Non-tuberculous mycobacterial infections following teclistamab in multiple myeloma

Teclistamab, a bispecific T-cell engager (BiTE) targeting B-cell maturation antigen (BCMA), is approved and increasingly being utilized for relapsed/refractory multiple myeloma (RRMM). A *post hoc* analysis of the phase I/II registration trial reported infections in 80% of patients after teclistamab.[1] Fifty-five percent of patients had grade ≥3 infections.[1] Pooled analyses of teclistamab trials estimate the rate of infections at 46-76% (grade≥3: 7-45%),[2,3] consistent with that seen in real-world studies: 41-62% of patients (grade ≥3: 33-58%).[4-6] Importantly, there are no reports of non-tuberculous mycobacteria (NTM) following teclistamab to date.

NTM are a heterogenous group of >190 opportunistic mycobacteria other than *Mycobacterium tuberculosis* and *Mycobacterium leprae*.[7] NTM infections are hard to diagnose due to vague symptoms, slow bacterial growth for identification, and challenges differentiating true infection from environmental colonization.[7]

This study was approved by the University of Pennsylvania Institutional Review Board, and informed consent was obtained from all participants. It is the first report of NTM infection following BiTE (including talquetamab) or anti-BCMA immunotherapies (including chimeric antigen receptor T-cell therapy [CAR T] and belantamab) in MM. As of August-2024, our institution has treated 236 patients (60 on clinical trials) with teclistamab. We present three patients with RRMM and one with monoclonal gammopathy-associated thrombotic microangiopathy (MGRS-TMA) who developed NTM infections after teclistamab (Table 1). All patients received routine prophylaxis with trimethoprim-sulfamethoxazole, acyclovir, and monthly intravenous immunoglobulin. The infections were caused by *Mycobacterium abscessus (M.abscessus)* (case 1), *Mycobacterium avium intracellulare (MAI)* (cases 2 and 3), and *Mycobacterium kansasii (M.kansasii)* (case 4). The median time to NTM infection was 127.5 (range, 26-1,322) days from teclistamab initiation, and the median time from presentation to start of anti-mycobacterial therapy was 48.5 (range, 47-99) days. The reasons for delay in therapy were insurance denial for antibiotics (case 1), diagnostic delay due to NTM masquerading as progressive MM (cases 2 and 3), and lag time for culture speciation (case 4).

The first case was a 57-year-old woman with κ-light chain MM who developed a right chest port/skin and soft tissue infection 26 days after starting teclistamab treatment, following completion of three weekly doses. Her chest port was removed and daptomycin started. One week later, catheter tip culture returned positive for *M.abscessus* complex (day-0). She was in an unconfirmed complete remission (uCR) and teclistamab was held. She began omadacycline 300 mg daily, tedizolid 200 mg daily, and cefoxitin 4 g every 8 hours on day-38. Cefoxitin was switched to azithromycin 500 mg daily on day-55 due to nausea and tedizolid was switched to clofazimine 100 mg daily on day-70 once this latter agent became available, completing a 3-month course of multiagent antibiotics (Figure 1). Her MM remained in remission and teclistamab (dosed every other week [EOW]) was restarted on day-83 and discontinued 3 months later as part of a phase II study of limited-duration teclistamab *(clinicaltrials gov. Identifier: NCT05932680)*.

Five months after discontinuation of teclistamab, she developed right knee pain and arthrocentesis was performed. Four days later, she developed right arm pain with draining nodular lesions tracking into her right axilla (Figure 2). Arthrocentesis and wound cultures returned positive for *M.abscessus* complex. Imipenem 1 g every 12 hours, eravacycline 1 mg/kg every 12 hours, azithromycin 500 mg daily, and amikacin were started. She had right knee implant removal and washout, with a plan to complete a 3-month course of imipenem, omadacycline, azithromycin, and amikacin.

The second case was a 68-year-old man with κ-light chain MM who had diffuse lytic bone lesions at disease onset. He achieved a stringent complete remission (sCR) after two cycles of weekly teclistamab and transitioned to EOW after 6 months. Forty-three months after teclistamab initiation, positron-emission tomography/computed tomography (PET/CT) showed multiple new foci of fluorodeoxyglucose (FDG) uptake within the skeleton, hilar lymph nodes, and multiple sub-centimeter lung nodules (Figure 2). Teclistamab was held. Biopsy of the T9 vertebral lesion was without clonal plasma cells. One month later, biopsy of the left iliac crest lesion showed non-necrotizing granulomatous inflammation with acid-fast bacilli, without clonal plasma cells. Broad-range bacterial polymerase chain reaction from iliac crest biopsy showed *MAI*. Daily azithromycin 500 mg, ethambutol 1,200 mg, and rifabutin 300 mg were started (Figure 1). One month later, he developed fevers, drenching night sweats, and weight loss from nausea, dysgeusia, and anorexia possibly from immune reconstitution inflammatory syndrome against *MAI* in the setting of stopping teclistamab.[8] Azithromycin was reduced to 250 mg daily to mitigate his gastrointestinal symptoms.

Repeat PET/CT after 2 months of *MAI*-directed therapy showed interval increase in FDG uptake within the skeleton. Serum MM markers were undetectable, suggesting non-secretory MM relapse *versus* progressive *MAI* infection. Teclistamab EOW was restarted. Repeat iliac crest and T9–10

Table 1. Clinical characteristics of four patients who developed non-tuberculous mycobacterial infections following teclistamab.

Characteristics	Case 1	Case 2	Case 3	Case 4
Age in years at teclistamab	57	68	69	68
Sex	F	M	F	M
Pertinent past medical history	Right TKA c/b periprosthetic MRSA infection on chronic doxycycline, LTBI s/p INH	N/A	Stage 1 RCC s/p right partial nephrectomy	ITP, CLL, DLBCL s/p alloSCT, EBV-associated smooth muscle tumor s/p resection
Ongoing comorbidities	T2DM, COPD	HTN, severe aortic stenosis, A.fib	Discoid lupus, CART-associated progressive multifocal leukoencephalopathy	Chronic paraspinal seroma, CKD
Teclistamab indication	KLC MM	KLC MM	IgGL extramedullary MM	IgGK MGRS (TMA)
N of lines before teclistamab	6 (Vd, CyBorD, IxaRd, CyKd + ASCT, DPd, CyKd)	6 (RVd + ASCT, CyBorD, VenKd, DPd, TAK573, VD-PACE)	10 (VRd + ASCT, Ixa, DPd, CyKPd, SelinexorPd, Panobinostat + Kd, VD-CE, ide-cel, EloPd, VD-AC)	4 (FCR, RCHOP, BOCE + alloSCT, DaraCyBorD, DRdàD daratumumab maintenance)
Best response to teclistamab	Unconfirmed sCR*	sCR	sCR	Unconfirmed sCR*
Status of MM at time of data cutoff	Ongoing remission 8 months after stopping teclistamab	Relapsed after 4 years and 3 months of teclistamab	Relapsed 14 months after stopping teclistamab	MGRS in remission 5 months after stopping teclistamab
WBC $\times 10^9$/L with differential prior to starting teclistamab	WBC: 7.7; ALC: 1.1; ANC: 5.2	WBC: 6.1; ALC: 1.1; ANC: 3.8	WBC: 4.9; ALC: 0.4; ANC: 4.4	WBC: 7.9; ALC: 0.6; ANC: 7.1 (1 day after teclistamab)
CD4 and CD8 count cells/ μL prior to starting teclistamab	Not available	Not available	CD4: <35; CD8: <45 (3 months prior to teclistamab)	CD4: 226; CD8: 189 (5 months prior to teclistamab)
MM/MGRS status at NTM diagnosis	Remission	Remission	Remission	Remission
WBC $\times 10^9$/L with differential at diagnosis of NTM	1. WBC: 6.8; ALC: 0.7; ANC: 4.4 2. WBC 8.1; ALC: 2.5; ANC: 4 3. WBC: 10.2; ALC: 1.1, ANC: 8.1	WBC: 5.9; ALC: 1.1; ANC: 5	WBC: 4.2; ALC: 0.2; ANC: 3.3	WBC: 8.8; ALC: 1.2; ANC: 6.1
CD4 and CD8 count cells/ μL at diagnosis of NTM	1. Not available 2. CD4: 340; CD8: 1778 3. Not available	Not available	CD4: 46; CD8: <45	CD4: 204; CD8: 262
Ig levels mg/dL at diagnosis of NTM	1. IgG: 349; IgA <2; IgM <10 2. IgG: 343; IgA <2; IgM: <5 3. IgG: 494; IgA: <2; IgM: <10	IgG: 685; IgA: 3; IgM: <10	IgG: 502; IgA: <2; IgM: <10	IgG: 488; IgA: <2; IgM: <10
NTM species	*M.abscessus*	*MAI*	*MAI*	*M.kansasii*
NTM presenting S/S	1. Chest port erythema 2. R prosthetic knee pain 3. R arm ulcers	Night sweats	Night sweats, anorexia, lymphadenopathy	Enlarging paraspinal abscess
Site of NTM infection	1. Port, SSTI 2. Prosthetic joint infection 3. SSTI	Bone, lung	Lymphadenitis	1. Superinfection of chronic paraspinal seroma, SSTI 2. Spinal hardware
Timing in days of NTM relative to start of teclistamab	1. 26 (on teclistamab) 2. 407 (off teclistamab) 3. 411 (off teclistasmab)	1,322 (on teclistamab)	153 (on teclistamab)	102 (on teclistamab)

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Characteristics	Case 1	Case 2	Case 3	Case 4
How NTM diagnosed	1. Catheter tip culture 2. Arthrocentesis 3. Skin swab	L iliac bone biopsy	Lymph node biopsy	Abscess biopsy
Time in days between presentation and diagnosis	1. 9 2. 16 3. 12	70	20	33
Time in days between presentation and treatment	1. 47 2. 16 3. 12	99 days	50 days	47 days
Reasons for delay in diagnosis/treatment	Insurance denial of antibiotics	Masquerading as progressive lytic lesions	Masquerading as progressive soft tissue plasmacytomas	Time to *M.kansasii* speciation
Duration in days of NTM therapy	1. 130 2. Ongoing (plan for 3 months) 3. Ongoing (plan for 3 months)	Ongoing (plan for 6 months)	1 year	Ongoing (final duration pending source control); developed surgical wound dehiscence 1 month after surgical debridement; underwent repeat debridement with muscle flap reconstruction

A.fib: atrial fibrillation; ALC: absolute lymphocyte count; alloSCT: allogeneic stem cell transplantation; ANC: absolute neutrophil count; ASCT: autologous stem cell transplantation; c/b: complicated by; BOCE: bendamustine, ofatumumab, carboplatin and etoposide; CAR T: chimeric antigen receptor T cell; CKD: chronic kidney disease; CLL: chronic lymphocytic leukemia; COPD: chronic obstructive pulmonary disease; CyBorD: cyclophosphamide + bortezomib + revlimid + dexamethasone; CyKd: cyclophosphamide + kyprolis + dexamethasone; CyKPd: cyclophosphamide + kyprolis + pomalidomide + dexamethasone; DaraCyBorD: daratumumab + cyclophosphamide + bortezomib daratumumab + revlimid + dexamethasone; DPd: daratumumab + pomalidomide + dexamethasone; EBV: Epstein-Barr virus; EloPd: elotuzumab + pomalidomide + dexamethasone; FCR: fludarabine + cyclophosphamide + rituximab; F: female; HTN: hypertension; Ide-cel: idecabtagene-vileucel; INH: isoniazid; IxaRd: ixazomib + revlimid + dexamethasone; DLBCL: diffuse large B-cell lymphoma; ITP: immune thrombocytopenia purpura; Kd: kyprolis + dexamethasone; KLC: k-light chain; MM: multiple myeloma; NTM: non-tuberculous mycobacteria; LTBI: latent tuberculosis infection; Pd: pomalidomide + dexamethasone; M: male; *M.abscessus: mycobacterium abscessus; M.kansasii: mycobacterium kansasii; MAI: mycobacterium avium intracellulare*; MGRS: monoclonal gammopathy of renal significance; MRSA: methicillin-resistant staphylococcus aureus; N/A: not available; R: right; L: left; RCC: renal cell carcinoma; s/p: status post; RCHOP: rituximab + cyclophosphamide + hydroxydaunomycin + oncovin + prednisone; RVd: revlimide + velcade + dexamethasone; sCR: stringent complete response; T2DM: type 2 diabetes mellitus; TKA: total knee arthroplasty; TMA: thrombotic thrombocytopenic purpura; SSTI: skin and soft tissue infections; VenKd: venetoclax + kyprolis + dexamethasone; Vd: velcade + dexamethasone; VD-PACE: velcade + dexamethasone + platinum + adriamycin + cyclophosphamide + etoposide; VGPR: very good partial response/remission; WBC: white blood count. * Repeat bone marrow biopsy not performed.

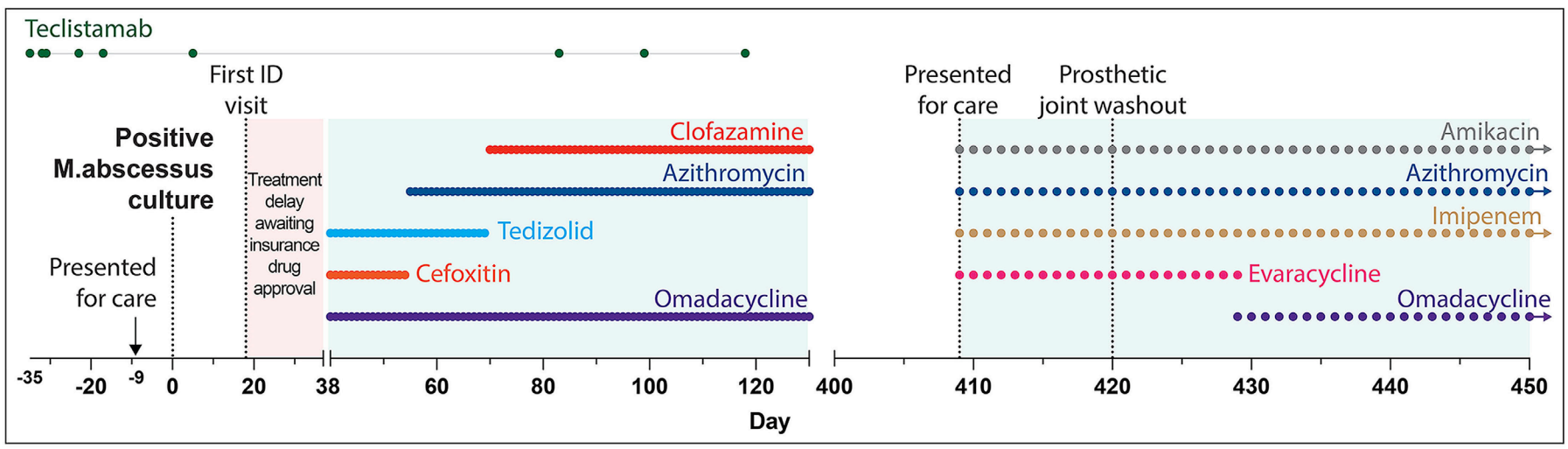
paraspinal mass biopsy showed necrotizing granulomatous inflammation, and cultures confirmed *MAI* susceptibility to amikacin and clarithromycin, without MM. Because it is not routine practice to re-sample non-pulmonary sites for clearance of NTM infection following treatment initiation, it was unclear if the positive bone cultures represented inadequate treatment response after 8 weeks of therapy. Nonetheless, amikacin was added due to worsening symptoms possibly attributable to *MAI* and ongoing immunosuppression from teclistamab. Between restarting teclistamab, dose-reducing azithromycin, and amikacin initiation, his symptoms resolved.

Repeat PET/CT 6 weeks after initiation of amikacin showed new foci of FDG uptake in the left humerus and sternum, with improvement in previously noted lesions. Serum κ-light chain levels had also begun to uptrend. Biopsy of left humerus revealed κ-restricted plasmacytoma consistent with MM recurrence. Bone cultures were negative for *MAI*.

The third case was a 68-year-old woman with IgGλ extramedullary MM with central nervous system involvement and diffuse soft-tissue plasmacytomas. Notably, she had been exposed to idecabtagene vicleucel (ide-cel), which was complicated by biopsy-confirmed progressive multifocal leukoencephalopathy (PML), requiring treatment with steroids and pembrolizumab. She started weekly teclistamab 10 months after ide-cel infusion and achieved sCR after 2 months.

Restaging PET/CT 5 months after teclistamab initiation showed new FDG-avid retroperitoneal and mesenteric lymphadenopathy (Figure 2). Retroperitoneal lymph node biopsy showed non-necrotizing granulomatous inflammation with acid-fast bacilli, without plasmacytoma. Cultures were positive for *MAI*. Teclistamab was discontinued and daily azithromycin 500 mg, ethambutol 800 mg, and rifabutin 300 mg started. One week later, she developed worsening bilateral visual changes. Due to the risk of optic neuritis, ethambutol was discontinued and switched to moxifloxacin 400 mg daily. Moxifloxacin was briefly switched to clofazimine once it became available but restarted after she developed GI side-effects from clofazimine. Magnetic resonance imaging (MRI) brain showed radiographic progression of PML, and she received JC virus-specific T

Case 1



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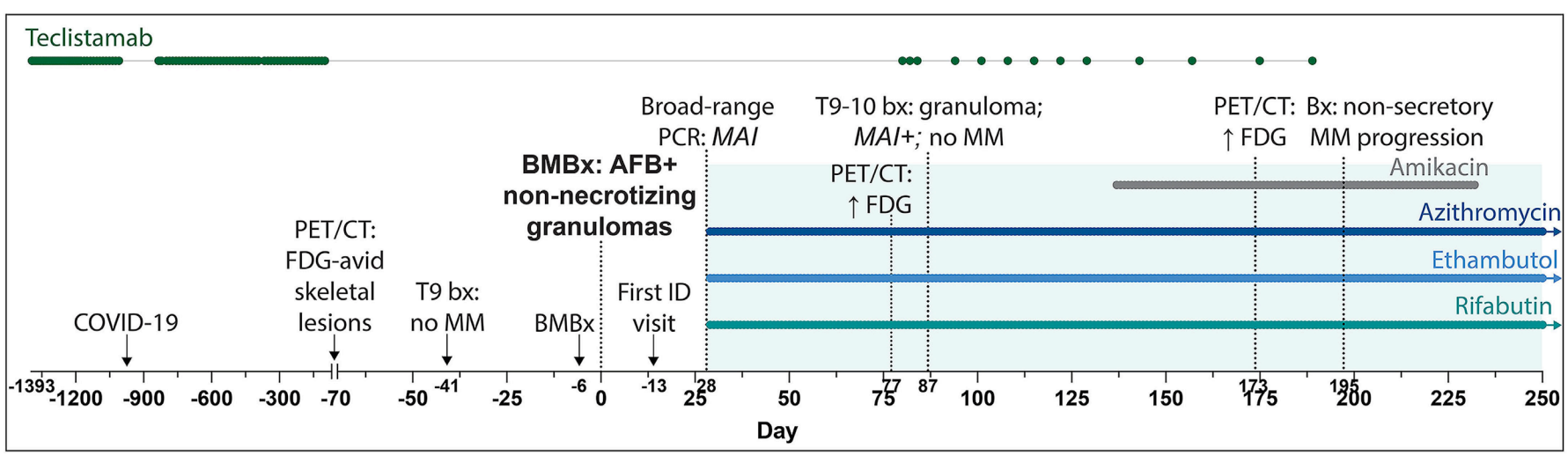

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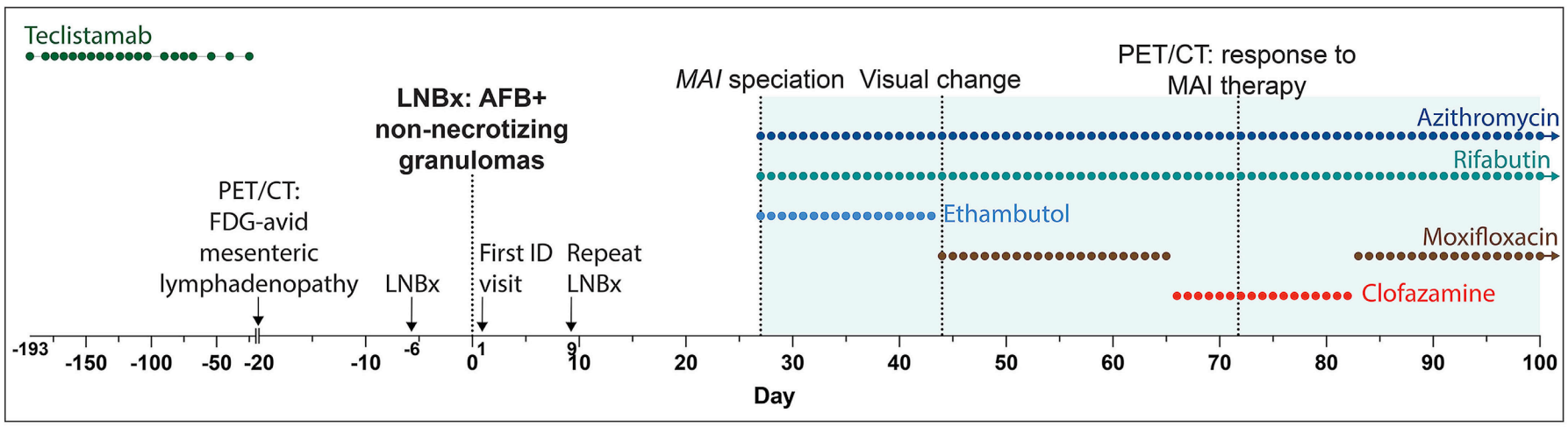

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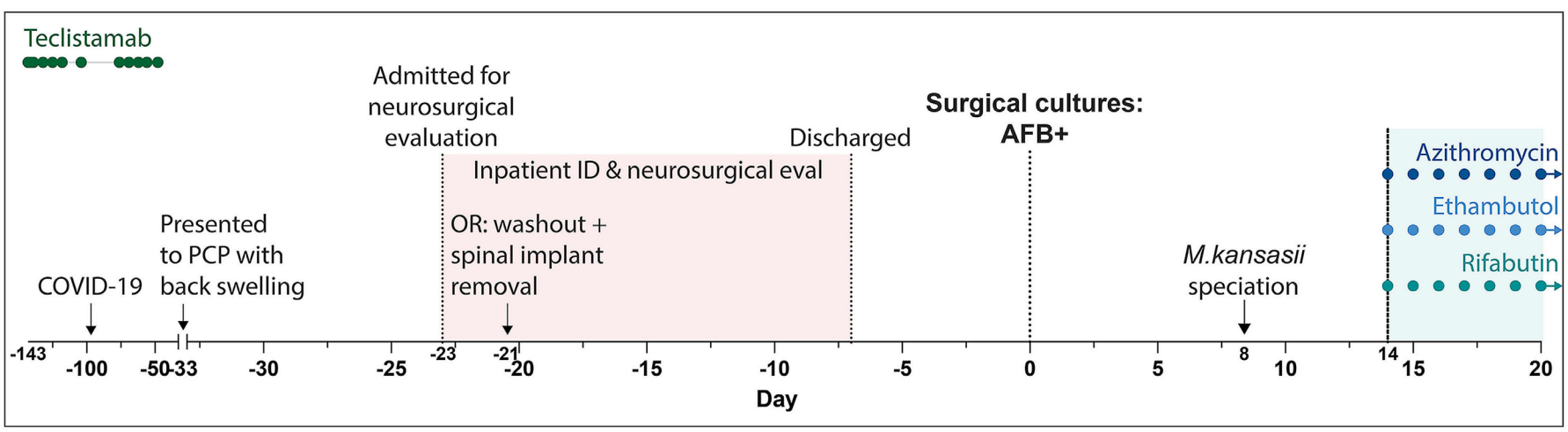

Figure 1. Diagnosis and treatment timeline. Timeline summarizing the clinical course from presentation to initial diagnosis and treatment of non-tuberculous mycobacterial (NTM) infection post-teclistamab. AFB+: acid-fast bacilli positive; BMBx: bone mar-

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row biopsy; Bx: biopsy; Eval: evaluation; FDG: fluorodeoxyglucose; LNBx: lymph node biopsy; *M.abscessus: Mycobacterium abscessus; M.kansasii: Mycobacterium kansasii; MAI: Mycobacterium avium intracellulare;* MM: multiple myeloma; ID: infectious diseases; OR: operating room; PCP: primary care provider; PET/CT: positron emission tomography/computed tomography scan.

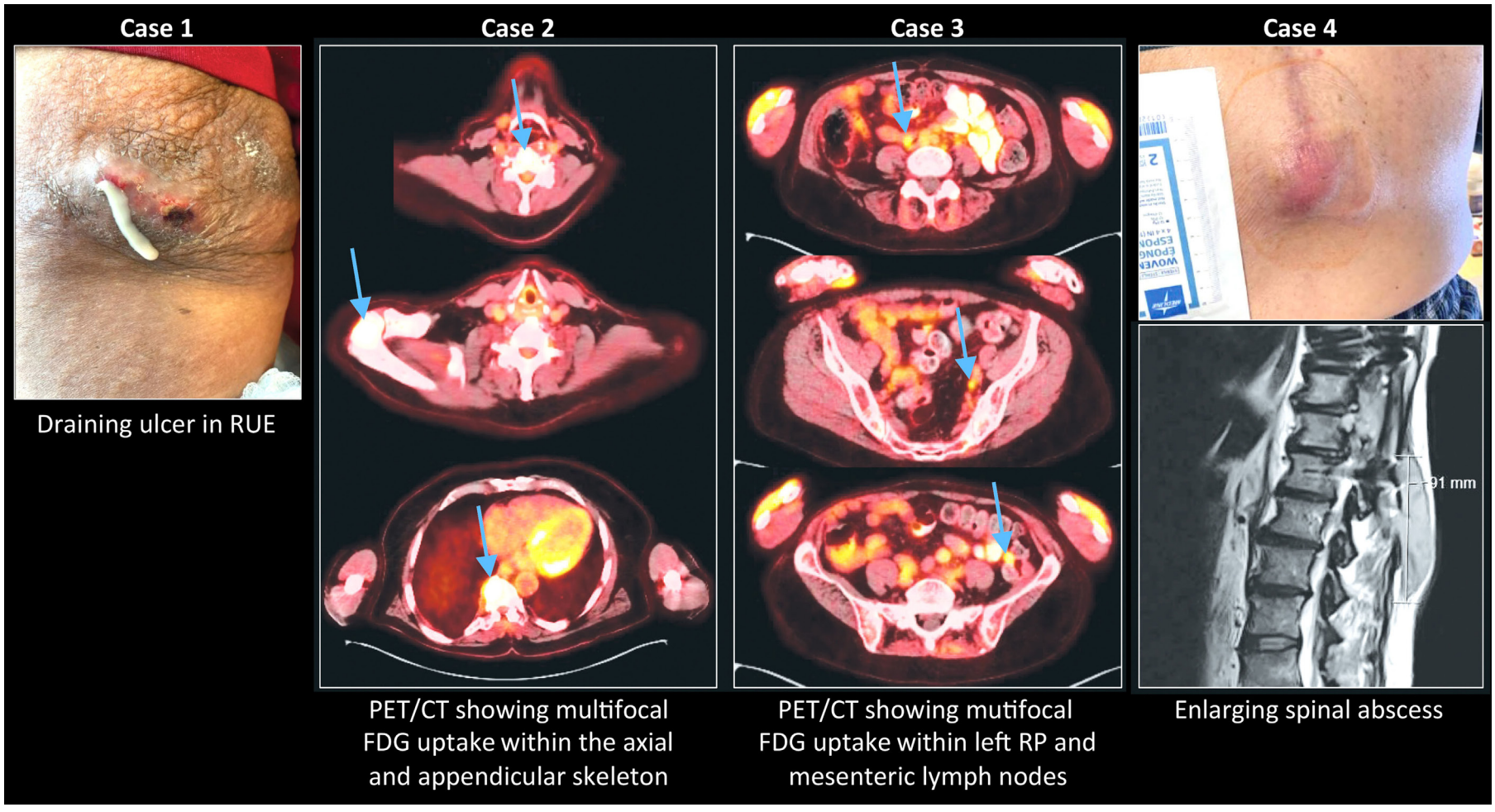

Figure 2. Clinical images. Case 1: draining ulcer in right upper extremity (RUE). Case 2: positron emission tomography/computed tomography scan (PET/CT) showing multifocal fluorodeoxyglucose (FDG) uptake within the axial and appendicular skeleton corresponding to *Mycobacterium avium intracellulare (MAI)* bone infection initially suspicious for progressive lytic lesions. The patient also had FDG uptake in hilar lymph nodes and sub-centimeter pulmonary nodules (not shown). Case 3: PET/CT showing multifocal FDG uptake within left retroperitoneal (RP) and mesenteric lymph nodes corresponding to *MAI* lymphadenitis masquerading as progressive soft-tissue plasmacytomas. Case 4: enlarging thoracic spinal abscess with corresponding magnetic resonance imaging.

cells on a clinical trial. Two months after initiation of *MAI* therapy, PET/CT showed decreasing lymphadenopathy and mesenteric lesions consistent with response.

The fourth case was a 68-year-old man with IgGκ MGRS-TMA who developed *M.kansasii* spinal implant infection. He had chronic lymphocytic leukemia complicated by Richter's transformation to Epstein-Barr virus-positive (EBV^+) Hodgkin-like lymphoproliferative disease treated with haploidentical myeloablative allogeneic stem cell transplant (allo-SCT) in 2012. He developed T9-12 EBV-associated smooth muscle tumor 5 years post-transplant and underwent radiation and surgical resection, which was complicated by chronic paraspinal seroma.

Weekly teclistamab was started in December 2023, and he achieved uCR after 2 months. He developed an enlarging mid-back bulge beneath his incisional scar 3.5 months after teclistamab initiation. Spine MRI showed stable fluid collection in the right epidural/paraspinal space at T9-12 and new abscesses in the subcutaneous dorsal soft tissues at T12-L2 and right paraspinal musculature at T7-8 with involvement of spinal implant (Figure 2). Teclistamab was held. Daptomycin and cefepime were given and he underwent washout of the abscesses and spinal hardware removal. Surgical pathology showed extensive necrotic tissue and cultures grew *M.kansasii*. Daptomycin and cefepime were discontinued and he started azithromycin 500 mg daily, ethambutol 1,000 mg three-times weekly, and rifabutin 300 mg daily (Figure 1).

Although immunosuppression is an established risk factor for NTM infections, there are no published reports of NTM infections following BiTE in MM to date. Teclistamab is associated with an increased infection risk, compared to conventional MM therapies[9] for several reasons: i) a highly immunosuppressed heavily pretreated population, ii) on-target, off-tumor depletion of BCMA-expressing non-neoplastic B cells and plasma cells, and iii) neutropenia from cytokine-mediated impairment of hematopoiesis.[1]

NTM are eliminated by T cells and therefore associated with T-cell exhaustion,[10] which can be induced by chronic activation from BiTE like teclistamab.[11] The patients in this

series had several additional risk factors which may have contributed to these infections, including prior SCT in all, indwelling hardware in two, and prior CAR T cells in one. NTM infections are reported in 0.4-10% of patients with hematologic cancers and are 50-600 times more common in SCT recipients compared to the general population.[12,13] The most frequent organisms are *MAI, M.abscessus-chelonae*, and *Mycobacterium haemophilum*, and the most common sites of infection in this population are catheter (40%), skin (30%), pleuropulmonary (20%).[13]

Two recent studies have highlighted NTM infection risks in oncologic patients: a nationwide retrospective study in Japan revealed high infection rates among specific groups of pediatric hematology/oncology patients,[12] while a case report documented NTM infections as a complication following bi-specific CD3/CD20 antibody therapy in patients with non-Hodgkin lymphoma.[14] Similarly, our report highlights the risk of NTM infections in teclistamab-treated patients and the challenges of diagnosing NTM infections in the context of non-specific clinical features and mimicry of the underlying disease. At the time of NTM infection, patients 2 and 3 had undetectable serum MM markers and FDG-avidity on PET/CT. However, discordant PET/CT findings can also be due to non-secretory relapse, which can be seen in about 2.4% of patients with secretory disease at diagnosis.[15] Typically, biopsies are needed to confirm non-secretory disease progression, but infectious testing is often overlooked, delaying diagnoses. Our findings support early NTM testing in teclistamab-treated patients with non-specific symptoms or atypical progression to reduce diagnostic delays and highlight the importance of coordination with surgical pathology and infectious diseases to ensure appropriate samples are sent for relevant microbiological testing whenever biopsies are planned.

Upon diagnosis of NTM infection, we recommend holding teclistamab and consulting infectious diseases for prompt initiation of empiric/susceptibility-guided multiagent antibiotic therapy. The decision to restart teclistamab should be individualized depending on severity of NTM infection, source control, MM response, and availability of anti-MM therapies.

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Contributions

SSA and SL provided detailed patients data, conceptualized the project, and edited the manuscript. MH collected the clinical data and drafted the manuscript. LP, JM, TV, DTV, ADC, ALG, AJW, SK, and EAS provided detailed patients data, feedback, and edited the manuscript. PD reviewed pathology slides and provided feedback.

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Data-sharing statement

Additional clinical data, if deemed relevant to the study, may be available on request from the corresponding author.

References

1. Nooka AK, Rodriguez C, Mateos MV, et al. Incidence, timing, and management of infections in patients receiving teclistamab for the treatment of relapsed/refractory multiple myeloma in the MajesTEC-1 study. Cancer. 2024;130(6):886-900.
2. Mazahreh F, Mazahreh L, Schinke C, et al. Risk of infections associated with the use of bispecific antibodies in multiple

myeloma: a pooled analysis. Blood Advances. 2023;7(13):3069-3074.
3. Reynolds G, Cliff ERS, Mohyuddin GR, et al. Infections following bispecific antibodies in myeloma: a systematic review and meta-analysis. Blood Adv. 2023;7(19):5898-5903.
4. Jourdes A, Cellerin E, Touzeau C, et al. Characteristics and incidence of infections in patients with multiple myeloma treated by bispecific antibodies: a national retrospective study. Clin Microbiol Infect. 2024;30(6):764-771.
5. Mohan M, Nagavally S, Dhakal B, et al. Risk of infections with B-cell maturation antigen-directed immunotherapy in multiple myeloma. Blood Advances. 2022;6(8):2466-2470.
6. Hammons L, Szabo A, Janardan A, et al. The changing spectrum of infection with BCMA and GPRC5D targeting bispecific antibody (bsAb) therapy in patients with relapsed refractory multiple myeloma. Haematologica. 2024;109(3):906-914.
7. Pennington KM, Vu A, Challener D, et al. Approach to the diagnosis and treatment of non-tuberculous mycobacterial disease. J Clin Tuberc Other Mycobact Dis. 2021;24:100244.
8. Lemoine M, Laurent C, Hanoy M, et al. Immune reconstitution inflammatory syndrome secondary to Mycobacterium kansasii infection in a kidney transplant recipient. Am J Transplant. 2015;15(12):3255-3258.
9. Raje N, Anderson K, Einsele H, et al. Monitoring, prophylaxis, and treatment of infections in patients with MM receiving bispecific antibody therapy: consensus recommendations from an expert panel. Blood Cancer J. 2023;13(1):116.
10. Lombardi A, Villa S, Castelli V, Bandera A, Gori A. T-cell exhaustion in Mycobacterium tuberculosis and nontuberculous mycobacteria infection: pathophysiology and therapeutic perspectives. Microorganisms. 2021;9(12):2460.
11. Meermeier EW, Welsh SJ, Sharik ME, et al. Tumor burden limits bispecific antibody efficacy through T cell exhaustion averted by concurrent cytotoxic therapy. Blood Cancer Discov. 2021;2(4):354-369.
12. Tsumura Y, Muramatsu H, Tetsuka N, et al. A Japanese retrospective study of non-tuberculous mycobacterial infection in children, adolescents, and young adult patients with hematologic-oncologic diseases. Haematologica. 2024;109(9):2988-2997.
13. Bergeron A, Mikulska M, De Greef J, et al. Mycobacterial infections in adults with haematological malignancies and haematopoietic stem cell transplants: guidelines from the 8th European Conference on Infections in Leukaemia. The Lancet Infectious Diseases. 2022;22(12):e359-e369.
14. Little JS, Hurtado RM, Boire N, et al. Disseminated Mycobacterium avium complex infection following CD3/CD20 bispecific antibody therapy in a patient with follicular lymphoma. Open Forum Infectious Diseases. 2024;11(9):ofae460.
15. Zamir I, Shragai T, Trestman S, et al. Multiple myeloma oligosecretory relapse, a non-negligible phenomenon. Frequency, clinical characteristics and outcomes in a single center. Blood. 2021;138(Suppl 1):3772.