



## Human immunodeficiency virus infection presenting as a fatal case of Guillain-Barré syndrome on a background of diabetes mellitus

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### ABSTRACT

**Background:** Guillain-Barré syndrome (GBS), a post-infective acute polyneuropathy, which occurs rarely among Africans, has been associated with HIV infection and less commonly with diabetes mellitus. **Aim:** The article documents a case of GBS occurring on the setting of HIV infection on a background of diabetes mellitus. **Findings:** A 47 years old man presented with features of inability to walk, “pins and needles” sensation in the lower limbs, progressive lower limb weakness of 3 days duration and later on admission involving the upper limbs and, finally respiratory distress leading to his death on the 3rd day of hospital admission. He had an antecedent history of a diarrheal illness 3 weeks prior to his admission, and was treated at a private hospital without any complication. He was observed to have concomitant HIV infection and diabetes mellitus. He was not previously known to have any of these diseases and initially diagnosed as having acute diabetic neuropathy. **Conclusion:** GBS can occur in the setting of HIV infection on a background of DM and may be associated with a poor prognosis. There is a need to have a high index of suspicion in making a diagnosis of GBS in diabetic patients when it occurs concomitantly with HIV infection.

**Key words:** HIV, Guillain-Barré Syndrome, diabetes mellitus, paralysis, presenting, progressive weakness

### INTRODUCTION

Guillain-Barré syndrome (GBS) includes a rapid-onset of clinical syndromes with a common pathophysiological basis; an acute inflammatory polyneuropathy with an autoimmune cause

characterized by a progressive flaccid paralysis with areflexia and a wide range of motor, sensory and autonomic symptoms in the absence of identifiable causes of genetic, metabolic, or toxic origin.<sup>[1-4]</sup> While in many people GBS is preceded 3-6 weeks prior to onset



of neurological symptoms by an infection (upper respiratory tract infection or diarrhea), in a smaller proportion of individuals, no apparent cause may be found.<sup>[5,6]</sup> Initial symptoms are limb numbness and tingling and pain, alone or in combination followed by progressive leg and arm weakness over several hours to 14 days to reach a maximum.<sup>[5]</sup> GBS is usually self-limiting and associated with a good prognosis in most patients, but the following are associated with a worse prognosis; older patients, severe neurological deficits at the beginning, cranial nerves involvement, patients requiring ventilation, and axonal lesion patterns in the central nervous system.<sup>[6]</sup>

The pathogenesis of GBS is still undergoing study, but this disease has an important autoimmune component with cellular and humoral activity where antibodies probably are predominantly implicated in association with demyelination of peripheral nerves.<sup>[4,5]</sup> A usual preceding history of a viral or bacterial infection such as Herpes Simplex virus (HSV), Varicella-Zoster (VZV), cytomegalovirus (CMV), Epstein-Barr virus (EBV), Hepatitis B and C virus, *Mycoplasma Pneumoniae*, *Haemophilus influenzae* and *Campylobacter jejuni* have all been implicated in GBS.<sup>[6]</sup> Human immunodeficiency virus (HIV) infection has also been associated with GBS either during sero-conversion or in association with central nervous system (CNS) infection in the chronic phase.<sup>[7-10]</sup> Also, GBS has been reported as a presentation in association with diabetes mellitus in the setting of diabetic neuropathy, diabetic coma or recent elevation of blood sugar levels.<sup>[11-14]</sup> But no report of GBS in Nigeria occurring in the setting of both HIV and diabetes mellitus has been observed.

Against this backdrop, we wish to report a case of clinically diagnosed GBS in a patient diagnosed with HIV infection and diabetes mellitus.

## CASE PRESENTATION

Mr AG was a 47 years old trader who presented with a week history of excessive passage of urine, progressive lower limb weakness associated with inability to walk three days prior to presentation. He also complained of lower limb "pins and needles" sensation. Three weeks prior to onset of present illness, he had been ill with 2 days of diarrhea and hypotension, and

was treated at a private hospital with IV fluids and oral antibiotics (name not given) for a few days without any immediate complication. There was no history of trauma to the head or back, headaches and no previous diagnosis of hypertension or diabetes mellitus. He was married to one wife with four children. On examination, he was not in any obvious distress, temperature was 37.4°C, and not pale. His respiratory rate was 18 breaths/minute, pulse rate was 84/minutes (regular), blood pressure 130/84mmHg and, oxygen saturation was 97% at room air. He had no gibbus or tenderness over the spine, but had diffuse tenderness over both lower limbs and was unable to stand unaided but could sit up. He had paraplegia associated with hyporeflexia.

The random blood sugar done was 278mg/dl. An initial diagnosis of an acute diabetic neuropathy was made and he was commenced on insulin therapy (regular insulin 8 units three times daily). On further investigation, he was discovered to be reactive and confirmed to be positive for HIV. The wife was negative for HIV. He had no previous diagnosis for HIV infection.

On the second day of admission, his temperature rose to 40.5°C and antibiotics (IV ceftriaxone 1gm two times daily) was commenced. A full blood count revealed: packed cell volume (PCV) was 36%, white blood cell (WBC) count was  $5.8 \times 10^9/L$  (neutrophils 76%, lymphocytes 24%), urinalysis revealed glycosuria (++);serum electrolytes, urea and creatinine, liver enzymes, serum lipid profile and, prostate specific antigen (PSA) were normal. He was observed to have upper limb weakness. A diagnosis of possible Guillain-Barré syndrome was then considered after a review of the history and presenting signs. His blood sugar level rose to 296mg/dl.

On the morning of the third day, he progressively became unresponsive to calls; temperature was 40.7°C. He soon after developed respiratory distress and cardiac arrest as he was being prepared for transfer to the ICU. He was confirmed dead after an unsuccessful resuscitative effort.

## DISCUSSION

Guillain-Barré syndrome is not common in Eastern Nigeria as reported cases in Nigeria have all been from Western Nigeria.<sup>[15]</sup> GBS

rarely occurs in Nigeria as only 14 cases were recorded over a 17 year period in the study done in Ile-Ife, Nigeria<sup>[15]</sup> making it easy to miss at presentation. This patient presented with an elevated blood sugar level and peripheral neuropathy which made the initial attending physician make a diagnosis of diabetic neuropathy, that was similar to the experience of Jin HY *et al.*<sup>[11]</sup> A later confirmation of HIV infection in the patient, and a review of the history including the antecedent diarrheal illness 3 weeks prior to admission and symptom presentation of rapid progressive limb weakness and paresthesia, similar with GBS presentation<sup>[5,6]</sup> made us consider a clinical diagnosis of Guillain-Barré syndrome. Our diagnosis of GBS was made on clinical grounds in line with its typical presentation<sup>[5,6]</sup> as we were unable to demonstrate the classical cerebrospinal fluid findings of albumino-cytology dissociation.<sup>[6]</sup> However, albumino-cytology dissociation is only positive in about 80% of patients with GBS making it imperative to consider other clinical signs.<sup>[4-6]</sup> The observed very high fever in the last two days of admission in the patient may suggest an autonomic component in the presentation of GBS as demonstrated by González-Suárez *et al.*<sup>[6]</sup>

Though GBS has been associated with HIV but not commonly with diabetes mellitus.<sup>[7-14]</sup> It is yet to be reported in a patient with concomitant HIV infection and diabetes mellitus as we observed. HIV is thought to be directly associated with the onset of GBS at sero-conversion or in the chronic phase.<sup>[7-10]</sup> We were unable to determine at what point in the infection GBS occurred in our patient, but without a prior diagnosis of HIV infection and the fact that the couple was discordant for HIV infection may suggest an acute HIV infection in our patient. The observation of diabetes mellitus was recent, a few days before hospital admission. It is thought that an antecedent viral infection, likely the diarrheal illness 3 weeks before, may be pathophysiologically linked to the GBS and onset of diabetes mellitus as observed by Flax H *et al.*<sup>[12]</sup> GBS is usually associated with a good prognosis.<sup>[3-6]</sup> HIV infection or diabetes mellitus occurring singly have not been associated with a different prognostic outcome as has been widely reported even among African populations.<sup>[8-13]</sup> The rate of progression of the disease in our patient was rather rapid with the outcome resulting in death. We believe that perhaps the combined existence of HIV infection and

diabetes mellitus will explain the rapid progression of GBS in our patient and combined with the late recognition of the disease may be responsible for the fatal outcome of the disease.

## CONCLUSION

GBS has not been reported in the eastern part of Nigeria in association with HIV infection or DM. GBS as a presentation of HIV infection has been widely reported but it is rarely associated with diabetes mellitus. Diagnosis of GBS was made clinically. GBS is also associated with a good prognostic outcome; however, a combined occurrence of HIV infection and diabetes mellitus as was the case in our patient may be associated with a poor prognosis. There is a need to have a high index of suspicion in making a diagnosis of GBS in diabetic patients when it occurs concomitantly with HIV infection.

## REFERENCES

1. Van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain-Barré Syndrome. *Lancet Neurol* 2008;7:939–950.
2. Vucic S, Kiernan MC, Cornblath DR. Guillain-Barré syndrome: An update. *J Clin Neurosci* 2009;16:733–741.
3. Tellería-Díaz A, Calzada-Sierra DJ. Síndrome de Guillain-Barré. *Rev Neurol* 2002;34:966–976.
4. Asbury AK, Arnason BGW, Karp HR, McFarlin DF. Criteria for diagnosis of Guillain-Barré syndrome. *Ann Neurol* 1978;3:565–566.
5. Yuki N, Hartung HP. Guillain-Barré syndrome. *N Engl J Med* 2012;366:2294-2304.
6. González-Suárez I, Sanz-Gallego, Rodríguez de Rivera FJ, Arpa J. Guillain-Barré Syndrome: Natural history and prognostic factors: a retrospective review of 106 cases. *BMC Neurology* 2013;13:95.
7. Iglesias-González IM, Dorta-Contreras AJ, Padilla-Docal B, Victoria-García M, Junco-Calzadilla R. Neuroimmunological response in HIV-associated Guillain-Barre syndrome: A case report. *World Journal of Neuroscience* 2012;2:32-35.
8. de Castro G, Bastos PG, Martinez R, de Castro Figueiredo JF. Episodes of Guillain-Barré syndrome associated with the acute phase of HIV-1 infection and with recurrence of viremia. *Arq Neuropsiquiatr* 2006;64:606-608.
9. Thornton CA, Latif AS, Emmanuel JC. Guillain-Barré syndrome associated with human

immunodeficiency virus infection in Zimbabwe. *Neurology* 1991;41:812-815.

10. Hagberg L, Malmvall BE, Svennerholm L, Alestig, Norkrans G. Guillain-Barré syndrome as an early manifestation of HIV central nervous system infection. *Scand J Infect Dis* 1986; 18:591-592.

11. Jin HY, Lee KA, Kim SY, Park JH, Baek HS, Park TS. A case of diabetic neuropathy combined with Guillain-Barre Syndrome. *Korean J Intern Med* 2010;25:217–220.

12. Flax H, Matthews DR. Diabetes associated with Guillain-Barre syndrome. *Diabetes Res.* 1990;14:47-50.

13. Roman-Filip C, Rociu C, Filip D, Poreanu M. Guillain-Barre syndrome in a patient with diabetic coma as initial manifestation. *Romanian J Neurol* 2009;8:128-131.

14. Fujiwara S, Oshika H, Motoki K, Kubo K, Ryujin Y, Shinozaki M, Hano T, Nishio I. Diabetic ketoacidosis associated with Guillain-Barré syndrome with autonomic dysfunction. *Intern Med* 2000;39:495-498.

15. Sunmonu TA, Komolafe MA, Adewuya A, Olugbodi AA. Clinically diagnosed Guillain-Barre syndrome in Ile-Ife, Nigeria. *West Afr J Med* 2008;27:167-70.

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**Conflict of Interest:** None declared

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