American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e954380
DOI: 10.12659/AJCR.954380

Received: 2026.06.07
Accepted: 2026.08.26
Available online: 2026.09.07
Published: 2026.XX.XX

HHV-6 Encephalitis Presenting With Severe SIADH and CO_2 Narcosis in Non-Transplant Smoldering Adult T-Cell Leukemia/Lymphoma

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDE 1 Kana Tateishi
ABCDEF 1 Toyoshi Yanagihara
BCD 1 Ryoma Kawabata
BCD 1 Eriko Kinoshita
BCD 2 Takeshi Miyazaki
BCDE 2 Yuji Tateishi
BCD 3 Kaori Koga
BCD 3 Makoto Hamasaki
BCDE 1 Masaki Fujita

1 Department of Respiratory Medicine, Fukuoka University Hospital, Fukuoka, Japan
2 Department of Neurology, Fukuoka University Hospital, Fukuoka, Japan
3 Department of Pathology, Fukuoka University Hospital, Fukuoka, Japan

Corresponding Author: Toyoshi Yanagihara, Department of Respiratory Medicine, Fukuoka University Hospital, 7-45-1 Nanakuma, Jonan-ku, Fukuoka 814-0180, Japan, Phone: +81-92-801-1011, e-mail: toyoshi.yana@fukuoka-u.ac.jp

Financial support: None declared
Conflict of interest: None declared

Patient: Female, 55-year-old
Final Diagnosis: HHV-6 encephalitis
Symptoms: Impaired consciousness • nausea
Clinical Procedure: Bronchoscopy • lumbar puncture
Specialty: Hematology • Infectious Diseases • Neurology
Objective: Rare coexistence of disease or pathology

Background: Human herpesvirus 6 (HHV-6) encephalitis is best recognized after allogeneic hematopoietic stem cell transplantation but can also occur in non-transplant immunocompromised patients. Hyponatremia and syndrome of inappropriate antidiuretic hormone secretion (SIADH) may precede or accompany HHV-6 encephalitis. Severe SIADH as an early dominant clue in non-transplant patients with smoldering adult T-cell leukemia/lymphoma (ATLL) is not fully described.

Case Report: A 55-year-old woman with smoldering ATLL received systemic corticosteroid therapy for clinically diagnosed human T-cell leukemia virus type 1 (HTLV-1)-associated uveitis and was admitted for progressive pulmonary infection. Bronchoscopic specimens revealed polymicrobial infection, including *Nocardia* and *Actinomyces*; invasive pulmonary aspergillosis was also clinically suspected. Although pulmonary findings improved with antimicrobial and antifungal therapy, she developed nausea, acute impairment of consciousness, and severe SIADH-associated hyponatremia. Serum sodium decreased to 113 mmol/L, and consciousness did not adequately improve after sodium correction. Cerebrospinal fluid obtained by lumbar puncture on hospital day 13 showed HHV-6 DNA positivity (1.0×10^3 copies/mL). Brain magnetic resonance imaging demonstrated bilateral cortical and limbic abnormalities, supporting a clinical diagnosis of HHV-6 encephalitis. Ganciclovir was initiated on hospital day 15 along with sodium correction, tolvaptan, and supportive care. The patient subsequently developed hypercapnic respiratory failure and CO_2 narcosis requiring mechanical ventilation. Her neurologic and respiratory status gradually improved, and she was discharged on hospital day 53.

Conclusions: In non-transplant patients with ATLL, corticosteroid exposure, and opportunistic infection, severe SIADH accompanied by new neurologic symptoms can be an early clue to HHV-6 encephalitis. Early recognition and prompt initiation of antiviral therapy may help to improve neurologic outcomes.

Keywords: Encephalitis • Hypercapnia • Roseolovirus Infections
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/954380

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Introduction

Human herpesvirus 6 (HHV-6), particularly HHV-6B, is an important cause of acute limbic encephalitis in immunocompromised patients. It is best recognized after allogeneic hematopoietic stem cell transplantation (HSCT) [1-5]. However, HHV-6 encephalitis has also been reported in a non-transplant patient with adult T-cell leukemia/lymphoma before chemotherapy [6]. Adult T-cell leukemia/lymphoma (ATLL) is associated with impaired cellular immunity; corticosteroid exposure can further increase the risk of opportunistic infections and viral reactivation.

This report illustrates that severe syndrome of inappropriate antidiuretic hormone secretion (SIADH) may be an early and clinically actionable clue to HHV-6 encephalitis in a non-transplant patient with smoldering ATLL. Coexisting CO_2 narcosis and opportunistic pneumonia were important concurrent features that complicated the interpretation of encephalopathy.

Case Report

A 55-year-old woman was referred to the ophthalmology department 69 days before admission because of visual impairment. Blood testing revealed human T-cell leukemia virus type 1 (HTLV-1) positivity. Evaluation by the hematology and infectious diseases departments led to a diagnosis of smoldering ATLL. Because the patient's visual impairment was clinically diagnosed as HTLV-1-associated uveitis, systemic prednisolone was initiated at 50 mg/day 57 days before admission and subsequently tapered.

On admission, she was alert and afebrile at the initial assessment, although a nocturnal fever of up to 38 °C was observed. Her temperature was 36.8 °C, blood pressure was 116/80 mm Hg, pulse rate was 90 beats/min, respiratory rate was 18 breaths/min, and oxygen saturation was 96% on room air. Coarse crackles were noted in the right lower lung field. Laboratory findings on admission are summarized in **Table 1**. Chest imaging showed progressive pulmonary infiltrates (**Figure 1**).

Table 1. Laboratory findings on admission.

Test (units)	Value	Reference range	Test (units)	Value	Reference range
WBC (/μL)	11 000	3300-8600	Band neutrophils (%)	0.5	0-6
Segmented neutrophils (%)	91.0	40-70	Lymphocytes (%)	6.5	20-50
Monocytes (%)	2.0	2-10	Hemoglobin (g/dL)	11.5	11.6-14.8
Platelets (/μL)	400 000	158 000-348 000	Total protein (g/dL)	5.8	6.6-8.1
Albumin (g/dL)	2.5	4.1-5.1	A/G ratio	0.8	1.32-2.23
CRP (mg/dL)	33.76	< 0.14	BUN (mg/dL)	22	8-20
Creatinine (mg/dL)	0.53	0.46-0.79	eGFR (mL/min/1.73 m^2)	90.9	≥ 60
Uric acid (mg/dL)	2.7	2.6-5.5	Sodium (mmol/L)	137	138-145
Potassium (mmol/L)	4.0	3.6-4.8	Chloride (mmol/L)	100	101-108
Corrected calcium (mg/dL)	10.0	8.8-10.1	Phosphate (mg/dL)	3.6	2.7-4.6
Total bilirubin (mg/dL)	0.6	0.4-1.5	AST (U/L)	28	13-30
ALT (U/L)	39	7-23	LDH (U/L)	259	124-222
ALP (U/L)	88	38-113	Gamma-GTP (U/L)	49	9-32
Glucose (mg/dL)	147	73-109	HbA1c (%)	6.9	4.9-6.0
Beta-D-glucan (pg/mL)	58.6	< 20	Aspergillus antigen (index)	0.7	< 0.5
MAC antibody	Negative	Negative	IgG (mg/dL)	424	861-1747

Abbreviations: A/G ratio, albumin/globulin ratio; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; gamma-GTP, gamma-glutamyl transpeptidase; HbA1c, hemoglobin A1c; IgG, immunoglobulin G; LDH, lactate dehydrogenase; MAC, *Mycobacterium avium* complex; WBC, white blood cell count.

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

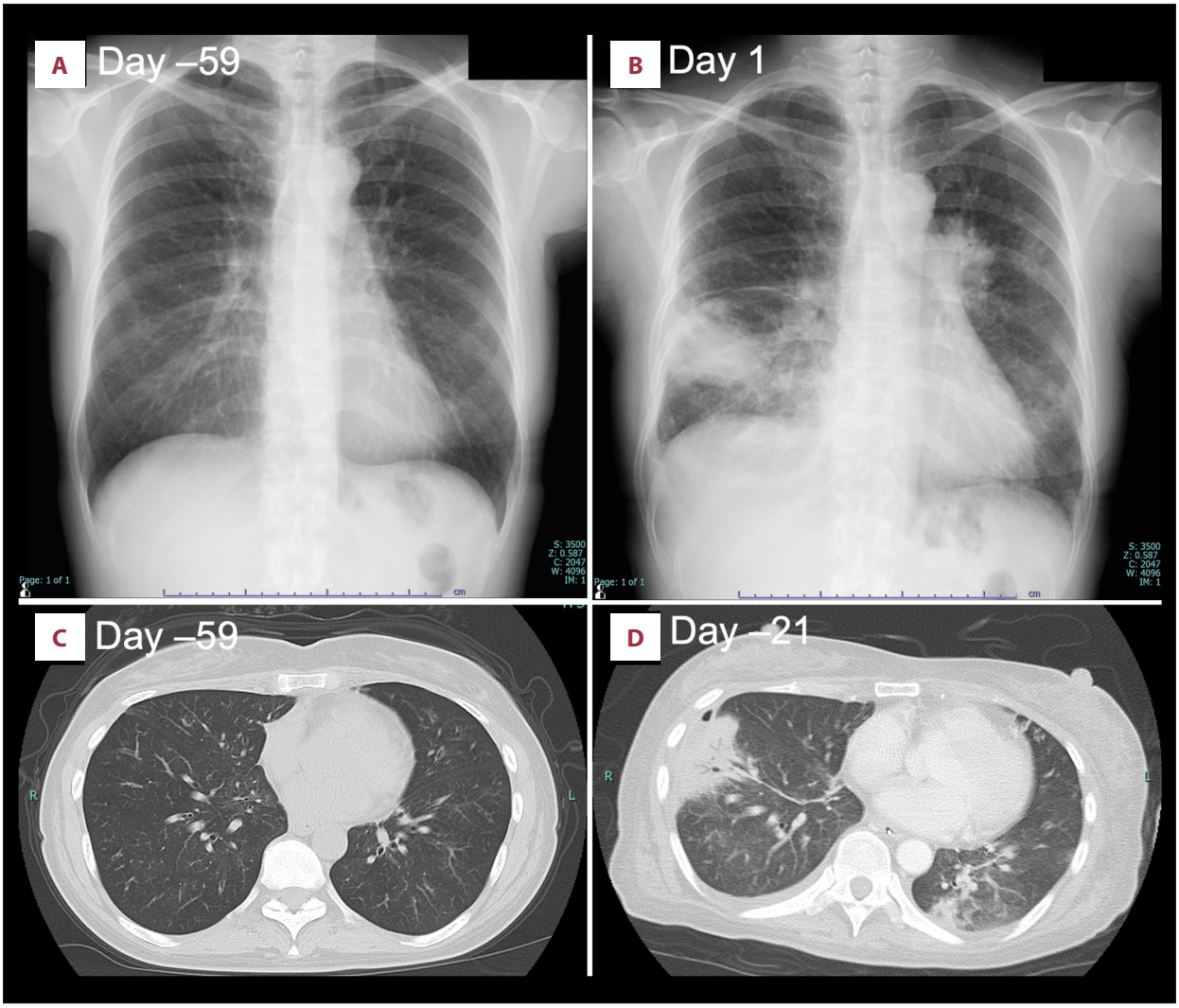

Figure 1. Chest imaging before admission and at admission. Chest radiography 59 days before admission showed no obvious pulmonary infiltrates (**A**), whereas chest radiography on hospital day 1 showed bilateral pulmonary opacities (**B**). Chest computed tomography 59 days before admission showed no corresponding lesions (**C**), whereas positron emission tomography-computed tomography 21 days before admission showed peripheral consolidation in the right lung and infiltrates in the left lower lobe (**D**).

On hospital day 2, bronchoscopy was performed. No obvious endobronchial abnormalities were observed; biopsy, brushing, and washing specimens were obtained from right B8a. After the specimens had been submitted for microbiologic and histopathologic testing, empirical treatment with imipenem/cilastatin, isavuconazole, and sulfamethoxazole/trimethoprim was initiated.

The bronchoscopic specimens subsequently revealed multiple pathogens and inflammatory findings. Tissue culture grew alpha- and gamma-hemolytic streptococci, *Nocardia*, and *Actinomyces*. Histopathologic examination showed inflammatory granulation tissue with neutrophilic and lymphocytic infiltration, as well as clusters of foamy histiocytes. Grocott staining did not detect fungal elements, and *Aspergillus* was not recovered from bronchoscopic cultures. Nevertheless, invasive pulmonary aspergillosis was clinically suspected based on the immunocompromised state associated with smoldering ATLL and systemic corticosteroid exposure, progressive bilateral pulmonary infiltrates, an elevated serum beta-D-glucan level (58.6 pg/mL), and a serum *Aspergillus* antigen index of 0.7 (laboratory reference value, < 0.5).

By hospital days 5-6, the fever had resolved, chest imaging showed improvement, and the C-reactive protein level was steadily decreasing (**Figure 2**). However, nausea and loss of appetite developed after initiation of isavuconazole. Given this temporal relationship, isavuconazole toxicity was initially

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

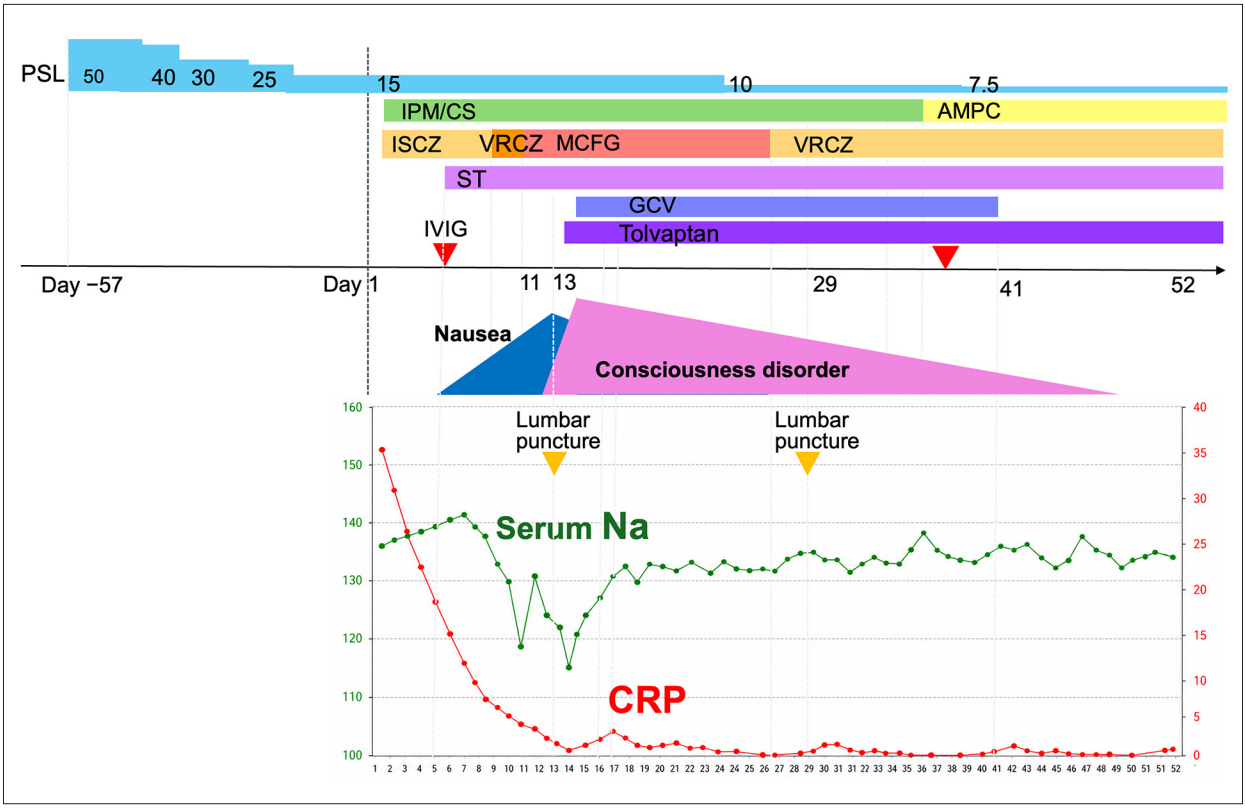

Figure 2. Clinical course. Timeline of corticosteroid therapy, antimicrobial and antifungal therapy, intravenous immunoglobulin, ganciclovir, tolvaptan, serum sodium, and C-reactive protein. The pulmonary infection improved clinically and biochemically after empirical treatment, but nausea and loss of appetite were followed by acute severe hyponatremia, impaired consciousness, human herpesvirus 6 (HHV-6) encephalitis, and intensive care management. AMPC, amoxicillin; CRP, C-reactive protein; GCV, ganciclovir; IPM/CS, imipenem/cilastatin; ISCZ, isavuconazole; IVIG, intravenous immunoglobulin; MCFG, micafungin; Na, sodium; PSL, prednisolone; ST, sulfamethoxazole/trimethoprim; VRCZ, voriconazole.

suspected, and isavuconazole was replaced with voriconazole; nevertheless, nausea and loss of appetite persisted.

On hospital day 11, the patient developed acutely impaired consciousness with rapidly progressive hyponatremia (serum sodium, 118 mmol/L). Head computed tomography showed no acute structural lesions. Symptomatic hyponatremia was initially regarded as the main cause of encephalopathy, and hypertonic saline was administered. Because azole-associated hyponatremia was considered, voriconazole was temporarily discontinued and replaced with micafungin. The persistence of nausea and loss of appetite despite sequential changes from isavuconazole to voriconazole and then to micafungin made azole-related toxicity less likely. Laboratory testing during this episode showed a serum osmolality of 238 mOsm/kg H_2O, urine osmolality of 670 mOsm/kg H_2O, urine sodium > 250 mmol/L, urine potassium of 35 mmol/L, creatinine of 0.41 mg/dL, glucose of 123 mg/dL, thyroid-stimulating hormone of 0.925 mIU/L, free T4 of 1.64 ng/dL, adrenocorticotropic hormone of 8.9 pg/mL, cortisol of 6.7 µg/dL, and serum antidiuretic hormone level of 4.3 pg/mL on hospital day 12. These findings supported SIADH in the setting of hypo-osmolar hyponatremia, with inappropriately concentrated urine, high urinary sodium, and an ADH level that was not suppressed despite hypo-osmolality.

On hospital day 13, because the patient's consciousness did not adequately improve after sodium correction, the neurology team was consulted and lumbar puncture was performed (**Table 2**). The FilmArray meningitis/encephalitis panel showed HHV-6 positivity. Quantitative HHV-6 DNA in cerebrospinal fluid was 1.0×10^3 copies/mL (laboratory reference value, $< 2.0 \times 10^2$ copies/mL). Brain magnetic resonance imaging revealed bilateral cortical and limbic hyperintense lesions (**Figure 3**). Because SIADH-associated hyponatremia persisted despite hypertonic saline, tolvaptan was initiated at 7.5 mg/day on hospital day 14. After initial sodium correction, serum sodium again decreased to 118 mmol/L on hospital day 14 and reached a nadir of 113 mmol/L on hospital day 15. Anti-HHV-6 therapy with ganciclovir was initiated on hospital day 15 following the diagnosis of HHV-6 encephalitis. The patient subsequently developed severe respiratory acidosis and CO_2 narcosis on hospital day 15. Her consciousness deteriorated to Japan Coma

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Table 2. Key findings at the onset of encephalopathy and SIADH.

Test (units)	Value	Reference range	Test (units)	Value	Reference range
Serum sodium on hospital day 14 (mmol/L)	118	138-145	Nadir serum sodium on hospital day 15 (mmol/L)	113	138-145
Serum osmolality on hospital day 14 (mOsm/kg H_2O)	238	275-295	Urine osmolality on hospital day 14 (mOsm/kg H_2O)	670	
Urine sodium on hospital day 14 (mmol/L)	> 250		Urine potassium on hospital day 14 (mmol/L)	35	
Creatinine on hospital day 14 (mg/dL)	0.41	0.46-0.79	Glucose on hospital day 14 (mg/dL)	123	73-109
TSH on hospital day 12 (mIU/L)	0.925	0.5-5.0	Free T4 on hospital day 12 (ng/dL)	1.64	0.9-1.7
ACTH on hospital day 12 (pg/mL)	8.9		Cortisol on hospital day 12 (μg/dL)	6.7	
ADH on hospital day 12 (pg/mL)	4.3		Arterial pH on hospital day 15	7.229	7.35-7.45
$PaCO_2$ on hospital day 15 (mm Hg)	77.5	35-45	HCO_3^- on hospital day 15 (mmol/L)	31.2	22-26
CSF cells on hospital day 13 (/μL)	1	0-5	CSF protein on hospital day 13 (mg/dL)	27	15-45
CSF glucose on hospital day 13 (mg/dL)	49		CSF HHV-6 PCR on hospital day 13	Positive	Negative
CSF HHV-6 DNA on hospital day 13 (copies/mL)	1.0×10^3	< 2.0×10^2			

Abbreviations: ACTH, adrenocorticotropic hormone; ADH, antidiuretic hormone; CSF, cerebrospinal fluid; HHV-6, human herpesvirus 6; PCR, polymerase chain reaction; TSH, thyroid-stimulating hormone.

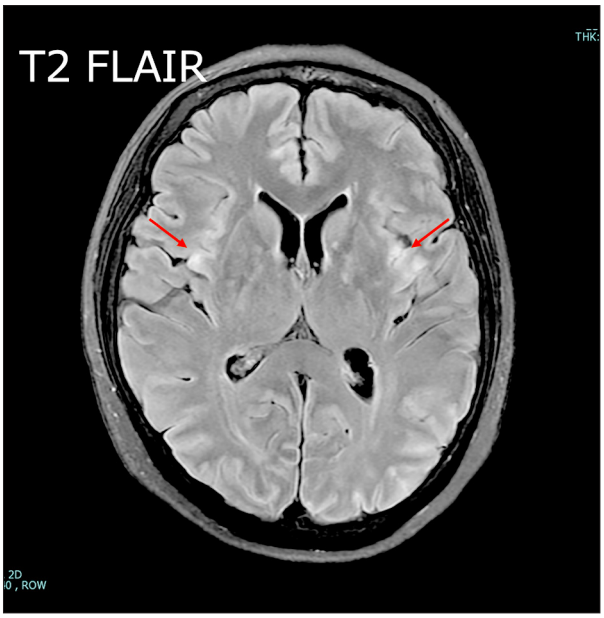

Figure 3. Brain magnetic resonance imaging scan at the onset of encephalopathy. T2-weighted fluid-attenuated inversion recovery (FLAIR) images showed bilateral cortical and limbic hyperintense lesions, including in the insular and temporal regions, providing clinical support for HHV-6 encephalitis.

Scale 300 (deepest level of unresponsiveness) before intensive care unit sedation was initiated; she underwent mechanical ventilation from hospital day 15 to hospital day 17. Fentanyl and midazolam were administered for sedation on hospital day 15. Midazolam was discontinued on hospital day 16, whereas fentanyl was continued until hospital day 17. She was then weaned from mechanical ventilation and extubated. Accordingly, neurologic assessment during mechanical ventilation, particularly on hospital days 15 and 16, was potentially confounded by sedative exposure.

Her serum sodium increased from a nadir of 113 mmol/L to 135 mmol/L by hospital day 18. Ganciclovir was continued until hospital day 41 after repeat CSF HHV-6 testing performed on hospital day 29 yielded results below the assay cutoff. On

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

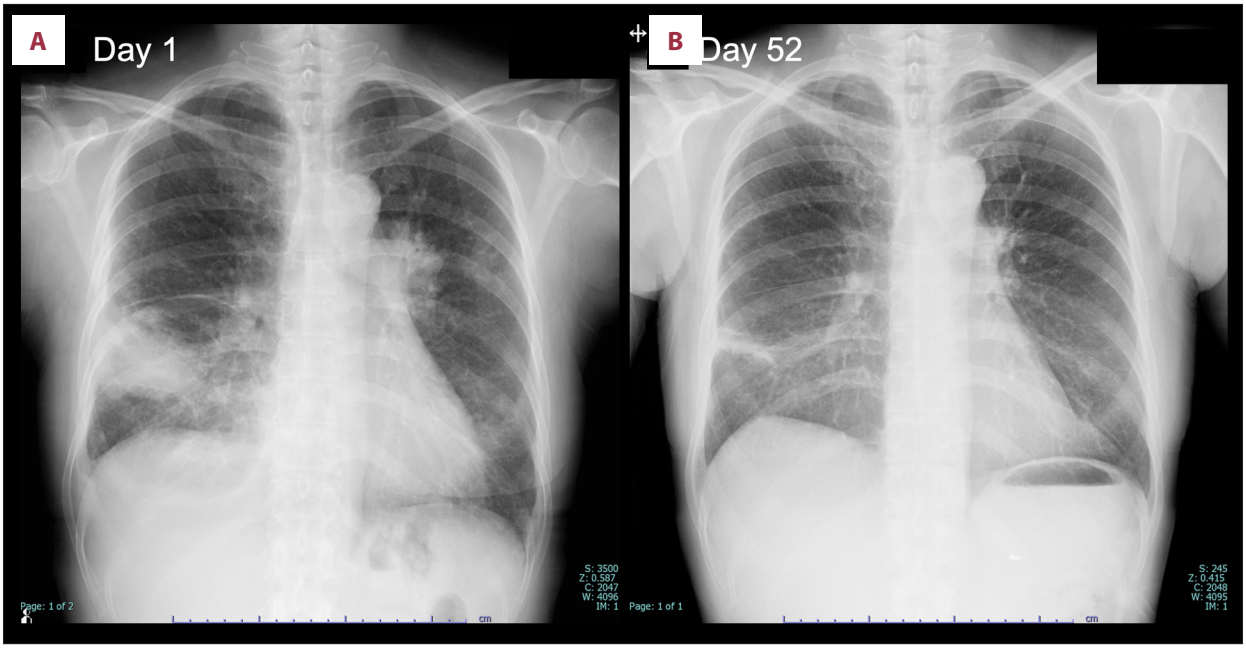

Figure 4. Chest radiographic improvement after treatment. Chest radiography on hospital day 1 showed bilateral lower-lung opacities, including dense consolidation in the right lower lung field (**A**). By hospital day 52, these opacities had substantially decreased; only a residual band-like opacity was evident in the right lower lung field after antimicrobial and antifungal therapy (**B**).

hospital day 52, serum sodium was 135 mmol/L and C-reactive protein was 0.10 mg/dL. The serum *Aspergillus* antigen index decreased from 0.7 on hospital day 1 to 0.3 on hospital day 12 and remained negative thereafter. Serum beta-D-glucan increased from 58.6 pg/mL on hospital day 1 to 422 pg/mL on hospital day 12 and remained elevated at 67.1 pg/mL on hospital day 53.

Follow-up chest radiography on hospital day 52 showed substantial reduction of the bilateral lower-lung opacities observed on hospital day 1; only a residual band-like opacity persisted in the right lower lung field (**Figure 4**). The patient was discharged on hospital day 53, and tolvaptan 7.5 mg/day was continued after discharge.

Discussion

The main diagnostic lesson from this case is that severe SIADH accompanied by new neurologic symptoms can be an early clue to HHV-6 encephalitis in a non-transplant patient with smoldering ATLL. A secondary lesson is that encephalopathy in this setting may be multifactorial; in our patient, hypercapnic respiratory failure and CO_2 narcosis occurred concurrently and thus required parallel assessment and treatment.

The novelty of this case is incremental but clinically relevant. Previous reports have described HHV-6 encephalitis in patients with ATLL and hyponatremia or SIADH in the setting of HHV-6 encephalitis, particularly after HSCT [6,7-10]. The present case provides additional evidence that this association can also occur in a non-transplant patient with smoldering ATLL after corticosteroid exposure and opportunistic infection. Thus, although this case does not establish a new disease mechanism, it extends a recognized diagnostic association to a less typical clinical context.

Our patient's clinical background was biologically plausible for HHV-6 reactivation despite the absence of HSCT. ATLL is associated with impaired cellular immunity and opportunistic infections, including in some patients with indolent subtypes [11,12]. In our patient, smoldering ATLL, high-dose corticosteroid therapy, and polymicrobial pulmonary infection likely combined to create a permissive immunologic setting.

Severe hyponatremia was a central feature of the present case. Previous studies have linked sodium abnormalities to HHV-6 encephalitis, particularly in HSCT recipients [7,8], and severe or persistent SIADH has mainly been described in transplant-related reports [9,10]. In our patient, SIADH preceded recognition of HHV-6 encephalitis and prompted evaluation for additional neurologic causes when consciousness did not sufficiently improve after sodium correction.

Several alternative or contributing causes of SIADH and encephalopathy required consideration. Pulmonary infection, nausea,

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

stress, respiratory failure, and azole antifungal therapy can all promote antidiuretic hormone secretion. Adrenocorticotropic hormone and serum cortisol were measured during the episode; however, because our patient was receiving systemic prednisolone, these single measurements could not definitively exclude adrenal insufficiency. Therefore, the present case supports HHV-6 encephalitis as a major and clinically actionable contributor to a multifactorial syndrome but does not establish that HHV-6 independently caused all of the hyponatremia or respiratory deterioration.

Treatment of HHV-6 encephalitis generally relies on foscarnet or ganciclovir. European Conference on Infections in Leukemia guidelines and Japanese recommendations suggest full-dose foscarnet or ganciclovir when tolerated, with treatment for at least 3 weeks and—when feasible—until viral DNA is cleared from the blood and cerebrospinal fluid [1,4]. In our patient, ganciclovir was selected on the basis of renal function, concerns regarding bone marrow toxicity, and concurrent infections. Improvement after sodium correction, respiratory support, and antiviral therapy supports the appropriateness of a combined management strategy, although the independent effect of each intervention cannot be determined.

The pulmonary infection itself illustrated the complexity of diagnosing pneumonia in an immunocompromised patient. *Nocardia* is an opportunistic pathogen associated with corticosteroid exposure, hematologic malignancy, chronic lung disease, and dissemination to the central nervous system [13,14]. *Actinomyces* can also cause chronic pulmonary infection and may coexist with other organisms [15,16]. In the present case, the pulmonary infection and its treatment provided important clinical context for the patient's immune vulnerability, as well as non-HHV-6 contributors to SIADH and encephalopathy.

A limitation of the pulmonary diagnosis is that invasive pulmonary aspergillosis was clinically suspected rather than microbiologically proven. Although *Aspergillus* was not recovered from bronchoscopic cultures, culture yield is low, and a negative culture does not exclude invasive aspergillosis [17]. Clinical suspicion in our patient was supported by smoldering ATLL, systemic corticosteroid exposure, progressive bilateral pulmonary infiltrates, an elevated serum beta-D-glucan level (58.6 pg/mL), and a modestly elevated serum *Aspergillus* antigen index of 0.7 (laboratory reference value, < 0.5). However, beta-D-glucan is not specific to *Aspergillus* and can be elevated in other infections, including disseminated nocardiosis [18]. Furthermore, the serum *Aspergillus* antigen result was supportive but not definitive, and improvement occurred during concurrent antibacterial and antifungal therapy; therefore, the response could not be specifically attributed to antifungal treatment. Accordingly, the pulmonary findings should be interpreted as a complex background of opportunistic infection, rather than as microbiologically proven invasive pulmonary aspergillosis.

Conclusions

In a non-transplant patient with smoldering ATLL, corticosteroid exposure, and opportunistic pulmonary infection, severe SIADH accompanied by new neurologic symptoms was an early clue to HHV-6 encephalitis. In selected immunocompromised patients with compatible neurologic features and clinical context, HHV-6 testing, brain magnetic resonance imaging, exclusion of alternative central nervous system infections, and consideration of anti-HHV-6 therapy may be appropriate. Early recognition and prompt initiation of antiviral therapy may help to improve neurologic outcomes in HHV-6 encephalitis.

Institution Where Work Was Done

Fukuoka University Hospital, Fukuoka, Japan.

Patient Consent

Written informed consent for publication was obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Ward KN, Hill JA, Hubacek P, et al. Guidelines from the 2017 European Conference on Infections in Leukaemia for management of HHV-6 infection in patients with hematologic malignancies and after hematopoietic stem cell transplantation. Haematologica. 2019;104(11):2155-63
2. Ogata M, Fukuda T, Teshima T. Human herpesvirus-6 encephalitis after allogeneic hematopoietic cell transplantation: what we do and do not know. Bone Marrow Transplant. 2015;50:1030-36
3. Ogata M, Oshima K, Ikebe T, et al. Clinical characteristics and outcome of human herpesvirus-6 encephalitis after allogeneic hematopoietic stem cell transplantation. Bone Marrow Transplant. 2017;52:1563-70
4. Ogata M, Uchida N, Fukuda T, et al. Clinical practice recommendations for the diagnosis and management of human herpesvirus-6B encephalitis after allogeneic hematopoietic stem cell transplantation: The Japan Society for Hematopoietic Cell Transplantation. Bone Marrow Transplant. 2020;55:1004-13
5. Imataki O, Uemura M. Ganciclovir-resistant HHV-6 encephalitis that progressed rapidly after bone marrow transplantation. J Clin Virol. 2015;69:176-78
6. Idutsu K, Abe Y, Otonari J, et al. [Human herpesvirus 6 encephalitis in a patient with adult T-cell leukemia/lymphoma.] Rinsho Ketsueki. 2007;48(8):664-66 [in Japanese]

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

7. Victoria NC, Phan TL, Agarwal KA. A systematic review of sodium disorders in HHV-6 encephalitis. Biol Blood Marrow Transplant. 2020;26(5):1034-39
8. Yoshida S, Ara T, Okada K, et al. The preceding hyponatremia is a useful hallmark for the diagnosis of HHV-6 encephalitis after allogeneic hematopoietic stem cell transplantation. Bone Marrow Transplant. 2023;58(1):97-99
9. Kawaguchi T, Takeuchi M, Kawajiri C, et al. Severe hyponatremia caused by syndrome of inappropriate secretion of antidiuretic hormone developed as initial manifestation of human herpesvirus-6-associated acute limbic encephalitis after unrelated bone marrow transplantation. Transpl Infect Dis. 2013;15(2):E54-57
10. Gannamani V, Varma A, Nathan S, Ustun C. Human herpesvirus 6 (HHV-6) associated permanent hyponatremia in umbilical cord blood transplant recipient. Transpl Immunol. 2023;76:101742
11. Kawano N, Nagahiro Y, Yoshida S, et al. Clinical features and treatment outcomes of opportunistic infections among HTLV-1 carriers and patients with adult T-cell leukemia-lymphoma (ATL) at a single institution from 2006 to 2016. J Clin Exp Hematop. 2019;59(4):156-67
12. Tanaka T, Sekioka T, Usui M, Imashuku S. Opportunistic infections in patients with HTLV-1 infection. Case Rep Hematol. 2015;2015:943867
13. Yetmar ZA, Marty PK, Clement J, et al. State-of-the-art review: Modern approach to nocardiosis-diagnosis, management, and uncertainties. Clin Infect Dis. 2025;80(4):e53-e64
14. Lafont E, Conan P-L, Rodriguez-Nava V, Lebeaux D. Invasive nocardiosis: Disease presentation, diagnosis and treatment-old questions, new answers? Infect Drug Resist. 2020;13:4601-13
15. Mabeza GF, Macfarlane J. Pulmonary actinomycosis. Eur Respir J. 2003;21(3):545-51
16. Boot M, Archer J, Ali I. The diagnosis and management of pulmonary actinomycosis. J Infect Public Health. 2023;16(4):490-500
17. Patterson TF, Thompson GR III, Denning DW, et al. Practice guidelines for the diagnosis and management of aspergillosis: 2016 update by the Infectious Diseases Society of America. Clin Infect Dis. 2016;63(4):e1-e60
18. Sawai T, Nakao T, Yamaguchi S, et al. Detection of high serum levels of beta-D-Glucan in disseminated nocardial infection: A case report. BMC Infect Dis. 2017;17:272

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]