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Parkinsonism-like delayed neurotoxicity after idecabtagene vicleucel therapy for multiple myeloma: clinicopathological, cerebrospinal fluid, and autopsy analyses

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Running heads: Delayed neurotoxicity post-BCMA CAR-T cell therapy

Data-sharing statement: Data are available on request from the corresponding author, Hiroyuki Takamatsu.

Contributions

T.I., H.T., and T.M. wrote the manuscript; H.Y., T.I., S.Y., S.N., D.H., S.S., S.F., A.H., T.H., T.Y., Y.Z., A.Y., H.M., T.U., H.T. collected the clinical data and blood samples; Y.N., K.H., and A.Y. analyzed the flow cytometry results; S.W., Y.I., M.H., and H.T. performed the immunopathological study; M.T. and K.M. performed the enzyme-linked immunosorbent assay.

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Chimeric antigen receptor T (CAR-T) cell therapy targeting B-cell maturation antigen (BCMA) has transformed the treatment landscape for relapsed or refractory multiple myeloma. Idecabtagene vicleucel (ide-cel)[1] and ciltacabtagene autoleucel (cilta-cel)[2] have demonstrated notable efficacy in heavily pretreated patients, leading to widespread clinical adoption. Nevertheless, CAR-T cell therapy is associated with unique immune-mediated toxicities, the most common being cytokine release syndrome (CRS) and immune effector cell–associated neurotoxicity syndrome (ICANS).[3] ICANS typically develops within days to weeks after CAR-T cell infusion and is characterized by encephalopathy, tremors, aphasia, and impaired consciousness. Contrastingly, delayed neurological toxicities occurring weeks to months after CAR-T cell infusion have been recognized recently and categorized as movement and neurocognitive treatment-emergent adverse events (MNTs).[4-6] MNTs comprise a spectrum of neurological manifestations, including movement disorders, cognitive decline, and behavioral or personality changes, which may progress irreversibly or cause death. Parkinsonism-like MNTs have been reported predominantly after BCMA-directed CAR-T cell therapy, occurring in approximately 5% of patients treated with cilta-cel[7] and in < 1% of those treated with ide-cel.[5, 6] These observations have raised concerns regarding potential on-target, off-tumor effects in the basal ganglia,where BCMA expression has been suggested.[4-6] A recent report demonstrated that BCMA expression in the brain is not specific, given the characteristics of anti-BCMA antibodies.[8] Here, we report the case of a patient who developed Parkinsonism-like MNTs after ide-cel therapy. This case is notable for the longitudinal evaluation of CAR-T cells and soluble BCMA (sBCMA) in the serum and cerebrospinal fluid (CSF), and detailed autopsy findings. Ethical approval was not required for this case report according to Japanese ethical guidelines. Written informed consent was obtained from the patient's family.

A 58-year-old Japanese man was diagnosed in September 2023 with IgGκ-type multiple myeloma, classified as International Staging System (ISS) stage III and Revised ISS stage III. The patient had no significant medical history or known allergies. The patient’s father has a family history of lung cancer, and no one in his family has a neurological disorder. The patient presented with anemia,

diffuse osteolytic bone lesions, renal dysfunction, hypercalcemia, and hyperviscosity syndrome. Serum IgG level was markedly elevated (10,393 mg/dL). Fluorescence in situ hybridization analysis of bone marrow (BM) plasma cells revealed high-risk cytogenetic abnormalities, including t(4;14) and amplification of chromosome 1q. Initial treatment with high-dose dexamethasone, followed by bortezomib-based regimens combined with cyclophosphamide or daratumumab, failed to achieve disease control.

In November 2023, therapy was switched to carfilzomib+lenalidomide+dexamethasone therapy, resulting in a transient reduction in serum M-protein. BM examination during the carfilzomib+lenalidomide+dexamethasone therapy demonstrated minimal residual disease negativity by multicolor flow cytometry (cutoff value: 10^{-5}),[9] which has comparable sensitivity as EuroFlow next-generation flow. Despite this finding, the extramedullary disease progressed with increasing soft tissue involvement and pleural effusion containing myeloma cells, causing worsening lumbar pain and progressive functional decline. By December 2023, the disease had become clinically aggressive, with re-elevation of serum M-protein and κ free light chain levels and severe pain limiting mobility.

Considering the triple-class refractory disease, ide-cel therapy was selected. Bridging therapy with one cycle of dexamethasone+cisplatin+doxorubicin+cyclophosphamide+etoposide (D-PACE)[10] chemotherapy was administered, followed by leukapheresis for CAR-T cell manufacturing in February 2024. After an additional D-PACE cycle, lymphodepletion with fludarabine and cyclophosphamide was performed, and ide-cel was infused in April 2024 (Figure 1). On day 7 after the ide-cel infusion, the patient developed a high fever consistent with grade 1 CRS. The fever persisted; hence, tocilizumab was administered on day 10, causing rapid defervescence. Fluorodeoxyglucose-positron emission tomography/computed tomography (FDG-PET/CT) performed on day 34 demonstrated a complete absence of fluorodeoxyglucose uptake in the extramedullary lesions, indicating a complete metabolic response. Throughout the early post-infusion period, the immune effector cell encephalopathy score[11] remained at 10, and no clinical

features of ICANS were observed before or after CRS resolution. There was no significant lymphocytosis after ide-cel, and the peak absolute lymphocyte count was 2.04 × 10^9/L on day 12. Approximately 25 days after infusion, the patient developed progressive bradykinesia, masked facial expressions, and a short-stepped gait. The symptoms gradually worsened, prompting readmission on day 40. Brain computed tomography and magnetic resonance imaging (MRI) revealed no structural abnormalities, such as infarction, hemorrhage, or demyelination. CSF analysis revealed no pleocytosis or biochemical evidence of meningitis. Viral polymerase chain reaction testing for cytomegalovirus, human herpesvirus 6, Epstein–Barr virus, herpes simplex virus 1/2, and varicella-zoster virus yielded negative results. Although the total CSF lymphocyte count was low (2 cells/μL), flow cytometric analysis revealed that 61.5% of CSF lymphocytes were CAR-T cells (Figure 1), and 76% of the CAR-T cells in the CSF were CD4-positive. Dopamine transporter scintigraphy demonstrated a mild, non-significant reduction in uptake in the left striatum. Based on the clinical presentation and the exclusion of alternative etiologies, the patient was diagnosed with ide-cel-associated MNTs.

The Movement Disorder Society-Unified Parkinson's Disease Rating Scale (MDS-UPDRS) part III score was 56, indicating moderate-to-severe motor impairment. Oral dexamethasone was initiated at a dose of 2–10 mg/day for 2 months. Given the persistence of the symptoms, four intrathecal injections of dexamethasone (3.3 mg each) were administered. Following treatment, the proportion of CAR-T cells in the CSF decreased (61.5–19.2%). Clinically, the masked facies and bradykinesia were partially ameliorated, and the MDS-UPDRS part III score decreased (56–49). The patient was discharged on day 93.

On day 119, the patient was readmitted due to rapidly progressive myeloma. Laboratory testing revealed elevated lactate dehydrogenase levels; FDG-PET/CT and whole-body MRI demonstrated widespread disease involvement. BM examination confirmed relapse, and isatuximab+pomalidomide+dexamethasone therapy was initiated on day 120. On the same day, the patient developed a high fever, which transiently responded to dexamethasone. On day 122, the C-

reactive protein level increased to 24.48 mg/dL, and blood cultures grew *Pseudomonas aeruginosa* and *Streptococcus viridans*. Despite intensive treatment with cefepime and norepinephrine, the patient developed refractory septic shock and died on day 124 (Figure 1).

Autopsy revealed extensive myeloma infiltration involving the BM, subcutaneous tissue, liver, kidneys, thyroid gland, lungs, and pleura. No intracranial mass lesions were observed. Histopathological examination of the brain showed minimal lymphocytic infiltration in the cerebral cortex, substantia nigra, striatum, caudate nucleus, and globus pallidus, without overt neuronal loss or structural damage to the white or gray matter (Figure 2A-D). Contrastingly, focal clusters of CD3-, CD4-, and CD8-positive lymphocytes were observed in the basal ganglia (Figure 2E-G). Immunohistochemical staining using two independent anti-BCMA antibodies demonstrated BCMA expression in the caudate nucleus (Figure 2H, cat. no. B0807-50 [rabbit IgG polyclonal antibody], USBiological, Salem, MA; Figure 2I, E6D7B, cat. no. 84031 [rabbit IgG monoclonal antibody], Cell Signaling Technology, Danvers, MA), whereas the cerebral cortex and cerebellum were negative for BCMA (Figure 2K-L). Serum sBCMA levels measured by enzyme-linked immunosorbent assay (Human BCMA/TNFRSF17 DuoSet ELISA DY193; R&D Systems, Minneapolis, MN, USA)[12] were 37.08 on day 91 and 5.31 ng/mL at the time of autopsy. CSF sBCMA levels were below the detection limit on day 91 but slightly increased to 0.103 ng/mL at the time of autopsy. Immunohistochemical staining of the BM demonstrated BCMA positivity at diagnosis (Figure 3A) but loss of BCMA expression at autopsy despite persistent CD138 and Igκ positivity (Figure 3B–E). These findings were consistent with the sBCMA results.

This case provides rare clinicopathological insights into Parkinsonism-like delayed neurotoxicity following ide-cel therapy. Despite the absence of overt ICANS, MNTs developed approximately 1 month after CAR-T cell infusion. Known risk factors, including high tumor burden, extramedullary disease, CRS, and early CAR-T cell expansion[3, 7] were present and may have contributed to sustained immune activation near the blood–brain barrier. Treatments for MNTs, including steroid pulse therapy, anti-IL-1 antibody, and plasma exchange, have been reported[7, 13],

however, their therapeutic effects are limited.[7, 14, 15]

Although BCMA immunoreactivity was detected in the caudate nucleus, no irreversible neuronal damage was identified, consistent with the partial reversibility of the symptoms after corticosteroid therapy. The detection of CAR-T cells and sBCMA in the CSF suggests CAR-T cell trafficking into the central nervous system or microscopic tumor involvement, particularly in the context of aggressive extramedullary disease. Importantly, the proportion of CAR-T cells in the CSF increased at the onset of MNTs and decreased following treatment, suggesting that CSF CAR-T cell monitoring may serve as a useful biomarker for early diagnosis and therapeutic decision-making. Inter-individual variability in BCMA expression within the basal ganglia (Figure 2J) may further explain the rarity of this complication. In general, higher BCMA mRNA expression levels were observed in younger individuals in their 20s, with a tendency to decline with age. However, rare cases of high expression (normalized Transcripts Per Million [nTPM] up to approximately 2.5) have also been identified in elderly individuals in their 60s (Figure 2J).

Consistent with previous reports on Parkinsonism-like NMTs, the cause of death was severe infection,[4-7] highlighting the profound immunosuppression associated with prolonged corticosteroid therapy. Careful infection surveillance and prevention are essential.

This report describes the first Asian case of Parkinsonism-like delayed neurotoxicity after ide-cel therapy, supported by CSF analyses and autopsy findings. Accumulation of additional cases is critical to elucidate the underlying mechanisms and establish optimal management strategies for this rare but serious complication.

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Figure Legends

Figure 1. Clinical course and dynamics of CAR-T cell ratio in peripheral blood and CSF.

Changes in extramedullary lesions observed by whole-body diffusion-weighted imaging (WBDWI) and positron emission tomography–computed tomography (PET-CT) before and after CAR-T cell therapy, as well as the timing of systemic and intrathecal corticosteroid therapy for cytokine release syndrome (CRS) and movement and neurocognitive treatment-emergent adverse events (MNTs), are shown. On dopamine transporter scintigraphy (DaT scan), decreased uptake in the left putamen showed slight improvement after corticosteroid therapy for MNTs. Peripheral blood or cryopreserved mononuclear cells and cerebrospinal fluid (CSF) cells were stained at 4°C with a panel of surface antibodies, and the proportion of CAR-T cells among CD3-positive T lymphocytes was determined using a FACS Canto flow cytometer.

Abbreviations: WBDWI, whole-body diffusion-weighted imaging; DaT scan, dopamine transporter scintigraphy; CRS, cytokine release syndrome; MNTs, movement and neurocognitive treatment-emergent adverse events; Ide-cel, idecabtagene vicleucel; DEX, dexamethasone; IsaPD, isatuximab, pomalidomide, and dexamethasone; CSF, cerebrospinal fluid; CAR-T cell, chimeric antigen receptor T cell; ALC, absolute lymphocyte count; PET, positron emission tomography; CT, computed tomography; IT, intrathecal therapy; PB, peripheral blood.

Figure 2. Macroscopic and microscopic findings in the basal ganglia and immunohistochemical findings in the striatum at craniotomy.

A: Macroscopic findings of the basal ganglia. The area enclosed by the rectangle corresponds to the caudate nucleus. R and L indicate the right and left sides, respectively. Hematoxylin and eosin (H&E) and Klüver–Barrera (KB) staining were performed to evaluate pathological changes in the brain parenchyma and white matter. To assess BCMA expression in central nervous system tissues, immunohistochemical staining was performed using two anti-BCMA antibodies: a rabbit polyclonal antibody (clone B0807-50, USBiological, Salem, MA) and a rabbit monoclonal

antibody (clone E6D7B, Cell Signaling Technology, Danvers, MA; Cat. No. 88183). B and C: H&E staining of the caudate nucleus and putamen, which together constitute the striatum. D: KB staining of the caudate nucleus. No significant pathological changes in the neural structures were observed. E–G: Minimal infiltration and small clusters of CD3-, CD4-, and CD8-positive T lymphocytes were observed in the basal ganglia. H and I: Immunohistochemical staining of the caudate nucleus using anti-BCMA antibodies demonstrated positive BCMA expression. J: BCMA mRNA expression levels in the basal ganglia obtained from The Human Protein Atlas (https://www.proteinatlas.org/ENSG00000048462TNFRSF17/brain/basal+ganglia#gtex_rnaseqbasal_ganglia). K and L: Immunohistochemical staining of the cerebral cortex and cerebellum, respectively, using the anti-BCMA antibody (clone E6D7B).

Abbreviations: BCMA, B-cell maturation antigen; H&E, hematoxylin and eosin; KB, Klüver–Barrera; nTPM, normalized transcripts per million.

Figure 3. Immunohistochemical findings in the bone marrow (BM) at diagnosis and autopsy. To assess BCMA expression in BM cells, immunohistochemical staining was performed using a rabbit monoclonal anti-BCMA antibody (clone E6D7B). **A** and **B:** BCMA expression in BM specimens obtained at diagnosis and at autopsy, respectively. **C–E:** Immunohistochemical staining for CD138, Igκ, and Igλ in the autopsy BM.

Figure 1

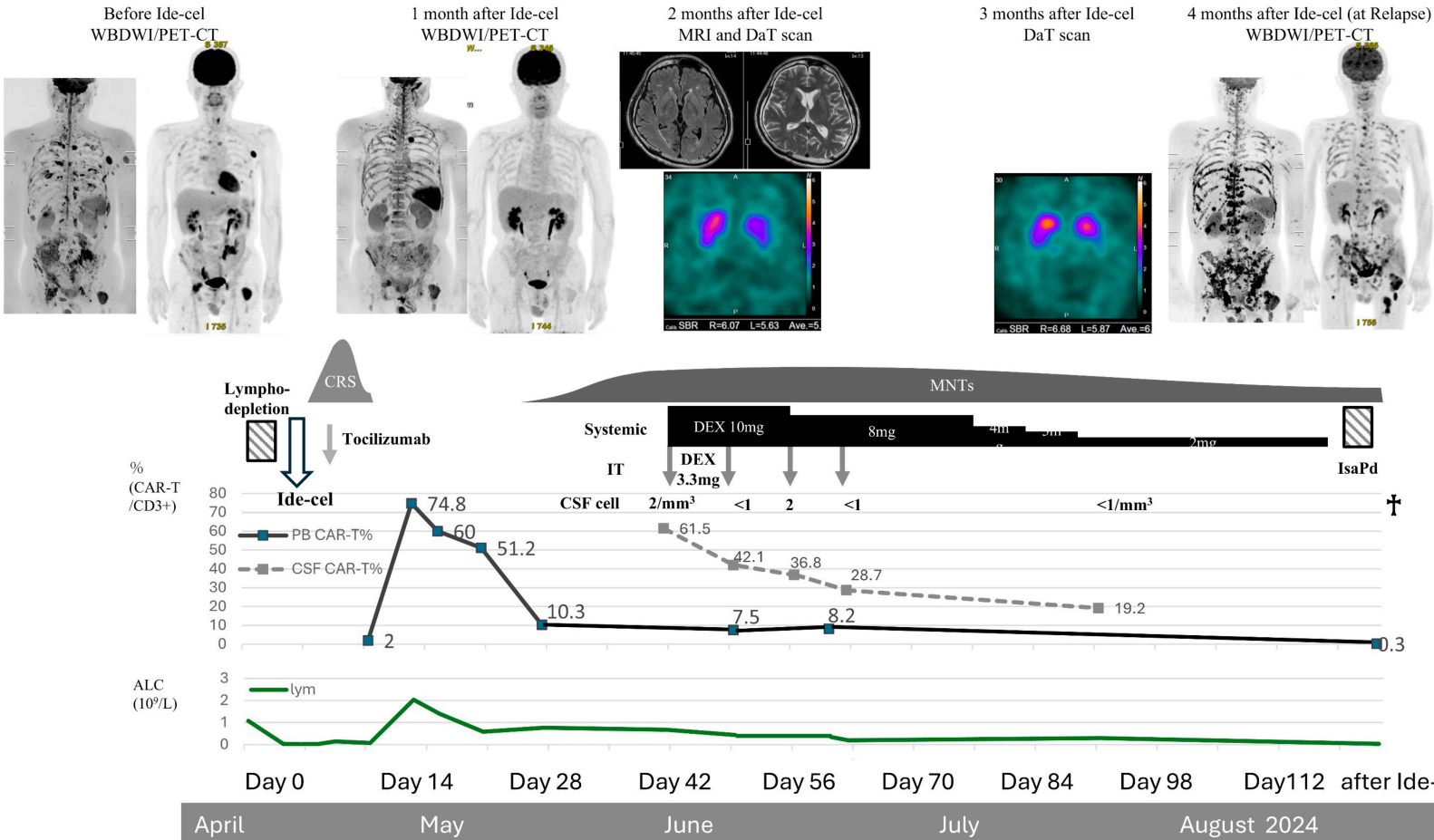
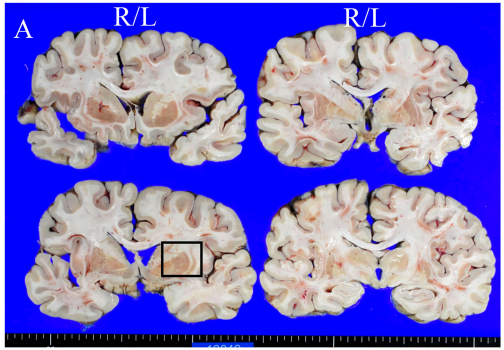
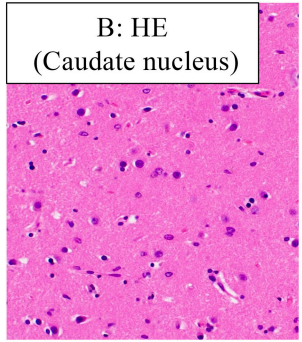
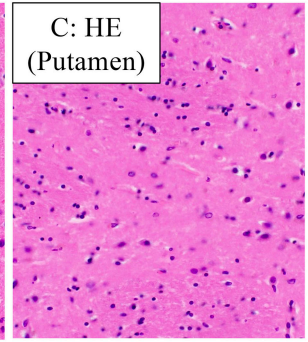
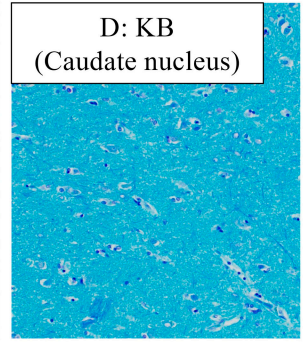

Before Ide-cel
WBDWI/PET-CT
1 month after Ide-cel
WBDWI/PET-CT
2 months after Ide-cel
MRI and DaT scan
3 months after Ide-cel
DaT scan
4 months after Ide-cel (at Relapse)
WBDWI/PET-CT
SBR R=6.07 L=5.63 Ave.=5
SBR R=6.68 L=5.87 Ave.=6
Lympho-depletion
CRS
Tocilizumab
Ide-cel
MNTs
Systemic
DEX 10mg
8mg
2mg
IT
DEX 3.3mg
IsaPd
CSF cell
2/mm³
<1
2
<1
<1/mm³
%
(CAR-T /CD3+)
PB CAR-T%
CSF CAR-T%
74.8
60
51.2
10.3
2
7.5
8.2
0.3
61.5
42.1
36.8
28.7
19.2
ALC
(10⁹/L)
lym
Day 0
Day 14
Day 28
Day 42
Day 56
Day 70
Day 84
Day 98
Day112
after Ide-cel
April
May
June
July
August 2024

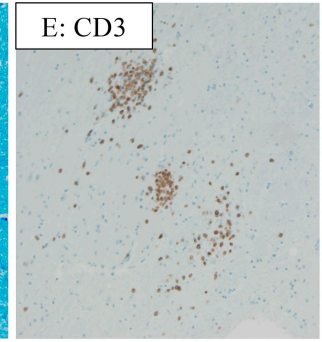
Figure 2

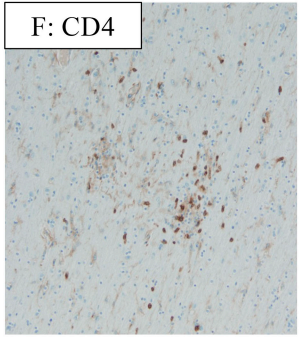

A
R/L
R/L

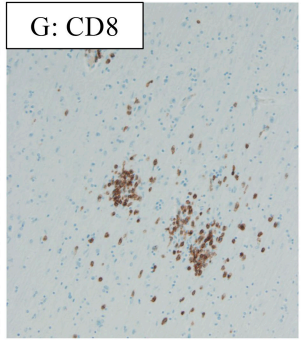

B: HE
(Caudate nucleus)

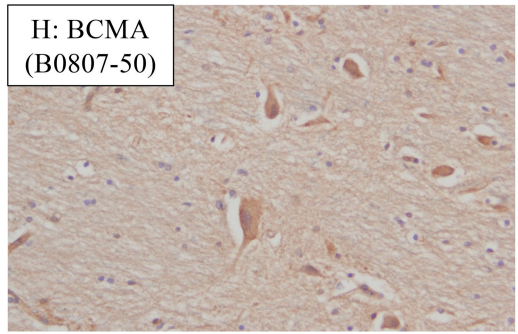

C: HE
(Putamen)

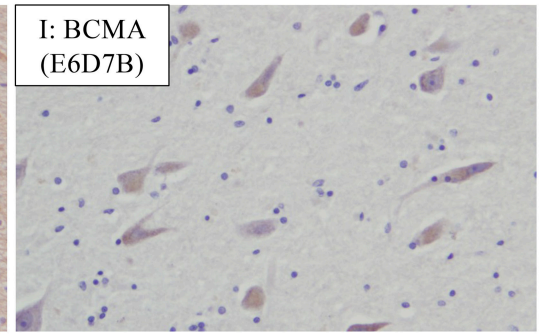

D: KB
(Caudate nucleus)

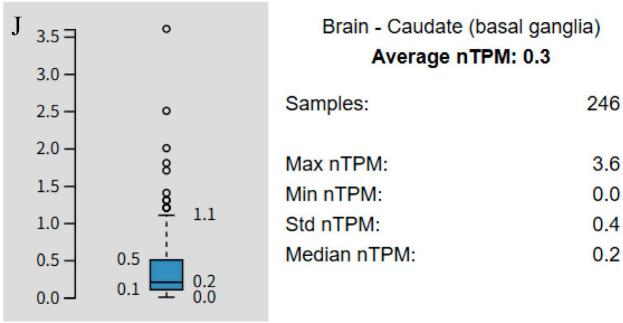

E: CD3

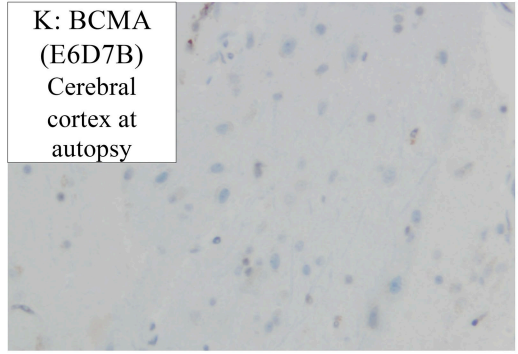

F: CD4


G: CD8


H: BCMA
(B0807-50)


I: BCMA
(E6D7B)


J
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
1.1
0.5
0.1
0.2
0.0
Brain - Caudate (basal ganglia)
Average nTPM: 0.3
Samples: 246
Max nTPM: 3.6
Min nTPM: 0.0
Std nTPM: 0.4
Median nTPM: 0.2


K: BCMA
(E6D7B)
Cerebral
cortex at
autopsy

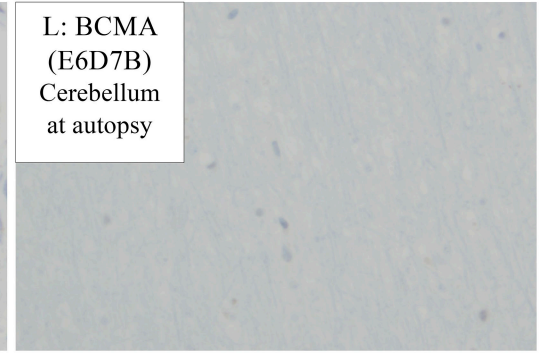
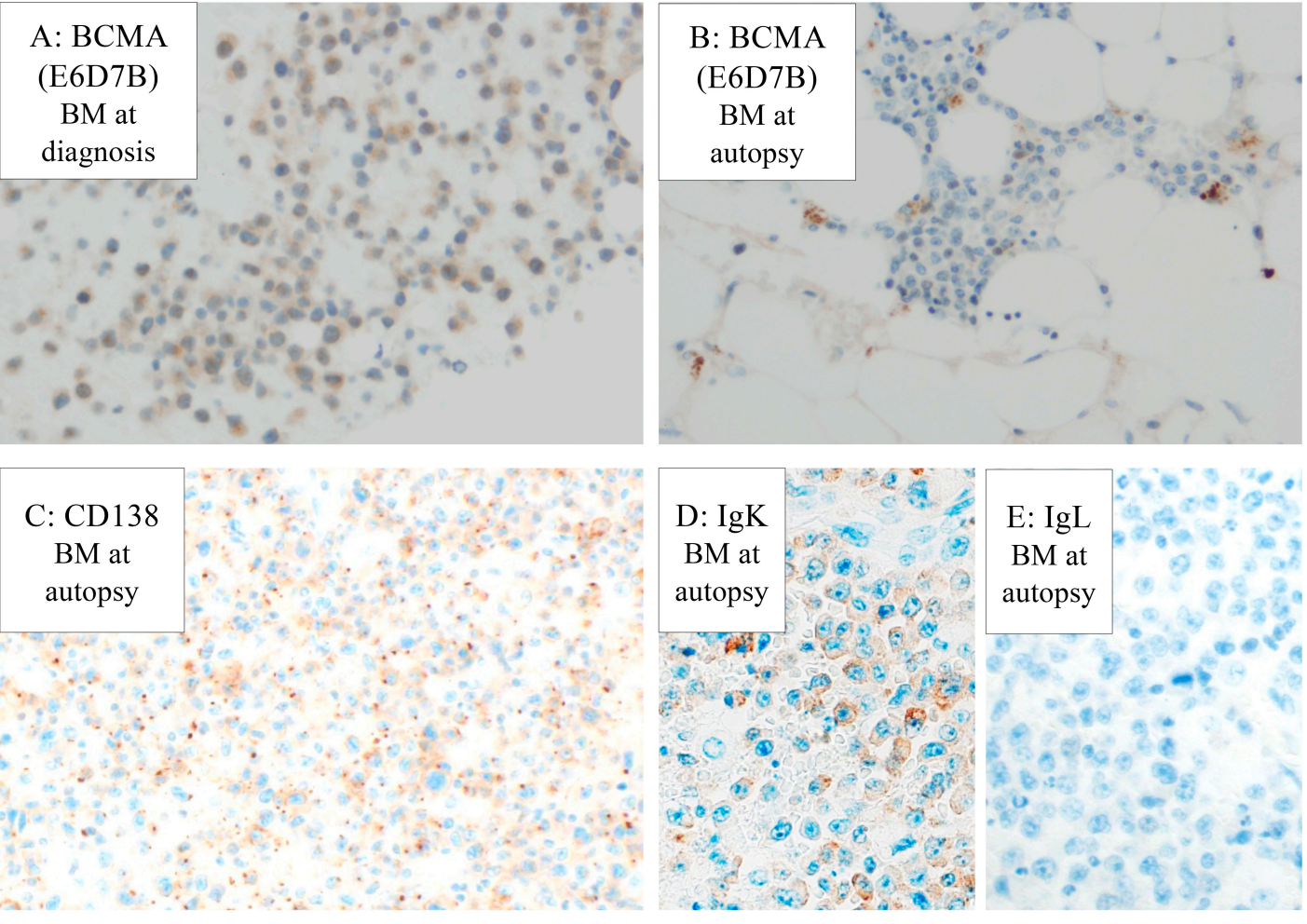

L: BCMA
(E6D7B)
Cerebellum
at autopsy

Figure 3


A: BCMA (E6D7B) BM at diagnosis
B: BCMA (E6D7B) BM at autopsy
C: CD138 BM at autopsy
D: IgK BM at autopsy
E: IgL BM at autopsy