

Communication

Sequential [^{11}C]Acetate and [^{18}F]FDG PET/CT Assessment of Systemic Chronic Active Epstein–Barr Virus Disease: An Exploratory Retrospective Study

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Abstract

Systemic chronic active Epstein–Barr virus disease (sCAEBV) is a rare and potentially fatal disorder characterized by inflammatory manifestations and organ infiltration by EBV-infected T- or NK-cells. Although [^{18}F]FDG PET/CT has limited utility for assessing the disease activity of sCAEBV, [^{11}C]acetate PET/CT has not previously been evaluated in this setting. We therefore conducted this exploratory retrospective study to assess the utility of sequentially performed [^{11}C]acetate and [^{18}F]FDG PET/CT in sCAEBV. Five patients diagnosed with sCAEBV according to the criteria of the Research Group on Measures against Intractable Diseases, Ministry of Health, Labour and Welfare of Japan (consistent with the 2017 WHO classification) and assessed between July 2017 and December 2018 were included; patients younger than 20 years were excluded. Each patient underwent both [^{11}C]acetate and 2-deoxy-2-[^{18}F]fluoro-D-glucose ([^{18}F]FDG) positron emission tomography/computed tomography (PET/CT) on the same day. The maximum and mean standardized uptake values (SUVmax and SUVmean) of the liver and spleen and the liver-to-spleen ratio (LSR) were correlated with laboratory parameters, including alanine aminotransferase (ALT) and lactate dehydrogenase (LDH), using Spearman’s rank correlation coefficient. The LSR was compared between active and inactive cases using the Mann–Whitney U test. Twenty-one lymph node regions were assessed in each patient, and the SUVmax of detected lesions was measured. The detection rate of lymph node lesions between the two tracers was compared using McNemar’s test, and the SUVmax of lymph node lesions was compared between the two tracers and between active and inactive cases using the Mann–Whitney U test. All statistical analyses were performed using R version 4.5.3 (R Foundation for Statistical Computing, Vienna, Austria), and a p -value < 0.05 was considered statistically significant. Five patients (three men and two women; mean age 31.8 years, range 21–39 years) were included. [^{11}C]acetate PET/CT showed significant negative correlations between spleen SUV and liver enzymes (AST, ALT, and LDH), and significant positive correlations between the LSR and all five liver enzymes tested (AST, ALT, LDH, γ GTP, and ALP) (Spearman’s rank correlation coefficient; $p < 0.05$ for all). No significant correlations were observed with [^{18}F]FDG PET/CT. The LSR on [^{11}C]acetate



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PET/CT was numerically higher in active cases than in inactive cases, though this difference was not statistically significant (0.88 ± 0.02 vs. 0.61 ± 0.02 ; $p = 0.20$, Mann–Whitney U test). Lymph node lesion detectability did not differ significantly between the two tracers (16 vs. 12 regions; $p = 0.13$, McNemar’s test). In this pilot study, [^{11}C]acetate PET/CT spleen SUV showed significant negative correlations with liver enzymes (AST, ALT, and LDH), and the LSR showed significant positive correlations with all measured liver enzymes, suggesting that [^{11}C]acetate PET/CT reflects both hepatic and splenic involvement in sCAEBV. [^{11}C]acetate PET/CT may therefore serve as a novel imaging biomarker for assessing disease activity in sCAEBV, warranting further investigation in larger cohorts.

Keywords: [^{11}C]acetate positron emission tomography/computed tomography; chronic active Epstein–Barr virus disease; dual tracer positron emission tomography/computed tomography

1. Introduction

Systemic chronic active Epstein–Barr virus disease (sCAEBV) is a rare and potentially fatal disorder that has been reported and studied mainly in Japan and other East Asian countries. The primary clinical manifestations of sCAEBV are inflammatory, including fever, lymphadenopathy, and liver dysfunction. Although most cases are not accompanied by solid tumors, sCAEBV is characterized by the clonal proliferation of Epstein–Barr virus (EBV)-infected T- and/or natural killer (NK) cells that infiltrate organs and can lead to organ failure. According to the World Health Organization (WHO) classification, sCAEBV is classified as an EBV-positive T- or NK-cell lymphoproliferative disease (EBV-T/NK LPD) [1,2].

2-deoxy-2- [^{18}F]fluoro-D-glucose ([^{18}F]FDG) positron emission tomography/computed tomography (PET/CT) is a useful imaging tool for assessing cancer and inflammatory diseases and plays a crucial role in the assessment of lymphoma [3]. Previous studies have shown that [^{18}F]FDG uptake in sCAEBV is generally low, whereas avid [^{18}F]FDG uptake suggests transformation to lymphoma [4,5]. Although [^{18}F]FDG PET/CT is therefore useful for detecting malignant transformation and guiding therapeutic decisions, its findings have not been established as helpful for assessing the disease activity of sCAEBV that has not progressed to malignant lymphoma, and provide limited information on the characteristics of such cases [4,5].

Acetate (acetic acid) serves two main metabolic roles in the cell: the formation of cell membranes and the production of energy. In tumor cells, acetate is used to synthesize cholesterol and fatty acids for cell membrane formation, whereas in cardiac myocytes, it is oxidized to water and carbon dioxide (CO_2) to produce energy. Accordingly, [^{11}C]acetate PET/CT has been considered useful for assessing both cardiac and oncologic diseases. Several studies have demonstrated that [^{11}C]acetate PET/CT is useful for assessing malignant diseases, including prostate cancer, hepatocellular carcinoma, renal cell carcinoma, bladder cancer, multiple myeloma, and slow-growing lung cancer [6–11]. A previous study also demonstrated its efficacy in evaluating less aggressive or indolent lymphomas that are not [^{18}F]FDG-avid [12]. These findings suggest that [^{11}C]acetate PET/CT may be useful for evaluating other non-[^{18}F]FDG-avid malignancies, including sCAEBV. Moreover, EBV upregulates fatty acid biosynthesis and desaturation—including stearoyl-CoA desaturase 1 (SCD1) and fatty acid desaturase 2 (FADS2)—and activates sterol regulatory element-binding protein (SREBP)-driven lipogenic programs, producing a metabolic phenotype that depends on a sustained supply of acetyl-CoA for membrane synthesis and signal-

ing lipids [13,14]. Although these findings were derived from studies of EBV-infected B cells, they may provide a mechanistic rationale for investigating whether altered lipid metabolism contributes to [^{11}C]acetate uptake in EBV-driven proliferative lesions, including sCAEBV. To the best of our knowledge, no previous study has evaluated [^{11}C]acetate PET/CT in sCAEBV; we therefore conducted this exploratory retrospective study to assess its utility using sequentially performed [^{11}C]acetate and [^{18}F]FDG PET/CT.

2. Methods

2.1. Diagnosis of sCAEBV

We reviewed the enrolled patients to determine whether they met the following diagnostic criteria proposed by the Research Group on Measures against Intractable Diseases, Ministry of Health, Labour and Welfare of Japan:

1. Elevated Epstein–Barr virus DNA load in peripheral blood (PB) ($>10^{2.5}$ copies/ μg DNA);
2. EBV infection of T- or NK-cells in the affected tissues or PB;
3. Systemic inflammatory symptoms (such as fevers, lymphadenopathy, liver dysfunction, progressive skin lesions, vasculitis, and uveitis) persisting for more than 3 months;
4. Exclusion of other possible diagnoses: primary EBV infection (infectious mononucleosis), autoimmune disease, congenital immunodeficiency, human immunodeficiency virus (HIV) infection, and other immunodeficiencies requiring immunosuppressive therapy, or underlying diseases with potential immunosuppression [15].

Patients who fulfilled criteria 1–4 were diagnosed with sCAEBV. In contrast, patients who presented only with hypersensitivity to mosquito bites or with hydroa vacciniforme-like eruptions—a photosensitivity reaction characterized by papulovesicular eruptions typically on the face—without sustained systemic inflammation, were not diagnosed with sCAEBV. In addition, patients with a pathological diagnosis of extranodal NK/T-cell lymphoma (nasal type), aggressive NK-cell leukemia, or peripheral T-cell lymphoma made concurrently with or before the suspected diagnosis of sCAEBV were excluded. These criteria are consistent with the definition of sCAEBV in the WHO classification revised in 2017 [16].

2.2. Ethics Statements

We included five patients who underwent [^{11}C]acetate PET/CT for the assessment of sCAEBV between July 2017 and December 2018 at our institution. Both patients with active sCAEBV and those with inactive disease were eligible for inclusion, irrespective of disease activity at the time of imaging; disease activity was classified as described in Section 2.3. The study was approved by our Institutional Review Board (M2016-088, approved on 2 August 2016), and written informed consent was obtained from all participants. Individuals under 20 years of age were excluded.

2.3. Definition of Disease Activity

Active disease was defined as the presence of persistent inflammatory findings (fever, liver dysfunction, progressive skin lesions, vasculitis, or uveitis). Liver dysfunction was defined as an alanine aminotransferase level greater than twice the upper limit of normal. Progressive skin lesions and vasculitis were diagnosed by pathological examination, and uveitis was diagnosed by the attending physician and an ophthalmologist. Patients without these clinical findings were classified as having inactive disease [15,17].

2.4. Laboratory Finding Collection

Within 3 weeks before and after PET/CT, we obtained laboratory parameters, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), alkaline phosphatase (ALP), and gamma-glutamyl transpeptidase (γ GTP) for analysis.

2.5. Imaging Protocol

[^{11}C]acetate was synthesized from [^{11}C]CO₂ by carboxylation of a Grignard reagent according to an established method, as previously described [12]; the detailed radiosynthesis and quality-control procedures are provided in Supplementary Methods S1. All patients fasted for no less than 6 h before the PET/CT scan, and their blood glucose levels were determined. At a mean of 7.2 min (range, 6–8 min) after administration of 3.7 MBq (0.1 mCi)/kg of [^{11}C]acetate, a whole-body static PET/CT scan was performed from the skull base to the mid-thigh region. This early acquisition time reflects the rapid tissue uptake and early plateau of [^{11}C]acetate, together with the short physical half-life of ^{11}C (20.4 min), which favor prompt imaging. Subsequently, [^{18}F]FDG was injected at a dose of 3.7 MBq (0.1 mCi)/kg, and [^{18}F]FDG PET/CT was performed approximately 60 min after injection. Both examinations were performed sequentially on the same day in a single imaging session—[^{11}C]acetate first, owing to the short physical half-life of ^{11}C , followed by [^{18}F]FDG—so that no maximum interval between the two modalities was prospectively defined. The median interval between the start of the [^{11}C]acetate and [^{18}F]FDG acquisitions was 107.5 min (mean, 105.8 min; range, 91–117 min; $n = 4$, Case 5 excluded). All PET/CT scans were completed using an advanced PET/CT scanner (Celesteion; Canon Medical Systems, Otawara, Japan). CT scans were acquired with the following parameters: tube voltage, 120 kV; field of view, 550 mm; pitch, 16.0; and slice thickness, 2.0 mm. PET emission scans were acquired in three-dimensional mode at 2 min per bed position, with a pixel size of 4 mm and a Gaussian filter of 6 mm. PET images were reconstructed using ordered-subsets expectation maximization with a time-of-flight algorithm (2 iterations, 10 subsets).

2.6. Imaging Analysis

[^{11}C]acetate and [^{18}F]FDG PET/CT images were independently reviewed by two board-certified nuclear medicine physicians. The liver and spleen were selected as the primary organs of interest because they are the principal sites of organ infiltration by EBV-infected T- or NK-cells in sCAEBV, and hepatic and splenic involvement is closely associated with disease activity and prognosis in this disease [15,17]. The distribution of each tracer was assessed by placing a 1 cm volume of interest (VOI) on the liver and spleen, and the maximum and mean standardized uptake values (SUVmax and SUVmean) were measured on a dedicated workstation (syngo.via; Siemens Healthineers, Erlangen, Germany). The liver-to-spleen ratio (LSR) was calculated as the SUVmax of the liver divided by the SUVmean of the spleen. Twenty-one lymph node regions were evaluated in each patient: bilateral cervical, Waldeyer's ring, occipital, bilateral supraclavicular, bilateral axillary, bilateral brachial, mediastinal, bilateral hilar, para-aortic, mesenteric, bilateral iliac, bilateral inguinal, and bilateral femoral. A lymph node lesion was considered positive when it visually showed uptake clearly exceeding that of adjacent adipose tissue, which served as background reference. Adjacent adipose tissue was selected as a uniform, low-activity background applicable to both tracers, because [^{11}C]acetate—unlike [^{18}F]FDG—has no established organ-based reference criteria for nodal assessment, and a common visual background allowed the two tracers to be compared on the same basis in this exploratory study. SUVmax was measured in positive lesions for semi-quantitative assessment.

When the two readers' assessments differed, a final decision was reached by consensus, and the SUV values from the two readers were averaged.

2.7. Statistical Analysis

Because of the small sample size and the non-normal distribution of the variables, non-parametric tests were used. Correlations between the laboratory data and the SUV values of the liver or spleen, as well as the LSR, were assessed using Spearman's rank correlation coefficient (ρ). The direction of each correlation was defined by the sign of ρ (positive or negative), and its strength was interpreted as negligible ($|\rho| < 0.10$), weak (0.10–0.39), moderate (0.40–0.69), strong (0.70–0.89), or very strong (0.90–1.00). The LSR on [^{11}C]acetate PET/CT and on [^{18}F]FDG PET/CT were each compared between active and inactive cases using the Mann–Whitney U test. The detection rate of lymph node lesions between the two tracers was compared using McNemar's test. The SUVmax of lymph node lesions was compared between the two tracers, and between active and inactive cases, using the Mann–Whitney U test. In addition, the SUVmax of lymph node lesions detected by both [^{11}C]acetate and [^{18}F]FDG PET/CT was compared using the Wilcoxon signed-rank test. Correlation coefficients are presented for descriptive purposes. Due to the ultra-rare nature of sCAEBV, a formal power calculation was not feasible; accordingly, all analyses should be considered exploratory and hypothesis-generating rather than confirmatory. The distribution of the SUVmax of lymph node lesions was visualized using box plots. All statistical analyses were performed using R version 4.5.3 (R Foundation for Statistical Computing, Vienna, Austria).

3. Results

Five patients were used in this study (three men and two women; mean age: 31.8 years, range: 21–39 years). Lymphadenopathy, splenomegaly, and hepatomegaly were present in two (40%), four (80%), and three (60%) patients, respectively. All the patients underwent [^{11}C]acetate PET/CT, and four underwent [^{18}F]FDG PET/CT on the same day. One patient did not undergo [^{18}F]FDG PET/CT and, therefore, was excluded from the analysis. The information of the five patients is presented in Table 1, and the laboratory results and SUV values of the five patients are presented in Table 2. Two patients (Cases 1 and 5) were therapy-naïve, whereas three had received prior therapy before PET/CT: Cases 2 and 3 had undergone allogeneic bone marrow transplantation, and Case 4 had received eight cycles of CHOP chemotherapy. Three patients had active disease, and one, Case 5, died 295 days after the PET/CT scanning due to disease progression. The median follow-up time was 71.7 months (range, 9.7–101.2 months).

The correlations between laboratory data (AST, ALT, LDH, γGTP , and ALP), the SUVs of the liver and spleen, and the LSR are shown in Table 3. Regarding [^{11}C]acetate PET/CT, significant negative correlations were found between AST and spleen SUVmax ($r = -1.00$, $p < 0.001$), AST and spleen SUVmean ($r = -0.97$, $p = 0.005$), ALT and spleen SUVmax ($r = -0.97$, $p = 0.005$), ALT and spleen SUVmean ($r = -0.92$, $p = 0.026$), and LDH and spleen SUVmax ($r = -0.90$, $p = 0.037$). Additionally, significant positive correlations were identified between the LSR and ALT ($r = 0.97$, $p = 0.005$), LDH ($r = 1.00$, $p < 0.001$), AST ($r = 0.90$, $p = 0.037$), γGTP ($r = 0.90$, $p = 0.037$), and ALP ($r = 0.90$, $p = 0.037$) of [^{11}C]acetate PET/CT (all by Spearman's rank correlation coefficient test). In contrast, no significant correlations were observed between any laboratory parameters and [^{18}F]FDG PET/CT SUV values or LSR.

Table 1. Patient characteristics.

Case	Age (Years)	Sex	EBV-Infected Cell Phenotype	Disease Activity	HLH	Cardiac Lesions	CNS Lesions	Outcome
1	33	Male	CD4+	Active (fever)	None	None	None	Alive
2	39	Male	CD4+	Active (fever)	None	None	None	Alive
3	21	Female	CD4+, CD56+	Inactive	None	None	None	Alive
4	30	Female	CD4+	Inactive	None	None	None	Alive
5	36	Male	CD4+	Active (fever)	None	Cardiac dilation, Mild pericardial effusion	Multiple old lacunar infarction	Died †

† Case 5 died 295 days after PET/CT scanning due to disease progression. EBV, Epstein–Barr virus; HLH, hemophagocytic lymphohistiocytosis; CNS, central nervous system. Yellow rows indicate active disease. The Cardiac lesions and CNS lesions columns report imaging findings; Case 3 had mild intellectual disability, a pre-existing clinical diagnosis (clinical comorbidity unrelated to sCAEBV), and brain imaging showed no abnormality.

Table 2. Laboratory parameters and SUV values.

Case	Disease Activity	Laboratory Values					^[11C] Acetate PET/CT					^[18F] FDG PET/CT				
		AST (U/L)	ALT (U/L)	LDH (U/L)	γGTP (U/L)	ALP (U/L)	Liver		Spleen		LSR	Liver		Spleen		LSR
							SUVmax	SUVmean	SUVmax	SUVmean		SUVmax	SUVmean	SUVmax	SUVmean	
1	Active	17	11	167	23	291	5.1	4.05	7.3	5.95	0.86	2.45	1.8	2	1.6	1.5
2	Active	22	20	403	68	269	3.9	3.2	5.25	4.5	0.87	2.35	1.8	2.25	1.6	1.5
3	Inactive	18	11	164	15	245	2.8	2.15	5.35	4.5	0.62	2.25	1.8	1.7	1.4	1.6
4	Inactive	14	9	138	18	152	3.55	2.7	7.5	6	0.59	2.4	1.9	2.2	1.85	1.3
5	Active	54	29	427	73	466	3.25	2.6	4.35	3.6	0.9	NA	NA	NA	NA	NA

SUVmax/mean, maximum/mean standardized uptake value; LSR, liver-to-spleen ratio; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; γGTP, gamma-glutamyl transpeptidase; ALP, alkaline phosphatase; NA, not available. Normal reference ranges: AST, 13–30 U/L; ALT, 10–42 U/L (male)/7–23 U/L (female); LDH, 124–222 U/L; γGTP, 13–64 U/L (male) / 9–32 U/L (female); ALP, 38–113 U/L. Yellow rows indicate active disease.

Table 3. Spearman’s rank correlation coefficients (r) and p-values between laboratory parameters and SUV values.

Parameter	^{[11} C]Acetate PET/CT										^{[18} F]FDG PET/CT									
	Liver				Spleen				LSR		Liver				Spleen				LSR	
	SUVmax r	SUVmax p	SUVmean r	SUVmean p	SUVmax r	SUVmax p	SUVmean r	SUVmean p	r	p	SUVmax r	SUVmax p	SUVmean r	SUVmean p	SUVmax r	SUVmax p	SUVmean r	SUVmean p	r	p
AST	−0.30	0.624	−0.30	0.624	−1.00	<0.001	−0.97	0.005	0.90	0.037	−0.60	0.400	−0.77	0.225	0.20	0.800	−0.63	0.368	0.63	0.368
ALT	−0.10	0.870	−0.10	0.870	−0.97	0.005	−0.92	0.026	0.97	0.005	−0.32	0.684	−0.82	0.184	0.32	0.684	−0.50	0.500	0.50	0.500
LDH	0.10	0.873	0.10	0.873	−0.90	0.037	−0.82	0.089	1.00	<0.001	0.00	1.000	−0.77	0.225	0.40	0.600	−0.32	0.684	0.32	0.684
γGTP	0.30	0.624	0.30	0.624	−0.70	0.188	−0.56	0.322	0.90	0.037	0.40	0.600	−0.26	0.742	0.80	0.200	0.32	0.684	−0.32	0.684
ALP	0.20	0.747	0.20	0.747	−0.70	0.188	−0.67	0.219	0.90	0.037	0.40	0.600	−0.77	0.225	0.00	1.000	−0.32	0.684	0.32	0.684

Spearman’s rank correlation coefficient test. n = 5 for [¹¹C]Acetate; n = 4 for [¹⁸F]FDG (Case 5 excluded). Yellow cells indicate $p < 0.05$. LSR, liver-to-spleen ratio; SUVmax/mean, maximum/mean standardized uptake value; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; γGTP, gamma-glutamyl transpeptidase; ALP, alkaline phosphatase.

Furthermore, the LSR on [^{11}C]acetate PET/CT did not differ significantly between active and inactive cases (0.88 ± 0.02 vs. 0.61 ± 0.02 ; $p = 0.20$, Mann–Whitney U test). Similarly, the LSR on [^{18}F]FDG PET/CT showed no significant difference between active and inactive cases (1.50 ± 0.00 vs. 1.45 ± 0.21 ; $p = 1.0$, Mann–Whitney U test).

In total, 16 lymph node lesions were evaluated across the cohort. Across four patients, [^{11}C]acetate PET/CT detected lymph node lesions in 16 regions, and [^{18}F]FDG PET/CT detected lesions in 12 regions ($p = 0.13$, McNemar’s test). [^{11}C]acetate PET/CT also identified all the lymph node lesions detected by [^{18}F]FDG PET/CT. The SUV_{max} of the lymph node lesions did not differ significantly between [^{11}C]acetate PET/CT and [^{18}F]FDG PET/CT (3.2 ± 1.4 vs. 2.6 ± 1.5 ; $p = 0.28$, Mann–Whitney U test). A boxplot of the SUV_{max} of lymph node lesions is shown in Figure 1. A paired analysis restricted to the 12 lesions detected by both modalities also showed no significant difference in SUV_{max} ($p = 0.052$, Wilcoxon signed-rank test).

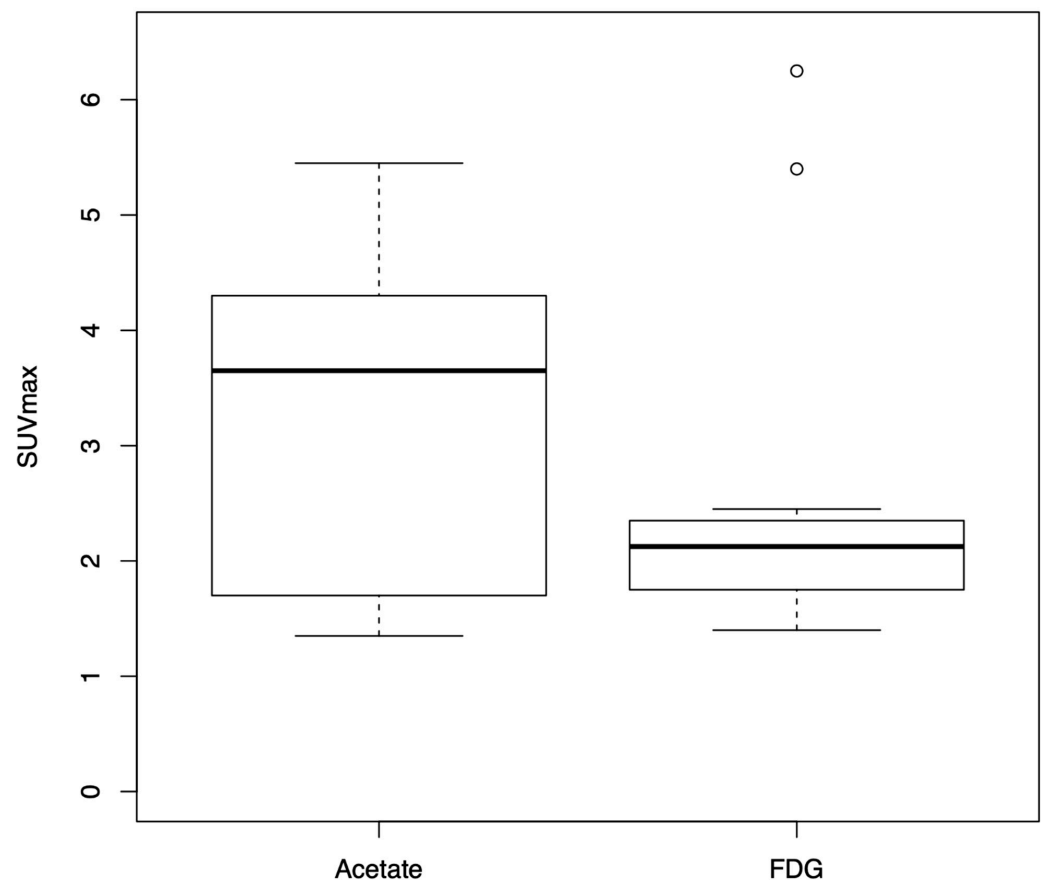


Figure 1. Boxplot of SUV_{max} of lymph node lesions detected by [^{11}C]acetate PET/CT and [^{18}F]FDG PET/CT. The median and interquartile range of SUV_{max} were higher for [^{11}C]acetate than for [^{18}F]FDG, with two high outliers observed in the [^{18}F]FDG group; however, the difference between the two tracers was not statistically significant ($p = 0.28$, Mann–Whitney U test).

Similarly, the SUV_{max} of lymph node lesions on [^{11}C]acetate PET/CT did not differ significantly between active and inactive cases (3.5 ± 1.4 vs. 2.1 ± 0.6 ; $p = 0.19$, Mann–Whitney U test; Figure 2). The SUV_{max} of lymph node lesions on [^{18}F]FDG PET/CT likewise showed no significant difference between active and inactive cases (2.9 ± 1.7 vs. 1.7 ± 0.4 ; $p = 1.0$, Mann–Whitney U test; Figure 3). All cases are shown in Figures 4–8. On visual assessment of maximum intensity projection images, cardiac [^{11}C]acetate uptake was more clearly demonstrated in the active case shown in Figure 8 (case 5) than in the inactive cases.

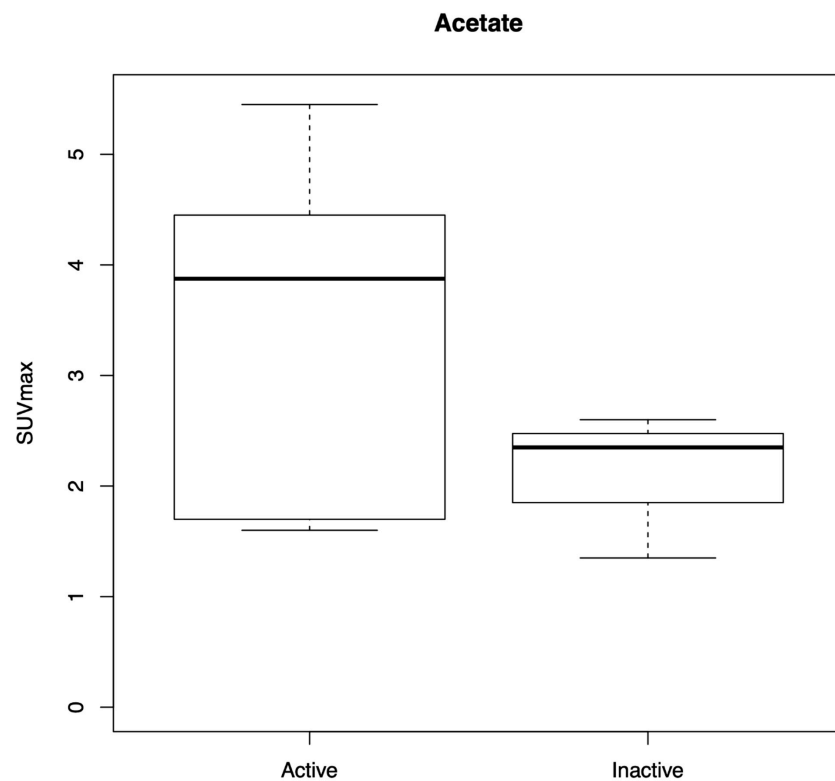


Figure 2. Boxplot of SUV_{max} of lymph node lesions on [¹¹C]acetate PET/CT, stratified by disease activity. The median and interquartile range of SUV_{max} were higher in the active group than in the inactive group; however, the difference between the two groups was not statistically significant ($p = 0.19$, Mann–Whitney U test).

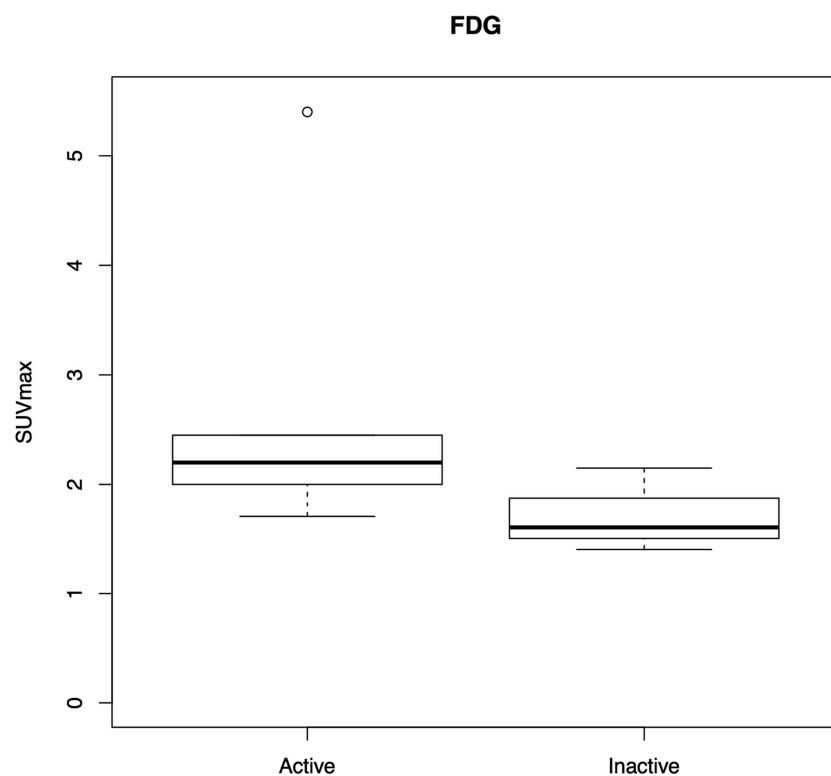


Figure 3. Boxplot of SUV_{max} of lymph node lesions on [¹⁸F]FDG PET/CT, stratified by disease activity. The median and interquartile range of SUV_{max} were slightly higher in the active group than in the inactive group, with one high outlier observed in the active group; however, the difference between the two groups was not statistically significant ($p = 1.0$, Mann–Whitney U test).

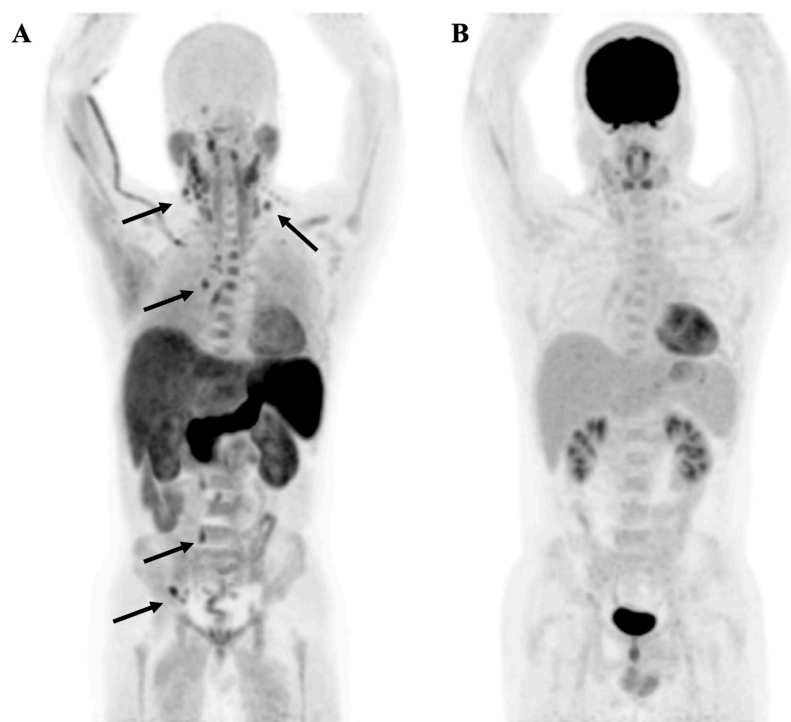


Figure 4. Sequential [^{11}C]acetate and [^{18}F]FDG PET/CT of a 33-year-old man with chronic active Epstein–Barr virus (EBV) disease (Case 1, active disease). [^{11}C]acetate uptake (A) is more clearly visualized in the bilateral cervical, right supraclavicular, right hilar, mediastinal, para-aortic, and right iliac lymph nodes (arrows) than [^{18}F]FDG uptake (B). The corresponding [^{11}C]acetate SUVmax values were 4.8 (right cervical), 4.3 (left cervical), 4.5 (right supraclavicular), 5.0 (right hilar), 4.1 (mediastinal), 3.8 (para-aortic), and 5.5 (right iliac).

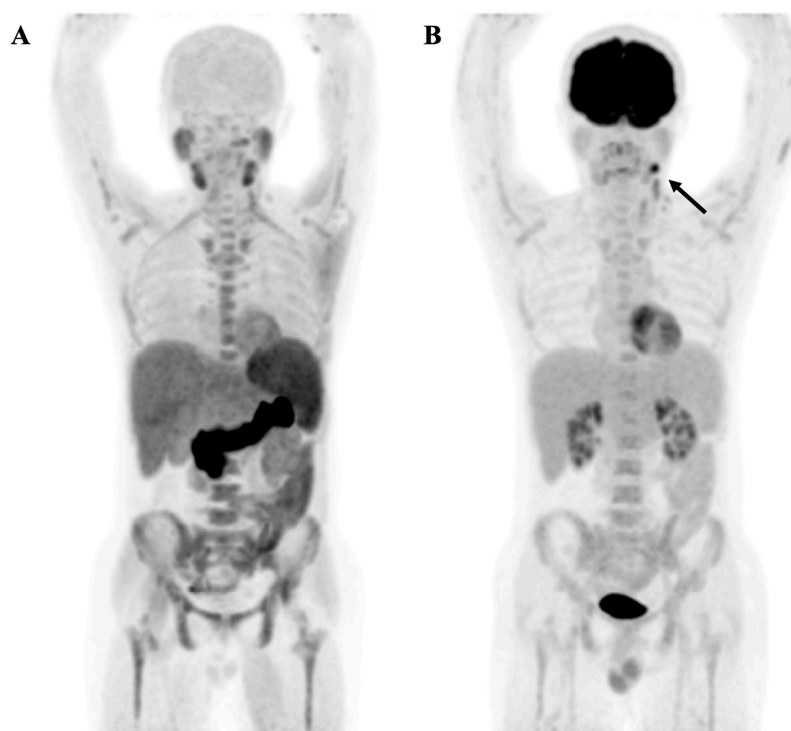


Figure 5. Sequential [^{11}C]acetate and [^{18}F]FDG PET/CT of a 39-year-old man with active chronic Epstein–Barr virus disease (case 2). [^{18}F]FDG (B) reveals focal uptake in the left cervical lymph node (arrow), which is more clearly visualized than on [^{11}C]acetate (A). The left cervical lymph node showed an [^{18}F]FDG SUVmax of 6.3 and a [^{11}C]acetate SUVmax of 3.5.

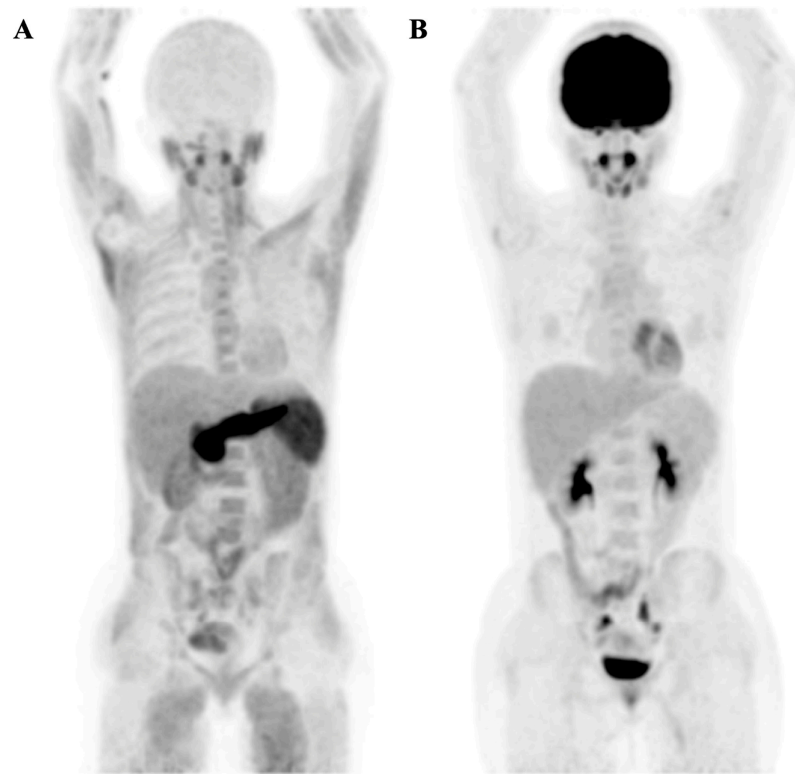


Figure 6. Sequential [^{11}C]acetate and [^{18}F]FDG PET/CT of a 21-year-old woman with chronic active EBV disease (case 3, inactive disease). No abnormal uptake is seen in [^{11}C]acetate PET/CT (A) or [^{18}F]FDG PET/CT (B).

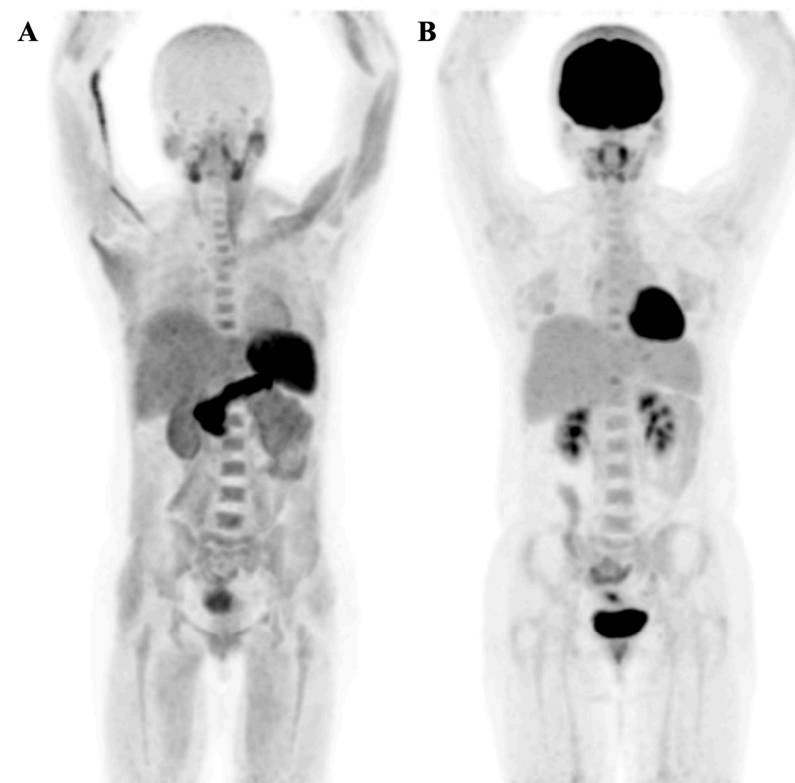


Figure 7. Sequential [^{11}C]acetate and [^{18}F]FDG PET/CT in a 30-year-old woman with chronic active EBV disease (Case 4, inactive disease). No abnormal uptake is seen on either [^{11}C]acetate PET/CT (A) or [^{18}F]FDG PET/CT (B).

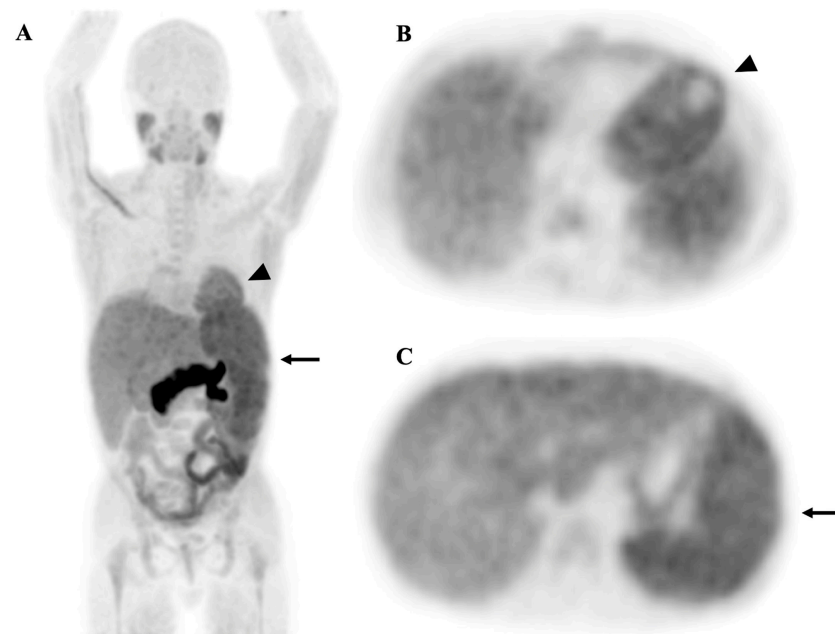


Figure 8. $[^{11}\text{C}]$ acetate PET/CT in a 36-year-old man with chronic active EBV disease (Case 5, active disease). The maximum intensity projection (MIP) image (A) demonstrates marked splenomegaly (arrow). On visual assessment of the MIP (A) and axial PET images, cardiac $[^{11}\text{C}]$ acetate uptake ((A,B); arrowheads) is comparable to splenic uptake ((A,C); arrows), consistent with decreased splenic acetate metabolism in active disease. The $[^{11}\text{C}]$ acetate SUVmax and SUVmean were 4.2 and 3.4 for the spleen and 3.93 and 1.76 for the myocardium, respectively.

4. Discussion

To the best of our knowledge, this is the first study to assess $[^{11}\text{C}]$ acetate PET/CT in patients with sCAEBV. In this exploratory analysis, $[^{11}\text{C}]$ acetate spleen SUVmax showed significant negative correlations with AST, ALT, and LDH, and spleen SUVmean with AST and ALT, while the $[^{11}\text{C}]$ acetate LSR showed significant positive correlations with all measured liver enzymes (AST, ALT, LDH, γ GTP, and ALP). These findings suggest that $[^{11}\text{C}]$ acetate uptake may reflect both hepatic and splenic disease activity in sCAEBV, although the small sample size precludes firm conclusions. No comparable correlations were observed with $[^{18}\text{F}]$ FDG PET/CT. The $[^{11}\text{C}]$ acetate LSR also tended to be higher in active than in inactive cases, although this difference did not reach statistical significance.

Additionally, normal uptake of $[^{11}\text{C}]$ acetate in the spleen tends to be slightly higher than that in the liver [18,19]. Among the five patients in this study, the patient who died had the highest LSR value of $[^{11}\text{C}]$ acetate and the lowest spleen SUV_{mean} value of $[^{11}\text{C}]$ acetate, in addition to splenomegaly (Figure 8). Furthermore, the $[^{11}\text{C}]$ acetate spleen SUV values and the liver enzymes showed a marginal negative correlation. However, a previous study revealed that elevated serum inflammatory cytokine levels, including tumor-necrosis-factor-alpha, interleukin-1, and interferon-gamma, in patients with sCAEBV and tumor-necrosis-factor-alpha acted as powerful inhibitors of lipogenesis [20,21]. Therefore, we speculate that tumor-necrosis-factor-alpha may reduce fatty acid synthesis and thereby decrease $[^{11}\text{C}]$ acetate uptake in the spleen. In addition, a previous study showed that EBV-infected T- or NK-cells infiltrate vessels and sinusoids of the liver [17]. Moreover, another study showed that EBV-infected T- or NK-cells infiltrating the liver directly induce hepatocellular apoptosis, resulting in liver dysfunction and elevated serum liver enzyme levels [22]. These findings imply that the correlation between elevated $[^{11}\text{C}]$ acetate LSR and liver enzymes in circulation may be caused by inflammation or apoptosis associated with the infiltrating EBV-infected T- or NK-cells. These results may contribute to the

positive correlation between [^{11}C]acetate LSR and liver enzymes and elevation of the LSR of [^{11}C]acetate in active cases. Additionally, liver dysfunction in patients with sCAEBV and disease activity of sCAEBV have been correlated with poor prognosis after hematopoietic stem cell transplantation [15,17,23,24]. Therefore, [^{11}C]acetate PET/CT LSR elevation may be related to poor prognosis in patients with sCAEBV.

According to the nationwide survey by Yonese et al. [15], lymphadenopathy was reported in 53% of patients with sCAEBV. Because the lymph nodes in sCAEBV are typically small and show low [^{18}F]FDG accumulation, they are often considered non-specific lesions. Therefore, the detectability of lymph node lesions was not significantly different between the two tracers. In a previous study, [^{11}C]acetate PET/CT detected more lymph node lesions in low-grade lymphoma than [^{18}F]FDG PET/CT [12]. This contrasts with our findings, which revealed no statistically significant difference in lymph node detection between [^{11}C]acetate PET/CT and [^{18}F]FDG PET/CT. However, some lymph node lesions detected by [^{11}C]acetate PET/CT were not detected by [^{18}F]FDG PET/CT. Furthermore, as shown in Figure 4, [^{11}C]acetate PET/CT revealed more lymph node lesions than [^{18}F]FDG PET/CT. Therefore, [^{11}C]acetate PET/CT may be equivalent or even superior to [^{18}F]FDG PET/CT for detecting lymph node lesions in CAEBV. Further studies are warranted to confirm the potential superiority of [^{11}C]acetate PET/CT over [^{18}F]FDG PET/CT.

Moreover, we found that the SUV_{max} of the lymph node lesions of the two tracers was not significantly different, which was consistent with a previous study [12]. In this study, the SUV_{max} of [^{11}C]acetate was higher than that of [^{18}F]FDG PET/CT in lymph node lesions, although the difference did not reach statistical significance ($p = 0.052$). Furthermore, the SUV_{max} distribution of [^{18}F]FDG had a narrower interquartile range than that of [^{11}C]acetate PET/CT, and the SUV_{max} values of [^{18}F]FDG were predominantly low. Although we found no statistically significant difference between the two tracers, our findings revealed that [^{11}C]acetate accumulates more than [^{18}F]FDG in a large fraction of sCAEBV lymph node lesions.

The SUV_{max} of [^{11}C]acetate PET/CT did not differ significantly between active and inactive cases, although its distribution was wider in active than in inactive cases. Mild-to-moderate [^{11}C]acetate uptake has been reported in reactive lymphadenopathy [19], suggesting that uptake in inactive cases primarily reflects physiological accumulation, whereas uptake in active cases reflects both physiological and pathological accumulation. The coexistence of these two mechanisms likely accounts for the wider SUV_{max} distribution in active cases and may explain why no statistically significant difference was observed between the two groups. The SUV_{max} of [^{18}F]FDG PET/CT in lymph node lesions likewise did not differ significantly between active and inactive cases, with almost all lesions showing only mild tracer accumulation. This is consistent with a previous report, in which mild-to-moderate [^{18}F]FDG uptake in lymph nodes was occasionally observed in patients with sCAEBV, whereas intense uptake suggested transformation to lymphoma [4].

On visual assessment of maximum intensity projection images, cardiac [^{11}C]acetate uptake was more evident in case 5 than in the inactive cases. Previous studies have shown that the myocardial clearance rate of [^{11}C]acetate reflects myocardial oxygen consumption, and that myocardial damage from ischemic, dilated, or alcoholic cardiomyopathy reduces this clearance rate [25–27]. In case 5, mild cardiac dilation and pericardial effusion suggestive of cardiac dysfunction were observed, and the patient died 295 days after the PET/CT scan. Although no overt cardiac lesions were detected and pathological confirmation of cardiac sCAEBV involvement was not available, the enhanced cardiac [^{11}C]acetate uptake in case 5 may reflect a reduced clearance rate possibly associated with underlying cardiac dysfunction; whether this represents true sCAEBV-related cardiac involvement or a comorbid condition cannot be determined from the available data.

Central nervous system (CNS) involvement was also observed in two patients in this cohort: Case 3 (inactive) presented with mild intellectual disability, and Case 5 (active) showed multiple old lacunar infarctions on prior imaging. Although neither tracer revealed abnormal cerebral uptake in our study—partly because physiologic [^{18}F]FDG uptake in the brain masks subtle pathological accumulation and [^{11}C]acetate cerebral uptake is generally low—CNS involvement in sCAEBV has been reported to occur in 7–20% of cases and is associated with poor prognosis [4,15]. The presence of cerebrovascular lesions in Case 5, who subsequently died of disease progression, may reflect chronic vasculopathy or microinfarction related to EBV-induced systemic inflammation. Dedicated brain MRI rather than whole-body PET/CT remains the modality of choice for evaluating CNS involvement in sCAEBV; however, the inability of either tracer to detect CNS lesions on whole-body imaging represents a limitation of dual-tracer PET/CT in this disease.

This study has several limitations inherent to pilot studies of ultra-rare diseases. First, the sample size was small due to the extremely low incidence of sCAEBV, which is reported predominantly in Japan and eastern Asia. This limits the statistical power, and larger multicenter studies are required to confirm and extend our preliminary findings. Second, the correlation between [^{11}C]acetate uptake and treatment response was not assessed, as patients had undergone various treatments. Third, the correlations between [^{11}C]acetate uptake and disease complications, EBV-infected cell phenotype, and prognosis could not be fully evaluated. In addition, for [^{18}F]FDG PET/CT we used adjacent adipose tissue rather than the liver as the visual background reference; because current [^{18}F]FDG lymphoma assessment guidelines rely on the liver (and mediastinal blood pool) as the reference standard, this approach may have overestimated lymph node positivity for [^{18}F]FDG, and the nodal [^{18}F]FDG findings should be interpreted with this caveat. Furthermore, we assessed only SUV-based metrics; volumetric PET parameters such as metabolic tumor volume and total lesion glycolysis were not evaluated. This omission may introduce a risk of selection bias and limit the comprehensiveness of the metabolic characterization, underscoring the need for further research into volumetric and other advanced PET metrics beyond SUV to more fully define the utility of [^{11}C]acetate and [^{18}F]FDG PET/CT in sCAEBV. Finally, because this study was conducted at a single tertiary care center in Japan, studies in additional populations are required to confirm the generalizability of our findings. Despite these limitations, our findings provide proof-of-concept evidence for the potential utility of [^{11}C]acetate PET/CT in sCAEBV assessment, warranting future prospective investigations.

In conclusion, we found significant correlations between liver enzyme levels and the LSR on [^{11}C]acetate PET/CT, and the LSR on [^{11}C]acetate was numerically higher in active cases than in inactive cases, though this difference did not reach statistical significance. Therefore, [^{11}C]acetate PET/CT might be useful for assessing patients with sCAEBV.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/diagnostics16132071/s1>, Supplementary Methods S1. Radiosynthesis and quality control of [^{11}C]acetate. Reference [28] is cited in the supplementary materials.

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Abbreviations

ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
EBV	Epstein–Barr virus
EBV-T/NK LPD	EBV-positive T- or NK-cell lymphoproliferative disease
[¹⁸ F]FDG	2-deoxy-2-[¹⁸ F]fluoro-D-glucose
LDH	Lactate dehydrogenase
LSR	Liver-to-spleen ratio
MIP	Maximum intensity projection
NK	Natural killer
PET/CT	Positron emission tomography/computed tomography
sCAEBV	Systemic chronic active Epstein–Barr virus disease
SUVmax/mean	Maximum/mean standardized uptake value
TNF- α	Tumor necrosis factor-alpha
VOI	Volume of interest
WHO	World Health Organization
γ GTP	Gamma-glutamyl transpeptidase

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