
9. Khandker, S.S., et al., *A systematic review on COVID-19 vaccine strategies, their effectiveness, and issues.* Vaccines, 2021. **9**(12): p. 1387.
10. Islam, M.A., et al., *Immunomodulatory effects of diet and nutrients in systemic lupus erythematosus (SLE): a systematic review.* Frontiers in immunology, 2020. **11**: p. 1477.
11. Islam, M.A., et al., *Prevalence of antiphospholipid antibodies in Behçet's disease: a systematic review and meta-analysis.* PLoS One, 2020. **15**(1): p. e0227836.
12. Jamiruddin, M.R., et al., *Microfluidics technology in SARS-CoV-2 diagnosis and beyond: a systematic review.* Life, 2022. **12**(5): p. 649.
13. Zahra, S., et al., *Prevalence of nephropathy among diabetic patients in North American region: a systematic review and meta-analysis.* Medicine, 2024. **103**(38): p. e39759.
14. Shabbir, N.A., et al., *Prevalence of HIV/AIDS among pregnant women in North American region: A systematic review and meta-analysis.* Medicine, 2024. **103**(44): p. e40339.
15. NIH. *Study Quality Assessment Tools*. 2021 [cited 2024 22 November]; Available from: https://www.nhlbi.nih.gov/health-topics/study-quality-assessment-tools.
16. UNC. *Systematic Reviews: Step 6: Assess Quality of Included Studies*. 2023 [cited 2024 22 November]; Available from: https://guides.lib.unc.edu/systematic-reviews/assess-quality.
17. Mughal, H.B., et al., *Brain natriuretic peptide in acute heart failure and its association with glomerular filtration rate: A systematic review and meta-analysis.* Medicine, 2024. **103**(8): p. e36933.
18. Khandker, S.S., et al., *Association between glomerular filtration rate and β-thalassemia major: a systematic review and meta-analysis.* Thalassemia Reports, 2023. **13**(3): p. 195-205.
19. Marinho-Dias, J., et al., *Post-transplant lymphoproliferative disorder in hematopoietic stem cell transplant patients: A single center retrospective study between 2005 and 2012.* Molecular Medicine Reports, 2018. **18**(5): p. 4650-4656.
20. Fu, L., et al., *Monomorphic post-transplant lymphoproliferative disorder after kidney transplantation and hematopoietic stem cell transplantation: clinicopathological characteristics, treatments and prognostic factors.* Indian Journal of Hematology and Blood Transfusion, 2017. **33**: p. 492-499.
21. Fujimoto, A., et al., *Risk factors and predictive scoring system for post-transplant lymphoproliferative disorder after hematopoietic stem cell transplantation.* Biology of Blood and Marrow Transplantation, 2019. **25**(7): p. 1441-1449.
22. Chiereghin, A., et al., *Prospective Epstein–Barr virus-related post-transplant lymphoproliferative disorder prevention program in pediatric allogeneic hematopoietic stem cell transplant: virological monitoring and first-line treatment.* Transplant Infectious Disease, 2016. **18**(1): p. 44-54.
23. Luo, L., et al., *Post-transplant lymphoproliferative disease after allogeneic hematopoietic stem cell transplantation: a single-center experience.* Ann Transplant, 2014. **19**: p. 6-12.
24. Kalra, A., et al., *Epstein-barr virus DNAemia monitoring for the management of post-transplant lymphoproliferative disorder.* Cytotherapy, 2018. **20**(5): p. 706-714.
25. Overkamp, M., et al., *Comparative analysis of post-transplant lymphoproliferative disorders after solid organ and hematopoietic stem cell transplantation reveals differences in the tumor microenvironment.* Virchows Archiv, 2021. **478**: p. 1135-1148.
26. Yoon, J.H., et al., *Comparative analysis of post-transplant lymphoproliferative disorder after kidney transplantation versus hematopoietic stem cell transplantation.* Transplant International, 2014. **27**(7): p. 721-732.
27. Office, F.P., *Erratum: Characterization and outcome of post-transplant lymphoproliferative disorders within a collaborative study.* Frontiers in Oncology, 2023. **13**: p. 1290218.

28. Kinzel, M., et al., *Risk factors for the incidence of and the mortality due to post-transplant lymphoproliferative disorder after hematopoietic cell transplantation.* Transplantation and Cellular Therapy, 2022. **28**(1): p. 53. e1-53. e10.
29. Sundin, M., et al., *The role of HLA mismatch, splenectomy and recipient Epstein-Barr virus seronegativity as risk factors in post-transplant lymphoproliferative disorder following allogeneic hematopoietic stem cell transplantation.* Haematologica, 2006. **91**(8): p. 1059-1067.
30. Fu, L., et al., *Allogeneic hematopoietic stem-cell transplantation for adult and adolescent hemophagocytic lymphohistiocytosis: a single center analysis.* International journal of hematology, 2016. **104**: p. 628-635.
31. Park, B.-K., et al., *Allogeneic hematopoietic stem cell transplantation in congenital hemoglobinopathies with myeloablative conditioning and rabbit anti-thymocyte globulin.* Blood research, 2018. **53**(2): p. 145.
32. Balaguer-Rosello, A., et al., *Central nervous system involvement in Epstein–Barr virus-related post-transplant lymphoproliferative disorders after allogeneic hematopoietic stem cell transplantation.* Transplantation and Cellular Therapy, 2021. **27**(3): p. 261. e1-261. e7.
33. Choi, J.-H., et al., *Clinical characteristics of monomorphic post-transplant lymphoproliferative disorders.* Journal of Korean medical science, 2010. **25**(4): p. 523-526.
34. Liu, Z.-X., et al., *Clinical Characteristics, Treatment and Prognosis of PTLD after allogeneic Hematopoietic Stem Cell Transplantation.* Zhongguo shi yan xue ye xue za zhi, 2018. **26**(2): p. 535-540.
35. Pagliuca, S., et al., *Epstein-Barr virus-associated post-transplantation lymphoproliferative disease in patients who received anti-CD20 after hematopoietic stem cell transplantation.* Biology of Blood and Marrow Transplantation, 2019. **25**(12): p. 2490-2500.
36. Watson, C., et al., *Retrospective database analysis of healthcare resource utilization and costs in patients who develop post-transplant lymphoproliferative disease within the first year following allogeneic hematopoietic stem cell transplants.* Journal of Medical Economics, 2020. **23**(10): p. 1159-1167.
37. Chen, D., et al., *Post-transplant lymphoproliferative disorder: a clinicopathologic study of 15 cases.* Zhonghua Bing li xue za zhi= Chinese Journal of Pathology, 2012. **41**(9): p. 607-612.
38. Gündüz, M., et al., *Subsequent malignancies after allogeneic hematopoietic stem cell transplantation.* Clinical Transplantation, 2017. **31**(7): p. e12987.
39. Fujimoto, A., et al., *Low incidence of posttransplant lymphoproliferative disorder after allogeneic stem cell transplantation in patients with lymphoma treated with rituximab.* Hematological Oncology, 2020. **38**(2): p. 146-152.
40. Czyzewski, K., et al., *Intrathecal therapy with rituximab in central nervous system involvement of post-transplant lymphoproliferative disorder.* Leukemia & Lymphoma, 2013. **54**(3): p. 503-506.
41. Ali, S., et al., *Hemophagocytic lymphohistiocytosis is a sign of poor outcome in pediatric Epstein-Barr virus-associated post-transplant lymphoproliferative disease after allogeneic hematopoietic stem cell transplantation.* Pediatric transplantation, 2019. **23**(1): p. e13319.
42. Wareham, N.E., et al., *The value of EBV DNA in early detection of post-transplant lymphoproliferative disorders among solid organ and hematopoietic stem cell transplant recipients.* Journal of Cancer Research and Clinical Oncology, 2018. **144**: p. 1569-1580.
43. Courville, E.L., et al., *EBV-negative monomorphic B-cell post-transplant lymphoproliferative disorders are pathologically distinct from EBV-positive cases and frequently contain TP53 mutations.* Modern Pathology, 2016. **29**(10): p. 1200-1211.
44. Hahn, S.M., et al., *Incidence and Features of Lymphoid Proliferation and Lymphomas after Solid Organ or Hematopoietic Stem Cell Transplantation in a National Database Cohort.* Cancer Research and Treatment: Official Journal of Korean Cancer Association, 2023. **56**(1): p. 305.
45. Styczynski, J., et al., *Response to rituximab-based therapy and risk factor analysis in Epstein Barr virus–related lymphoproliferative disorder after hematopoietic stem cell transplant in children and adults: a study from the Infectious Diseases Working Party of the European Group for Blood and Marrow Transplantation.* Clinical infectious diseases, 2013. **57**(6): p. 794-802.
46. Xu, L.P., et al., *Epstein-Barr Virus-Related Post-Transplantation Lymphoproliferative Disorder after Unmanipulated Human Leukocyte Antigen Haploidentical Hematopoietic Stem Cell Transplantation: Incidence, Risk Factors, Treatment, and Clinical Outcomes.* Biol Blood Marrow Transplant, 2015. **21**(12): p. 2185-2191.

47. Gilman, A.L., et al., *Alternative donor hematopoietic stem cell transplantation for sickle cell disease.* Blood advances, 2017. **1**(16): p. 1215-1223.
48. Gonzalez-Farre, B., et al., In vivo *intratumoral Epstein–Barr virus replication is associated with XBP1 activation and early-onset post-transplant lymphoproliferative disorders with prognostic implications.* Modern Pathology, 2014. **27**(12): p. 1599-1611.
49. Kroll, J., et al., *Lytic and latent EBV gene expression in transplant recipients with and without post-transplant lymphoproliferative disorder.* Journal of clinical virology, 2011. **52**(3): p. 231-235.
50. Zhou, X., et al., *Clinical value of plasma and peripheral blood mononuclear cells Epstein–Barr Virus DNA dynamics on prognosis of allogeneic stem cell transplantation.* Frontiers in Cellular and Infection Microbiology, 2022. **12**: p. 980113.
51. d'Aveni, M., et al., *The clinical value of concomitant Epstein Barr virus (EBV)-DNA load and specific immune reconstitution monitoring after allogeneic hematopoietic stem cell transplantation.* Transplant Immunology, 2011. **24**(4): p. 224-232.
52. Nash, R.A., et al., *Epstein-Barr virus-associated posttransplantation lymphoproliferative disorder after high-dose immunosuppressive therapy and autologous CD34-selected hematopoietic stem cell transplantation for severe autoimmune diseases.* Biology of Blood and Marrow Transplantation, 2003. **9**(9): p. 583-591.
53. Comoli, P., et al., *T cell therapy of Epstein–Barr virus and adenovirus infections after hemopoietic stem cell transplant.* Blood Cells, Molecules, and Diseases, 2008. **40**(1): p. 68-70.
54. Blaes, A.H., et al., *Monitoring and preemptive rituximab therapy for Epstein-Barr virus reactivation after antithymocyte globulin containing nonmyeloablative conditioning for umbilical cord blood transplantation.* Biology of Blood and Marrow Transplantation, 2010. **16**(2): p. 287-291.
55. Comoli, P., et al., *Preemptive therapy of EBV-related lymphoproliferative disease after pediatric haploidentical stem cell transplantation.* American Journal of Transplantation, 2007. **7**(6): p. 1648-1655.
56. Park, S.H., et al., *Clinical characteristics and outcomes of posttransplant lymphoproliferative disorders following allogeneic hematopoietic stem cell transplantation in Korea.* Journal of Korean Medical Science, 2006. **21**(2): p. 259.
57. Althubaiti, S., et al., *Lymphocyte subset at time of Epstein-Barr viremia post-allogeneic hematopoietic stem cell transplantation in children may predict development of post-transplant lymphoproliferative disease: CD8: CD20 ratio as a sensitive predictor.* Pediatric Transplantation, 2019. **23**(8): p. e13574.
58. Clinic, M. *Autologous stem cell transplant*. 2025 [cited 2025 1 February]; Available from: https://www.mayoclinic.org/tests-procedures/autologous-stem-cell-transplant/pyc-20384859.
59. Niederwieser, D., et al., *Hematopoietic stem cell transplantation activity worldwide in 2012 and a SWOT analysis of the Worldwide Network for Blood and Marrow Transplantation Group including the global survey.* Bone marrow transplantation, 2016. **51**(6): p. 778-785.
60. Gale, R.P., et al., *Identical-twin bone marrow transplants for leukemia.* Annals of internal medicine, 1994. **120**(8): p. 646-652.
61. Briggs, J.D., *Causes of death after renal transplantation.* Nephrology Dialysis Transplantation, 2001. **16**(8): p. 1545-1549.
62. Washer, G.F., et al., *Causes of death after kidney transplantation.* Jama, 1983. **250**(1): p. 49-54.
63. Dew, M.A., et al., *Depression and anxiety as risk factors for morbidity and mortality after organ transplantation: a systematic review and meta-analysis.* Transplantation, 2016. **100**(5): p. 988-1003.
64. Al-Mansour, Z., B.P. Nelson, and A.M. Evens, *Post-transplant lymphoproliferative disease (PTLD): risk factors, diagnosis, and current treatment strategies.* Current hematologic malignancy reports, 2013. **8**: p. 173-183.
65. Cockfield, S., *Identifying the patient at risk for post-transplant lymphoproliferative disorder.* Transplant infectious disease, 2001. **3**(2): p. 70-78.
66. Landgren, O., et al., *Risk factors for lymphoproliferative disorders after allogeneic hematopoietic cell transplantation.* Blood, The Journal of the American Society of Hematology, 2009. **113**(20): p. 4992-5001.
67. Styczynski, J., et al., *Management of Epstein-Barr Virus infections and post-transplant lymphoproliferative disorders in patients after allogeneic hematopoietic stem cell transplantation: Sixth European Conference on Infections in Leukemia (ECIL-6) guidelines.* Haematologica, 2016. **101**(7): p. 803.

 doi:10.20944/preprints202603.0439.v1

68. Elstrom, R., et al., *Treatment of PTLD with rituximab or chemotherapy.* American Journal of Transplantation, 2006. **6**(3): p. 569-576.
69. Cheng, J., et al., *Systematic review and meta-analysis of post-transplant lymphoproliferative disorder in lung transplant recipients.* Clinical transplantation, 2018. **32**(5): p. e13235.
70. Dang, B.N., et al., *Treatment of post-transplant lymphoproliferative disorder (PTLD) in a heart transplant recipient with chimeric antigen receptor T-cell therapy.* Pediatric Transplantation, 2021. **25**(5): p. e13861.