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Case Report

Spontaneous intraspinal and intracranial subdural hematoma in a highly active antiretroviral therapy-naïve-patient with HIV

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Abstract Spontaneous intraspinal and intracranial subdural hematomas (SDHs) are rare, potentially life-threatening complications. We present the case of a 38-year-old highly active antiretroviral therapy-naïve human immunodeficiency virus (HIV)-positive patient who developed simultaneous intraspinal and intracranial SDHs without hemostatic abnormalities. The patient presented with acute lower limb weakness and back pain, and later developed neurological symptoms, including diplopia and incontinence. This case highlights the importance of considering SDHs in HIV-positive patients presenting with neurological symptoms, and underscores the need for prompt diagnosis and multidisciplinary management.

Key words: HIV, Hemorrhage, Hematoma, subdural

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INTRODUCTION

Spinal subdural hematoma (sSDH) is a rare but serious medical condition with a high risk of significant neurological impairment. sSDHs typically occur in patients with identifiable risk factors, such as anticoagulation therapy, coagulation disorders, lumbar puncture, postoperative complications, and vascular malformations.[1] Despite these known associations, the precise mechanisms underlying sSDH development remain poorly understood. Nevertheless, several hypotheses have been proposed, including platelet dysfunction, brain atrophy, vasculopathy, and intracranial hypotension, that may contribute to the pathogenesis of spontaneous sSDHs.[2]

Human immunodeficiency virus (HIV) infection is associated with a wide range of neurological complications, including symptoms caused the virus itself, opportunistic infections, or antiretroviral therapy.[3] Although subdural hematomas (SDHs) are relatively rare in HIV-positive patients, they have a higher incidence in patients with thrombocytopenia or coagulopathy. One recent review on thrombocytopenia in people living with HIV/acquired immune deficiency syndrome (AIDS) reported that the overall prevalence of thrombocytopenia ranged from 4.5% to 26.2%.[4] Some studies have presented sporadic reports of intracranial SDHs in HIV-positive individuals, often linked to hemostatic

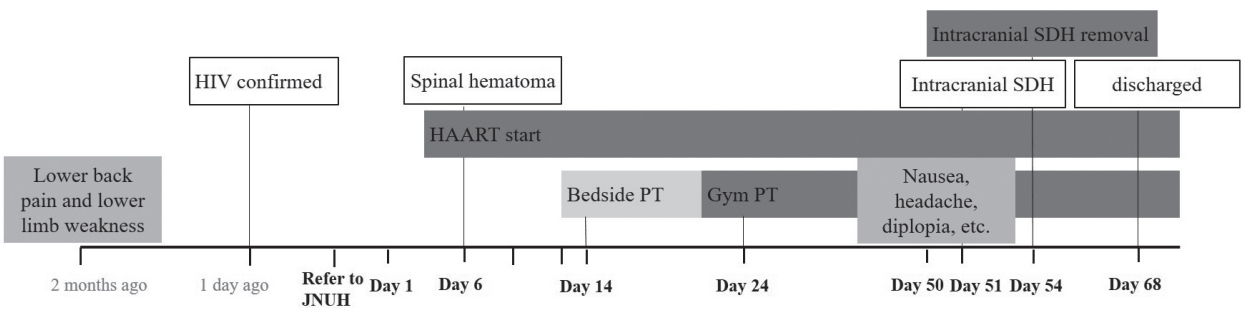
abnormalities or opportunistic infections.[2,5,6] However, the occurrence of spontaneous sSDH in HIV-positive patients without hemostatic abnormalities, particularly in highly active antiretroviral therapy (HAART)-naïve patients, has not been extensively investigated.

One notable study from Korea reported a case of spontaneous intracranial and spinal SDH in an elderly patient with a history of lumbar fracture.[7] This case highlights the rarity of spontaneous SDHs without common risk factors. To the best of our knowledge, no prior cases of spontaneous spinal SDH have been reported in HAART-naïve HIV-positive patients without any identifiable hemostatic abnormalities have been reported. This report aims to fill this gap by presenting such a case, highlighting the clinical presentation, diagnostic challenges, and therapeutic strategies, and comparing our findings with the existing literature. This study was approved by the Institutional Review Board (IRB) of Jeju National University Hospital (IRB No. 2023-02-003).

CASE REPORT

A 38-year-old HIV-positive man was referred to our hospital. He had developed lower limb weakness and upper back pain 2 months prior to admission, with no history of trauma or acupuncture. On admission, he had a Glasgow coma scale score of 15, blood pressure of 105/65 mmHg, pulse rate of 65/min, respiration rate of 14 breaths/min, and body temperature of 36.4℃. His white blood cell count was 27,000/mm^3 (normal range, 4,000-10,000), platelet count of 206,000/mm^3 (normal range, 150,000-400,000), prothrombin time international normalized ratio test of 1.09% (normal range, 0.88-1.20), and an activated partial thromboplastin clotting time of 35 seconds (normal range, 20.0-36.0) (Fig. 1). The patient's CD4+ T-cell count and HIV RNA load were 32/mm^3 and 779,000 copies/mm^3, respectively.

HAART (bictegravir, emtricitabine, tenofovir, alafenamide; one tablet every 24 hours) was initiated on the 3rd day of admission. He had no opportunistic infectious diseases and was not a drug abuser or heavy alcoholic. Despite treat-



	Normal	Admission days	Day 6	Day 12	Day 18	Day 22	Day 32	Day 54
WBC (/μL)	4,000–10,000	27,000	2300	6400	7400	4400	2900	3700
Platelet (10^3/μL)	150–450	206	127	233	237	339	254	237
PT INR	0.88–1.20	1.09						1.17
aPTT (seconds)	20.0–36.0	35						33
CD4$^+$ T cell (%)	28.4–56.4							
CD4$^+$ T cell (μL)	N/A	32	63	124	172	168	129	265
HIV RNA load (copies/mm^3)	Not detected	779,000		2020	489	525	299	256

Figure 1. Timeline of the clinical course and laboratory findings of the patient with HIV and spinal subdural hemorrhage without trauma. SDH: subdural hemorrhage, HIV: human immunodeficiency virus, HAART: highly active antiretroviral therapy, PT: physical therapy, JNUH: Jeju National University Hospital, WBC: white blood cell counts, PT INR: prothrombin time international normalized ratio test, aPTT: activated partial thromboplastin time, N/A: not applicable.

ment and conservative management of AIDS, the patient was unable to ambulate. On the 9th day of admission, a comprehensive diagnostic workup was conducted, including cervical and thoracic spine and brain magnetic resonance imaging (MRI). The patient was diagnosed with an intraspinal subacute hematoma in the early subacute stage (Fig. 2A, B), and brain MRI revealed a right hygroma without hemorrhage. A neurosurgical consultation was conducted due to the SDH extending from T10 to L2. After evaluating the findings that indicated minimal cord compression, it was determined that the patient could be managed conservatively, and surgical intervention was deemed unnecessary. Consequently, the patient started rehabilitation therapy on the 14th day after admission without undergoing surgery (Table 1). His back pain and lower extremity muscle strength resolved after 1 month, and his lower extremity muscle strength improved from grossly poor to fair. The manual muscle test improved muscle strength from grossly

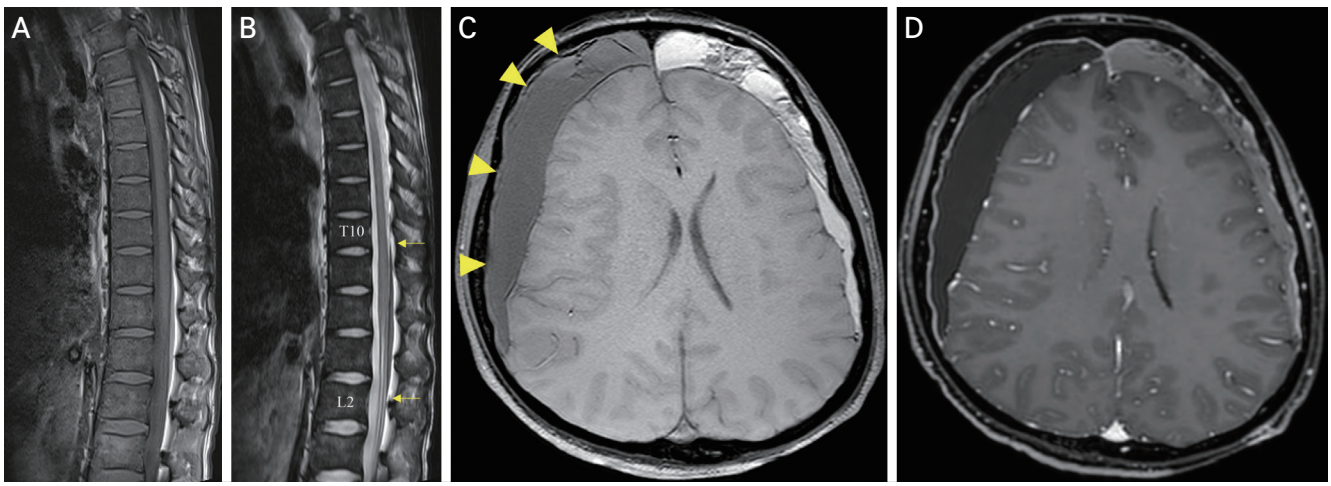


Figure 2. Thoracic and lumbar spine magnetic resonance imaging (MRI) results of the patient. (A) The T1-weighted thoracic and lumbar spine MRI showing a high signal intensity. (B) T2-weighted thoracic and lumbar spine MRI showing a low signal intensity (yellow arrows). (C) T1-weighted brain MRI showing a high signal intensity (yellow arrowheads). (D) T2-weighted brain MRI showing a low signal intensity.

Table 1. Comparison of manual muscle test and functional status of spinal hematoma in a patient with HIV at the initial consultation with the department of rehabilitation and at discharge

	14th day of admission	68th day of admission
Manual muscle test	Right/left	Right/left
Hip flexion	Fair-/poor+	Fair/fair
Hip extension	Fair-/poor+	Fair/fair
Knee flexion	Fair-/poor+	Fair/fair
Knee extension	Fair-/poor+	Fair/fair
Ankle plantar flexion	Fair-/poor	Fair/fair
Ankle dorsiflexion	Fair-/poor	Fair/fair
Functional examination		
Sitting balance (static/dynamic)	Poor/trace	Good/good
Standing balance (static/dynamic)	Trace/trace	Good/fair
Functional ambulation category	0	1
Modified Rankin scale	5	3

HIV: human immunodeficiency virus.

poor to fair. Dynamic sitting balance improved from trace to good grade, dynamic standing balance improved from trace to fair grade, and gait function increased from functional ambulation category 0 to 1. Dependence in daily life improved from 5 to 3 on the modified Rankin scale. In addition, the patient had no opportunistic infectious diseases.

On the 49th day of admission, the patient presented with nausea and headache without any history of trauma. Subsequently, on the 50th day of admission, he developed sudden diplopia and urinary and stool incontinence without limb weakness. On the 51st day of admission, an intracranial left SDH with late subacute stage and midline shift to left side without trauma was observed on brain MRI (Fig. 2C, D). He underwent emergency evacuation of the intracranial SDH, with no neurological sequelae. On the 60th day of admission, his CD4+ T cell count and HIV RNA load were 265/mm^3 and 256 copies/mm^3, respectively. The patient recovered and was discharged on the 68th day of admission (Table 1).

DISCUSSION

Spontaneous sSDH is extremely rare, particularly in HAART-naïve HIV patients without hemostatic abnormalities. Nevertheless, this case emphasizes the importance of considering atypical presentations in patients with no common risk factors. Initially, the patient presented with persistent weakness, which prompted further diagnostic evaluation. During this workup, spinal SDH was unexpectedly diagnosed, highlighting the need for comprehensive diagnostic evaluations such as spinal MRI, even for non-specific symptoms in HIV-positive patients. Given the rarity of spinal SDH, and the generally non-specific nature of its initial presentation, it is crucial for clinicians to maintain a high index of suspicion and ensure thorough evaluation of HIV patients presenting with such symptoms. The patient's muscle strength and gait performance improved without neurological complications following management of intraspinal and intracranial SDH.

The patient was diagnosed through imaging and neurological examinations, prompted by persistent lower-limb weakness, despite systemic improvement from medication. Cases reported in the literature have both similarities and differences. For example, one case reported by Kim et al.[7] in Korea involved an elderly patient with a history of lumbar fracture who developed both intracranial and intraspinal SDHs. This patient had predisposing factors, including advanced age and previous spinal injury, which likely contributed to hematoma formation. In contrast, our patient was younger, had no history of trauma or lumbar puncture, and had significantly different clinical and demographic characteristics. Other cases of concomitant intracranial and spinal SDH have shown good clinical outcomes after conservative treatment. However, prompt decompression should be considered when neurological deficits progress rapidly.[6,8]

Severe thrombocytopenia in HIV patients has frequently been cited as a major risk factor for SDH formation. Kutty et al.[5] previously described a case of intracranial SDH in an HIV-positive patient with marked thrombocytopenia. Unlike this latter case, our patient did not exhibit any significant hematological abnormalities, indicating that alternative mechanisms might be at play. Additionally, the decision to perform conservative management of intraspinal SDH over surgical intervention is based on several clinical considerations. First, the patient's neurological deficits did not progress rapidly at the time of initial presentation. Furthermore, his lower limb weakness and back pain did not show acute deterioration, necessitating immediate surgical decompression. Moreover, his hematological parameters, including platelet count and coagulation profiles, were within normal limits, indicating that immediate surgical intervention was not urgently required. The absence of significant hemostatic abnormalities reduced the immediate risk of further hemorrhage, allowing for conservative management.

The existing literature supports the efficacy of conservative treatment in certain cases of spontaneous sSDH, particularly when neurological symptoms are stable or improved. For example, Kim et al.[7] reported a case in which conservative treatment was successfully employed for simultaneous intracranial and spinal SDHs without rapid progression of

neurological deficits. Similarly, Kutty et al.[5] presented the resolution of intracranial SDH using conservative measures in an HIV-positive patient. In our patient, gradual improvements in muscle strength and neurological function through intensive physiotherapy and HAART further validated the decision to pursue conservative management. Over the course of rehabilitation, significant improvements were observed in the patients' motor function and balance, indicating successful recovery without the need for surgical intervention. Additionally, the patient's improved immune status following HAART initiation contributed to the conservative approach, as the enhancement of his immune function likely aided in the spontaneous resolution of the hematomas.

From a diagnostic perspective, healthcare providers should maintain a high index of suspicion for spontaneous SDH in HIV patients presenting with neurological symptoms, even in the absence of common risk factors. Early imaging studies, such as MRI, are crucial for a timely diagnosis. This case underscores the importance of performing a comprehensive neurological examination and SDH in the differential diagnosis of HIV patients with new or worsening neurological deficits. In addition, the rehabilitation outcomes in our patient further highlight the effectiveness of intensive physiotherapy in improving functional status post-SDH.[9] In our case, the patient's lower extremity muscle strength and gait performance improved markedly over the course of rehabilitation, underscoring the importance of a multidisciplinary treatment approach.

This study has several limitations. First, we did not evaluate any differential diagnoses of other bleeding disorders, such as clotting factor deficiencies or genetic disorders. This could provide additional insights into potential underlying causes. Further research is required to better understand the underlying mechanisms and develop tailored management strategies for similar cases. Second, the patient's response to HAART and its impact on the resolution of SDH were not systematically monitored using specific biomarkers, such as interleukin-5 or vascular endothelial growth factor A levels, which may have offered further understanding of the hematoma absorption process.[10] Third, while the patients' neurological and functional recovery was documented, a more detailed and standardized assessment of cognitive function, mood, and quality of life post-recovery would have provided a more comprehensive view of the long-term outcomes. Finally, the rarity of such cases in the literature makes it difficult to draw broad conclusions, emphasizing the need for more extensive case series or multicenter studies to validate these observations.

In conclusion, this case report illustrates the complexity and unique challenges of managing spontaneous SDHs in HAART-naïve HIV patients. Overall, this case emphasizes the need for a prompt diagnosis, early initiation of antiretroviral therapy, and a comprehensive treatment approach to optimize patient outcomes. Further studies are warranted to elucidate the underlying mechanisms and improve management strategies for spontaneous SDH in this patient population.

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