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Original Article
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Incidence and Characteristics of Infectious Complications in Multiple Myeloma Patients Treated With Bispecific Antibodies

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ABSTRACT

Background: Bispecific antibodies (BsAbs) are a new class of immunotherapeutic agents for patients with multiple myeloma (MM). Although this new class of drug is associated with good disease control, they are also associated with increased risk of infectious complications. Since endemic community-acquired and nosocomial infections vary across the globe, we conducted this study to report real-world data of infectious complications associated with BsAbs in Korean population.

Methods: We retrospectively reviewed all MM patients who received BsAb therapy between January 2021 and January 2024 at Seoul National University Hospital. We identified 61 patients who underwent BsAb therapy at our center with median follow-up of 34 weeks (95% confidence interval, 25.85–55.85). Thirty-three patients (54%) received B-cell maturation antigen (BCMA)-targeting BsAb, and 30 (49%) received combination therapy.

Results: Of the 61 patients, 39 (64%) had at least one episode of infection. A total of 69 infections affecting patient management occurred during the study period, 3% grade 1 infection, 8% grade 2, 72% grade 3, 8% grade 4 and 8% grade 5. The most common type of infection was lower respiratory tract infection (n = 32/69, 46%), followed by systemic infection (n = 21/69, 30%). Etiology wise, viral infections were most common (67%), followed by fungal infections (13%) and bacterial infections (10%). Among viral infections, cytomegalovirus (CMV) was most common. Patients treated with BCMA-targeting BsAb or combination therapy were associated with higher incidence of CMV reactivation and clinically significant CMV infection.

Conclusion: Particular pattern of infectious complications including CMV infection was noted in Korean patients. Identifying and determining the nature of infectious disease dynamics is becoming increasingly important for optimal resource allocation and shaping healthcare policies. In this regard, our first-in-Asian population study holds its value.

Keywords: Multiple Myeloma; Bispecific Antibodies; Infection; Epidemiology; Adverse Events

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The authors have no potential conflicts of interest to disclose.

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

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INTRODUCTION

Multiple myeloma (MM) patients are at an increased risk of infection from diagnosis due to the immunosuppressive nature of the disease itself and the host factors (older age at diagnosis, compromised renal function).[1] Ensuing treatment based on immunomodulatory drugs (IMiDs), proteasome inhibitors (PIs) and anti-CD38 antibody aggravates the risk of infection as IMiDs are associated with neutropenia while PIs and anti-CD38 antibody are associated with certain viral reactivations.[2]

Now we have a new class of immunotherapeutic agents: the bispecific antibodies (BsAbs). BsAbs form an immune synapse between T-cells via surface marker CD3 and malignant cell markers including B-cell maturation antigen (BCMA), G protein-coupled receptor (GPRC5D) and Fc Receptor-Like 5 (FcRH5).[3] Although this new class of drug is associated with good disease control, they are also associated with increased risk of cytopenia, hypogammaglobulinemia, and infectious complications.[4-6]

Since seroprevalence of latent viruses or epidemiology of community-acquired and nosocomial infections vary across the globe, it is important to comprehensively understand the similarities and differences amongst patients undergoing the same treatment. Recognizing the lack of East Asian representation in recent studies reporting the infection risk associated with BsAbs,[7,8] we conducted this study.

METHODS

Patients and event definitions

All MM patients who received BsAb therapy between January 2021 and January 2024 at Seoul National University Hospital were included. Patients receiving BsAb therapy through a range of clinical trials were also included. We collected clinical data retrospectively, including patient characteristics, clinical features, laboratory findings, bone marrow examination results, immunophenotype findings, molecular analyses, treatment, treatment outcomes, and any events associated with the treatments.

Triple refractory MM is defined as disease refractory to 3 previous therapies including IMiDs and PIs. Penta refractory MM is defined as disease refractory to 5 previous therapies including 2 IMIDs, 2 PIs and monoclonal antibodies (daratumumab).

Infections were categorized as microbiologically defined infection (MDI), clinically defined infection (CDI), or fever of unknown focus (FUF)[7,8] and severity graded according to published criteria.[9] The MDI was defined as a laboratory isolated pathogen with a compatible clinical syndrome, CDI was defined by a compatible clinical syndrome without an isolated pathogen, and FUF was defined as the presence of fever and the absence of compatible microbiology or focal symptoms. Systemic infection included bacteremia, viremia, fungemia and infections affecting multiple organs. Complications of BsAb therapy such as cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS) were defined according to published criteria.[10]

CMV reactivation was defined as the detection of CMV replication in blood (or other sites) among CMV-seropositive patients.[11] Clinically significant CMV infection was defined as

CMV disease or CMV viremia leading to preemptive treatment.[12] CMV disease was defined as the presence of attributable clinical symptoms and/or signs with the documentation of CMV in tissue from the relevant organ by histopathology, virus isolation, rapid culture, immunohistochemistry, or DNA hybridization.[13]

At our institution, valaciclovir 500 mg once daily is routinely used for herpes simplex virus (HSV) and varicella zoster virus (VZV) prophylaxis. Trimethoprim/sulfamethoxazole 80 mg/400 mg once daily is also routinely given as *Pneumocystis jirovecii* pneumonia (PJP) prophylaxis. Antibacterial and antifungal prophylaxis is guided by an individualized risk-based approach. We generally used prophylactic antifungal agents when the patient has the past medical history of fungal infection, and used prophylactic antibacterial agents when the patient has the past medical history of systemic bacterial infection or showed prolonged neutropenia of any grade. We used granulocyte-colony stimulating factor for all grade 3–4 neutropenic patients.

Hypogammaglobulinemia is assessed every 4 to 6 weeks by subtracting out the monoclonal immunoglobulin (Ig) and defined as IgG < 400 mg/dL. Intravenous immunoglobulin (IVIG) is routinely replaced for hypogammaglobulinemia.

Statistical analysis

Differences between groups were assessed using a Student's *t*-test or one-way analysis of variance for continuous variables, and Pearson χ^2 test for categorical variables, as indicated. Data available up to 30 April 2024 were used. *P* values of < 0.05 were considered statistically significant.

Infectious events were analyzed with time-dependent cumulative incidence using the Fine-Gray competing risk model with death or progression as a competing risk event. These data were analyzed using the Statistical Package for the Social Sciences software (IBM® SPSS® Statistics, version 22.0; IBM Corp., Armonk, NY, USA) and R language (version 4.2.1 or higher; R Foundation for Statistical Computing, Vienna, Austria).

Ethics statement

This study was conducted according to the Declaration of Helsinki and was approved by the Institutional Review Board at Seoul National University Hospital (H-2404-110-1532). The requirement for informed consent was waived by the board.

RESULTS

Patient characteristics

Table 1 shows the baseline characteristics of the enrolled patients. We identified 61 patients who underwent BsAb therapy at our center with median follow-up of 34 weeks (95% confidence interval [CI], 25.85–55.85). Thirty-three patients (54%) received BCMA-targeting BsAb, and 30 (49%) received combination therapy (CD38 targeting monoclonal antibody + BsAb or dual BsAb). The median age at BsAb therapy was 63 years (range 33–81), with a median of 4 prior lines of treatment exposure. CRS occurred in 37 (60.7%) and ICANS in 0 patients.

Incidence and characteristics of infection

Of the 61 patients, 40 (66%) had at least one episode of infection (MDI, CDI or FUF). A total of 69 infections affecting patient management occurred during the study period, of which

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Table 1. Baseline disease characteristics

Characteristics	Values
No. of patients	61
Age at BsAb tx, yr	63 (33–81)
Sex, male	42 (68.9)
Duration of BsAb tx, mon	9 (1–30)
BCMA-targeting BsAb	33 (54.1)
Combination therapy	30 (49.2)
ISS	
I	37 (60.7)
II	15 (24.6)
III	9 (14.8)
Immunochemical subtype	
IgG	30 (49.2)
IgA	9 (14.8)
IgM	1 (1.6)
IgD	1 (1.6)
Light chain	20 (32.8)
Prior treatment	
Triple refractory	37 (60.7)
Penta refractory	23 (37.7)
Prior lines	4 (1–12)
Prior HSCT	44 (72.1)
All grade CRS	37 (60.7)
Grade 1	30 (81.1)
Grade 2	7 (18.9)
Corticosteroids	9 (24.3)
Tocilizumab	16 (43.2)
Both	7 (18.9)
All grade ICANS	0
Other neurotoxicity	5 (8.2)
Neutropenia	
Any grade	37 (60.7)
Grade 3–4	29 (47.5)
CMV status	
Seropositivity	58 (95.1)
Viremia	0
Antigenemia	0
IVIG administration	20 (32.8)

Values are presented as median (range) or number (%).
BsAb = bispecific antibody, Tx = treatment, ISS = International Staging System, Ig = immunoglobulin, HSCT = hematopoietic stem cell transplantation, CRS = cytokine release syndrome, ICANS = immune effector cell-associated neurotoxicity syndrome, CMV = cytomegalovirus, IVIG = intravenous immunoglobulin.

63 (91%) were MDI, 4 (6%) were CDI and 2 (3%) were FUF. As shown in **Table 2**,[14,15] the predominant localization was the lower respiratory tract infection (n = 32/69, 46%), followed by systemic infection (n = 21/69, 30%).

Hospitalization was required in 54 (88%) infectious episodes, and intensive care unit admission was required in 7 (10%) episodes. There was a total of 5 (8%) deaths attributed to infection during the study period. As shown in **Table 3**, in 69 infection events, 2 (3%) episodes were grade 1 infection, 5 (8%) were grade 2, 44 (72%) were grade 3, 5 (8%) were grade 4 and 5 (8%) were grade 5.

Etiology wise, viral infections were most common (**Table 2**), followed by fungal infections and bacterial infections. For fungal infections, there were 2 cases of invasive pulmonary aspergillosis and 7 cases of PJP. For bacterial infections there were 3 cases of bacteremia, 1 streptococcal bacteremia, 1 *Escherichia coli* bacteremia and 1 *Pseudomonas aeruginosa* bacteremia.

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Table 2. Incidence and characteristics of infectious complications

Characteristics	Total
Site of infection, all	69 events
Systemic	21 (30)
Upper respiratory tract	3 (4)
Lower respiratory tract	32 (46)
Gastrointestinal tract	2 (3)
Genitourinary tract	1 (1)
Skin and soft tissue	8 (12)
CNS and ocular	2 (3)
Viral infections	46 (67)
CMV, clinically significant[14]	15
HSV	6
VZV	6
HBV	2
Respiratory viruses[a]	16
Severe SARS-CoV-2[15] requiring oxygen	12
JC virus	1
Fungal infections	9 (13)
Aspergillus spp.	2
Pneumocystis jirovecii	7
Bacterial infection	7 (10)
Tuberculosis	1 (1)

Values are presented as number (%).
CNS = central nervous system, CMV = cytomegalovirus, HSV = herpes simplex virus, VZV = varicella zoster virus, HBV = hepatitis B virus, SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2, JC = John Cunningham.
[a]Includes influenza/parainfluenza virus (n = 2), respiratory syncytial virus (n = 1), and SARS-CoV-2 (n = 13).

Table 3. Comparison with previous studies involving patients receiving bispecific antibodies therapy

Characteristics	Current study	Australia[7]	France[8]
No. of patients	61	39	229
BCMA vs. non-BCMA	33 (54) vs. 28 (46)	12 (31) vs. 27 (69)	200 vs. 29
Prior lines of therapy	4 (1–12)	6 (4–7)	4 (0–15)
Infection episodes	69	111	234
Infection category			
MDI	63 (91)	33 (30)	165 (71)
CDI	4 (6)	43 (39)	62 (26)
FUF	2 (3)	35 (32)	7 (3.0)
Bacterial, %	10	39	56
Viral, %	67	58	38
CMV (CMV events/total viral events)	15/46 (33)	4/22 (18)	8/63 (13)
Fungal, %	13	0	5
Tuberculosis, %	1	3	0
Grade of infections			
1	2 (3)	37 (33)	12 (5)
2	5 (8)	50 (45)	98 (41)
3	44 (72)	22 (20)	75 (32)
4	5 (8)	0	28 (12)
5	5 (8)	2 (2)	20 (9)

Values are presented as median (range) or number (%).
BCMA = B-cell maturation antigen, MDI = microbiologically defined infection, CDI = clinically defined infection, FUF = fever of unknown focus, CMV = cytomegalovirus.

The last patient with *Pseudomonas* bacteremia demised due to infection while in complete remission after 4 cycles of treatment. There was one case of pulmonary tuberculosis (TB) in a 47-year-old male without previous history of TB.

Global cumulative incidence of first infection was 76.9% (95% CI, 63.1–90.7%), with specific rates of 13.5% at 30 days (95% CI, 4.1–22.8%), 36.5% at 90 days (95% CI, 23.3–49.5%), and 63.5% at 180 days (95% CI, 49.7–77.3%) (**Fig. 1**).

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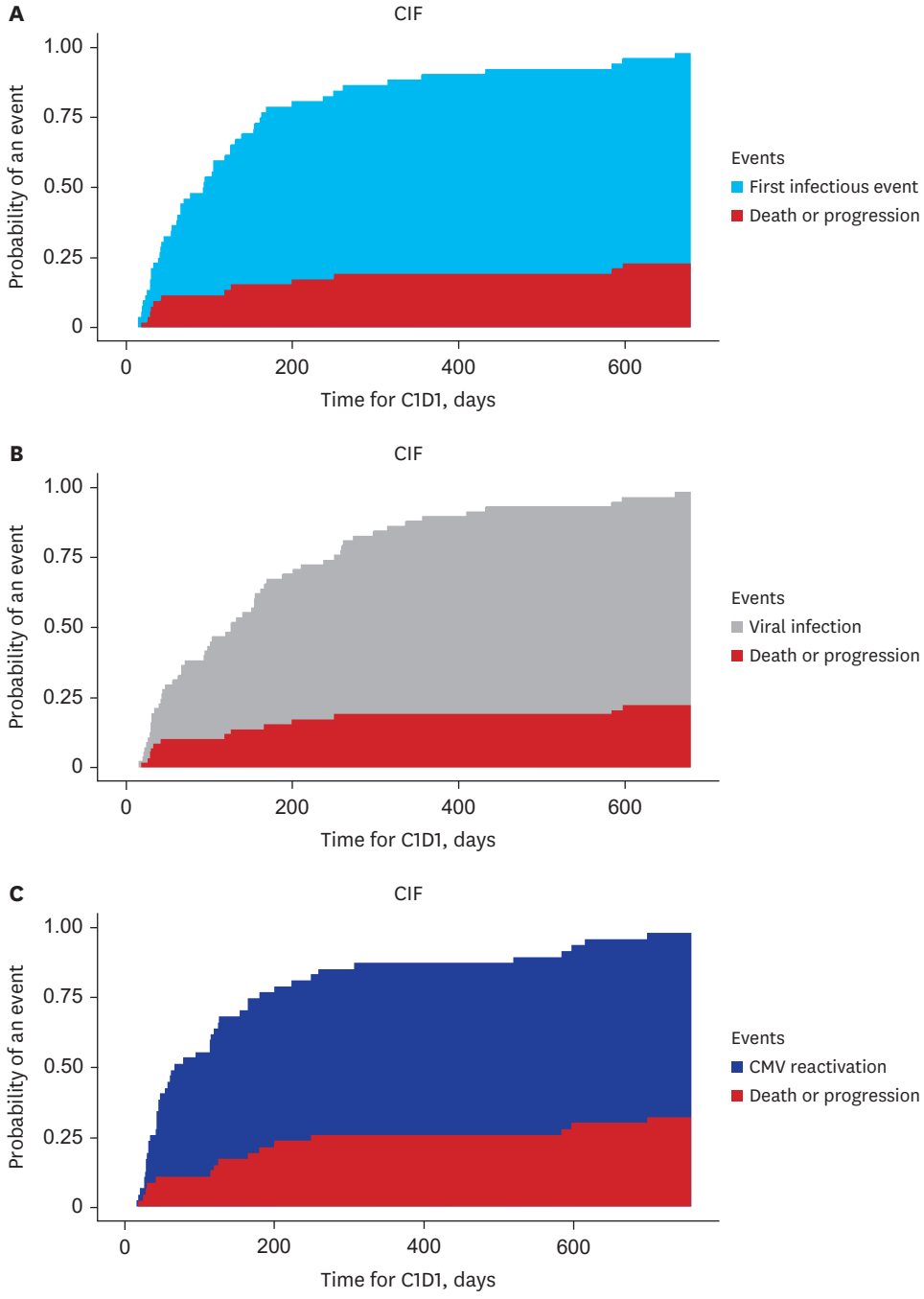

Fig. 1. Cumulative incidence of outcomes.
(**A**) CIF of first infectious event. Death or disease progression was used as a competing risk event. (**B**) The CIF of viral infections. (**C**) The CIF of CMV reactivation.
CIF = cumulative incidence function, CMV = cytomegalovirus.

Viral infections

There were 46 (67%) episodes of viral infections. Respiratory viruses were most common with 16/46 (35%) events, mostly attributed to SARS-CoV-2 infection. There were 2 deaths associated with severe SARS-CoV2 infection. One patient was in very good partial response after 1 cycle of therapy, while the other patient showed stable disease after 2 cycles of therapy. Global cumulative incidence of viral infection was 77.6% (95% CI, 64.5–90.6%), with specific

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Table 4. CMV specifics

Characteristics	Total (N = 61)	BCMA (n = 33)	Non-BCMA (n = 28)	*P* value[a]	Single immunotherapy (n = 31)	Combination therapy (n = 30)	*P* value[b]
Reactivation[11]	30 (49.2)	22 (66.7)	8 (28.6)	0.005	9 (29.0)	21 (70.0)	0.002
Median time to reactivation, days	58	58	58.5	0.307	43	65	0.287
Clinically significant CMV infection	15 (24.6)	13 (39.4)	2 (7.1)	0.006	5 (16.1)	10 (33.3)	0.119
CMV disease	4	3 (9.1)	1 (3.6)	0.385	1 (3.2)	3 (10.0)	0.354
Lung	3	3	0		0	3	
Retinitis	1	0	1		1	0	

Values are presented as number (%).
CMV = cytomegalovirus, BCMA = B-cell maturation antigen.
[a]BCMA group vs. non-BCMA group; [b]Single immunotherapy group vs. combination therapy group.

rates of 12.1% at 30 days (95% CI, 3.6–20.5%), 27.6% at 90 days (95% CI, 16.0–39.2%), and 51.7% at 180 days (95% CI, 38.7–64.8%) (**Fig. 1**).

Cytomegalovirus (CMV) infection was next in line with 15/46 (33%) events. To be specific (**Table 4**), CMV reactivation[11] was seen in 49% of the patients (30/61). Global cumulative incidence of CMV reactivation was 66.0% (95% CI, 51.4–80.5%), with specific rates of 10.6% at 30 days (95% CI, 1.7–19.6%), 42.6% at 90 days (95% CI, 28.2–56.9%), and 55.3% at 180 days (95% CI, 40.8–69.9%) (**Fig. 1**). Among them, 15 were clinically significant[12] requiring intervention. There were 4 cases of CMV disease: 3 CMV pneumonia and 1 CMV retinitis. Two of the 3 patients with CMV pneumonia demised due to infection. Both were in very good partial response at the time of death.

Interestingly, patients receiving BCMA-targeting agents showed higher incidence of CMV reactivation as shown in **Table 4** ($P = 0.005$). The incidence of clinically significant CMV infection was also higher in BCMA group ($P = 0.006$). Not surprisingly, CMV reactivation was associated with combination therapy. Fortunately, there were no significant differences in clinically significant CMV infections among patients receiving single immunotherapy vs. combination therapy.

DISCUSSION

As newer immunotherapies including BsAbs prolong survivals of MM patients, supportive care is becoming increasingly important to ensure good quality of life and sustain treatment response. Infection control constitutes a major pillar of such supportive care, not only because MM disease itself is associated with B-cell depletion and T-cell dysfunction,[16] but also because of the susceptibility and the frailty of the patients from undergoing multiple prior lines of therapy (**Tables 5** and **6**). When dealing with infections varying prevalence and determinants of opportunistic infections per region should also be taken into consideration. We thought the first step to optimizing preventative and therapeutic strategies was determining the nature of infectious disease dynamics.

As results, we found that more than half of the patients (64%) treated with BsAbs experience infectious complications. This percentage is comparable to previous reports,[4,6-8] but we had more cases of grade 3 or higher infections (**Table 3**). Due to the retrospective nature of the study and the different patterns of healthcare utilization, we cannot draw any definitive conclusions. However, considering the generally lower proportion of chemotherapy tolerable patients in Asian population,[17,18] caution is warranted. Spacing out the BsAbs delivery

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Table 5. Baseline characteristics with BsAb target

Characteristics	BCMA	Non-BCMA
No. of patients	33	28
Age at BsAb tx, yr	63 (33–76)	62 (43–81)
Sex, male	26 (78.8)	16 (57.1)
BsAb tx duration, mon	8 (1–24)	13 (1–30)
ISS		
I	20 (60.6)	17 (60.7)
II	8 (24.2)	7 (25.0)
III	5 (15.2)	4 (14.3)
Immunochemical subtype		
IgG	19 (57.6)	11 (39.3)
IgA	2 (6.1)	7 (25.0)
IgM	1 (3.0)	0
IgD	1 (3.0)	0
Light chain	10 (30.3)	10 (35.7)
Prior treatment		
Triple refractory	20 (60.6)	17 (60.7)
Penta refractory	13 (39.4)	10 (35.7)
Prior lines	4 (1–8)	4 (1–12)
Prior HSCT	26 (78.8)	16 (64.3)
All grade CRS	19 (57.6)	18 (64.3)
Grade 1	17 (89.5)	13 (72.2)
Grade 2	2 (10.5)	5 (27.8)
Corticosteroids	5 (26.3)	4 (22.2)
Tocilizumab	6 (31.6)	10 (55.6)
All grade ICANS	0	0
Other neurotoxicity	2 (6.1)	3 (10.7)
Neutropenia		
Any grade	23 (69.7)	14 (50.0)
Grade 3–4	19 (57.6)	10 (35.7)
IVIG administration	17 (51.5)	3 (10.7)
Any infection	26 (78.8)	14 (50.0)

Values are presented as median (range) or number (%).
BsAb = bispecific antibody, BCMA = B-cell maturation antigen, Tx = treatment, ISS = International Staging System, Ig = immunoglobulin, HSCT = hematopoietic stem cell transplantation, CRS = cytokine release syndrome, ICANS = immune effector cell-associated neurotoxicity syndrome, IVIG = intravenous immunoglobulin.

schedule after achieving adequate MM disease control might be an option in frail patients, though further study is required.

The higher proportion of viral infections in our patients, with special regards to CMV, also deserves attention. We saw higher rates of viral infections (67%) compared to Australia (58%)[7] or France (38%).[8] More specifically, 49.2% of the patients had CMV reactivation within 58 days of BsAbs treatment and 24.6% had clinically significant CMV infection (**Table 4**). The high level of CMV seropositivity in Korea[19-21] and Asia countries[22] is a well-documented phenomenon. Recent guideline by Raje et al.[5] strongly endorses use of IVIG for hypogammaglobulinemia and antiviral prophylaxis against HSV and VZV in all patients, but monitoring of CMV is only recommended in high-risk patients. Based on our experience, we consider all our MM patients undergoing BsAbs high-risk and actively monitor quantitative CMV DNA PCR on regular basis. We consider targeted preemptive therapy at a viral load of ≥ 500 copies/mL.[23] More systematic evidence-based prophylactic regimen, such as use of fixed duration letermovir,[14] should be assessed in further prospective study.

Lastly, it seems that patients undergoing BCMA-directed BsAbs requires more vigilance compared to those receiving non-BCMA agents (**Table 7**). BCMA is a cell surface marker which is expressed on both malignant and normal plasma cells, though the expression level is

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Table 6. Baseline characteristics with single or combination therapy

Characteristics	Single	Combination
No. of patients	31	30
Age at BsAb tx, yr	63 (43–81)	60 (33–75)
Sex, male	18 (58.1)	14 (46.7)
BsAb tx duration, mon	11 (1–30)	11 (1–26)
ISS		
I	16 (51.6)	21 (70.0)
II	9 (29.0)	6 (20.0)
III	6 (19.4)	3 (10.0)
Immunochemical subtype		
IgG	11 (35.5)	19 (63.3)
IgA	7 (22.6)	2 (6.7)
IgM	0	1 (3.3)
IgD	1 (3.2)	0
Light chain	12 (38.7)	8 (26.7)
Prior treatment		
Triple refractory	24 (77.4)	13 (43.3)
Penta refractory	14 (45.2)	9 (30.0)
Prior lines	5 (1–12)	4 (1–7)
Prior HSCT	21 (67.7)	21 (70.0)
All grade CRS	18 (58.1)	19 (63.3)
Grade 1	13 (72.2)	17 (89.5)
Grade 2	5 (27.8)	2 (10.5)
Corticosteroids	7 (38.9)	2 (10.5)
Tocilizumab	8 (44.4)	8 (42.1)
All grade ICANS	0	0
Other neurotoxicity	1 (3.2)	4 (13.3)
Neutropenia		
Any grade	19 (61.3)	18 (60.0)
Grade 3–4	11 (35.5)	18 (60.0)
IVIG administration	7 (22.6)	13 (43.3)
Any infection	15 (48.4)	25 (83.3)

Values are presented as median (range) or number (%).
BsAb = bispecific antibody, Tx = treatment, ISS = International Staging System, Ig = immunoglobulin, HSCT = hematopoietic stem cell transplantation, CRS = cytokine release syndrome, ICANS = immune effector cell-associated neurotoxicity syndrome, IVIG = intravenous immunoglobulin.

much increased in malignant plasma cells.[24] It is easily assumed that BCMA-targeting BsAbs may decrease the normal mature B cell count,[25] followed by the higher risk of infections for patients treated with BCMA-targeting agents than non-BCMA-targeting agents.[4,6,25] In our study, patients with BCMA-targeting agents showed higher incidence of CMV reactivation ($P = 0.005$, **Table 4**) and clinically significant CMV infection ($P = 0.006$, **Table 4**) despite the similar baseline patient/disease characteristics (**Tables 5** and **6**). Likewise, in patients receiving combination therapy, more comprehensive plasma cell aplasia would be induced than patients with BsAb monotherapy. With the combination therapy, higher all-grade and grade ≥ 3 infections were observed than monotherapy in other studies.[6] Similarly, our study showed significant difference in CMV reactivation between patients treated with combination therapy and BsAb monotherapy as shown in **Table 4** ($P = 0.002$).

All in all, we provide the first set of infectious complication data from homogenous East Asian MM patients treated with BsAbs. Our study has several limitations, including the retrospective nature of the study in single institution, reliance on descriptive analysis, and the absence of a standardized treatment protocol. However, newer therapeutic agents including BsAbs are already very expensive, thus dealing with complications secondary to these types of treatment will ultimately tantamount to significant financial burden in any given healthcare system. Therefore, implementing risk adaptive prophylactic measures and establishing

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Table 7. Incidence and characteristics of infectious complications with subgroups

Characteristics	BCMA	Non-BCMA	Single	Combination
Site of infection, all	52 events	17 events	24 events	45 events
Systemic	17 (33)	4 (24)	5 (21)	16 (36)
Upper respiratory tract	2 (4)	1 (6)	1 (4)	2 (4)
Lower respiratory tract	25 (48)	7 (41)	11 (46)	21 (47)
Gastrointestinal tract	2 (3)	0	0	2 (4)
Genitourinary tract	1 (2)	0	1 (4)	0
Skin and soft tissue	4 (8)	4 (24)	5 (21)	3 (7)
CNS and ocular	1 (2)	1 (6)	1 (4)	1 (2)
Viral infections	35 (67)	11 (65)	16 (67)	30 (67)
CMV, clinically significant[14]	13	2	5	10
HSV	3	3	3	3
VZV	3	3	4	2
HBV	2	0	0	2
Respiratory viruses[a]	13	3	4	12
Severe SARS-CoV-2[15] requiring oxygen	10	2	1	11
JC virus	1	0	0	1
Fungal infections	7 (13)	2 (12)	3 (13)	6 (13)
Aspergillus spp.	0	2	1	1
Pneumocystis jirovecii	7	0	2	5
Bacterial infection	4 (8)	3 (18)	4 (17)	3 (7)
Tuberculosis	1 (2)	0	0	1 (2)

Values are presented as number (%).
BCMA = B-cell maturation antigen, CNS = central nervous system, CMV = cytomegalovirus, HSV = herpes simplex virus, VZV = varicella zoster virus, HBV = hepatitis B virus, SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2, JC = John Cunningham.
[a]Includes influenza/parainfluenza virus (n = 2), respiratory syncytial virus (n = 1), and SARS-CoV-2 (n = 13).

active monitoring know-hows will become even more important for fair allocation of limited medical resources. We believe our data will bridge the gap in knowledge regarding novel therapy in MM and more importantly sets ground for region-specific adaptations of current guidelines on infection risk mitigation and subsequently treatment optimization.

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