

Spontaneous chronic subdural hematomas in human immunodeficiency virus-infected patients with normal platelet count and no appreciable brain atrophy: Two case reports and review of literature

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Abstract

Background: Chronic subdural hematomas (CSDHs) usually occur in elderly patients following minor head trauma. Their occurrence is usually linked to cerebral atrophy secondary to alcohol, old age, or human immunodeficiency virus (HIV) infection. Spontaneous CSDHs have also been documented but are rare. They are usually caused by coagulopathies and various pathologies resulting in intracranial hypotension.

Cases: We have observed a number of spontaneous CSDHs in HIV patients with normal platelet counts and no appreciable cerebral atrophy possibly caused by platelet dysfunction, hence we report about two such cases. To the best of our knowledge, no such cases have been reported in literature before.

Conclusion: It is important to include CSDHs in the differential diagnosis of HIV patients presenting with neurological deficits even without a history of trauma.

Key Words: Human immunodeficiency virus coagulopathy, spontaneous chronic subdural hematoma, subdural bleeds

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BACKGROUND

The incidence of chronic subdural hematoma (CSDH) is increasing.^[11] This may be attributed to an improvement in diagnosis due to wide availability of brain imaging and also the increased number of population living beyond 65 years of age.^[11] The advent of human immunodeficiency virus (HIV) infection has greatly altered a lot of pathologies regarding etiological factors as well as presentations. CSDHs have not been spared. CSDH is considered a benign disease of the elderly, with 80% of the cases occurring in those over 40 years and the peak being the eighth decade.^[1,11,12]

However, with a high prevalence of HIV/acquired immune deficiency syndrome (AIDS) in Sub-Saharan Africa, a

significant proportion of younger patients have been noted to develop CSDHs from our observation. This may be attributed to cerebral atrophy occurring at a younger age and also thrombocytopenia,^[1,12] all due to retroviral

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infection. To the best of our knowledge, no case reports have been documented of spontaneous CSDH in retroviral infection with normal platelet counts and without any appreciable brain atrophy. We, hereby, present two such cases which we hypothesize that dysfunctional platelets secondary to HIV/AIDS predisposed to the bleed.

CLINICAL PRESENTATIONS

Case 1

A 35-year-old HIV-positive male patient on TenoLam-N (tenofovir, lamivudine, and nevirapine) with CD4 T-cell lymphocyte count of 198 cells/ μ L (normal 410–1590) presents with a 3-day history of headaches associated with vomiting and a day’s history of confusion. He also had an isolated episode of diarrhea but had no history of; trauma, taking alcohol, nonsteroidal anti-inflammatories, herbal medicines, or any other medications other than his antiretrovirals. He works as a school teacher and was otherwise relatively well before this.

On examination, he was confused, having a Glasgow Comma Scale of 14/15. He had bilateral parotid fullness and was apyrexial with no neck stiffness. He had right-sided hyperreflexia with positive Babinski sign. There were no signs of recent trauma. The rest of the examination was normal. Electrolytes and full blood count were normal. Figure 1 shows images and Table 1 shows blood results.

He was taken for emergency evacuation of the hematoma via an enlarged burr hole. The operation and recovery were uneventful. The confusion improved and the patient was discharged fully consciousness on day 6.

Case 2

A 42-year-old HIV-positive female patient on TenoLam-N with a CD4 T-cell lymphocyte count of 49 cells/ μ L

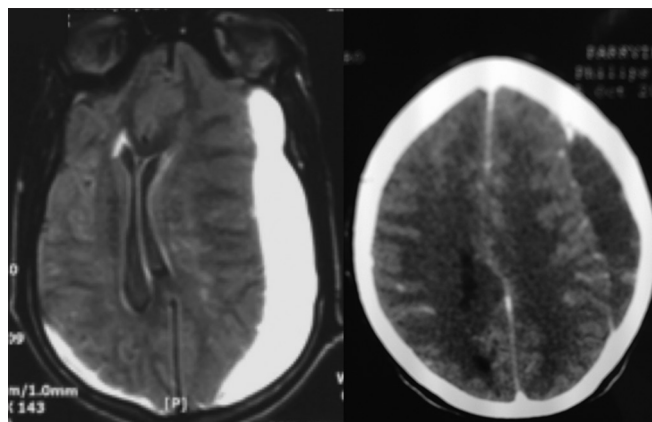


Figure 1: Magnetic resonance imaging (left, Case 1) showed large left-sided fronto-parieto-occipital chronic subdural hematoma, with a small contralateral occipital collection. Computer tomography (right, Case 2) shows left-sided parietal bleed. Both show significant midline shift with compression of the right lateral ventricle but no appreciable brain atrophy

presented with a month’s history of gradually worsening headaches and 2-day history of confusion with right-sided weakness. Her baseline CD4 T-cell lymphocyte count had been 240 cells/ μ L before commencing antiretrovirals about 1 year ago. She had no history of taking anticoagulants, nonsteroidal anti-inflammatories, herbal medicines, alcohol use, or any other medications other than her antiretrovirals. No history of trauma could be elicited even after recovery.

On examination, she was drowsy and confused. Her left pupil was dilated with sluggish response to light. She had right-sided hemiparesis with no facial involvement. She had no physical signs of trauma. The rest of the examination was noncontributory. The full blood count, urea, and electrolytes were unremarkable. Figure 1 and Table 1 show computer tomography scan and blood results, respectively.

The patient was taken for emergency burr hole evacuation of the CSDH. Operation and recovery were uneventful, and the patient was discharged on day 5. On follow-up, at 2 weeks, the level of consciousness was 15/15. She was referred to the physicians for possible HIV treatment failure on account of her diminishing CD4 cell count.

DISCUSSION

The most common cause of CSDH is trauma, in a predisposed brain. Two-thirds of CSDH patients recall some type of minor trauma.^[1] Sometimes, the trauma is so trivial that it may be unnoticed, hence the difficulty to confirm with certainty that a subdural bleed is actually spontaneous as opposed to trauma related.

However, spontaneous CSDHs are recognized in literature.^[9] The known causes are coagulopathies and also cerebrospinal fluid (CSF) shunting.^[9,11] Procedures that reduce the volume of CSF abruptly such as lumbar puncture,^[6] spinal anesthesia,^[5] and transventricular approaches for tumor surgery^[8] have also been noted to cause spontaneous CSDHs.

In our two patients, there was no history of trauma that could be elicited. The patients were of sober habits, hence alcoholism could not be considered as a possible cause. The imaging did not show any appreciable cerebral atrophy

Table 1: Relevant blood results for the two cases

Test	Case 1	Case 2	Normal range
Platelets	227 $\times 10^9$ /L	149 $\times 10^9$ /L	150-450 $\times 10^9$ /L
INR	1.56	1.0	0.8-1.2
PT (s)	18.0	10.5	10.5-13.5
APTT (s)	27.7	28.3	26-36
Urea	5.2	4.1 mmol/L	2-6.7 mmol/L
Creatinine	62	75 mmol/L	48-131 mmol/L

INR: International normalized ratio, PT: Prothrombin time, APTT: Activated prothrombin time

that could have predisposed our patients to subdural bleeds. The patients were quite young to fit the generally accepted age group of chronic subdural bleeds. Some rarer documented causes include also straining^[4] and vascular malformations.^[11] Our patients, however, did not have any history or imaging to suggest these causes. Systemic hypotension resulting in intracranial hypotension can also occur in dehydration,^[2] which can be secondary to severe diarrhea. Our first patient had an episode of diarrhea, but it was not significant since it was just an isolated episode and the patient was not dehydrated on admission.

Cases of spontaneous CSDH without an identifiable cause have been documented in literature.^[13] In our cases, all the other documented causes seem not to fit the presentation of our patients. We postulate that these CSDHs occurred as a complication of the coagulopathy in retroviral infection, particularly with low CD4 counts. This is confirmed in the first case with increased thromboplastin time and international normalized rate. There is need to evaluate further the spontaneous occurrence of CSDH in HIV/AIDS. We postulate that the coagulopathy was linked to retroviral infection.

HIV infection predisposes to both hemorrhage and thrombosis, and the mechanisms are believed to be multifactorial with some still to be elucidated ranging from blood vessel vasculitis to true blood coagulopathies.

Although relatively rare, vasculitides are important complications of all stages of HIV infection. The vasculitides seen in HIV-infected patients represent the full spectrum of disease, ranging from that caused by specific infective agents to nonspecific vasculitis, and affecting large, medium, and small vessels. A wide range of pathogenic mechanisms are involved in the development of necrotizing vasculitis, many of which remain to be elucidated, but all of which are linked by an underlying inflammation of the vessel wall.

Liver dysfunction, caused by the viral infection itself or the effects of antiretrovirals, may lead to coagulation factor deficiencies. Further, the presence of antibodies is believed to coat the platelets interfering with their function. In some cases, high immunoglobulin proteins are believed to interfere with coagulation factors leading to hemorrhage.^[3,7,10]

CONCLUSION

HIV has many neurological manifestations. Given the hematological effects of retroviral infection, CSDHs need to be considered early in the differentials of an HIV patient even if history of trauma cannot be elicited. Brain imaging should be done early in HIV-positive patients with symptoms suggestive of CSDH. This will allow prompt diagnosis, early appropriate intervention with generally good prognosis in retroviral infected patients without significant comorbidity. Further studies need to be done with regards to bleeding abnormalities in HIV infection.

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Conflicts of interest

There are no conflicts of interest.

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