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Guillain barre syndrome in HIV patient : A case report

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ABSTRACT

Guillain Barre Syndrome (GBS) is an autoimmune acute polyneuropathy affecting the peripheral nervous system in which immune system mistargets host nerve tissue through molecular mimicry. This acute monophasic paralysing illness is usually provoked by a preceding infection.

INTRODUCTION

Guillain Barre Syndrome (GBS) is an autoimmune acute polyneuropathy affecting the peripheral nervous system in which immune system mistargets host nerve tissue through molecular mimicry. This acute monophasic paralysing illness is usually provoked by a preceding infection.

HIV associated GBS has been reported since 1985.GBS typically occurs early in HIV infection even at zero conversion before developing AIDS [1]. HIV infection causes a dysfunction in immune regulation resulting in an autoimmune attack on neural myelin sheaths. Additional neural damage from HIV-1 infection may be caused by neurotoxins produced by infected or activated macrophages or monocytes or by toxic products of HIV-1 itself. Direct invasion of nerves by HIV has also been postulated.

CASE REPORT

42 year old male patient started fever with vomiting which was relieved by self antipyretic medications. He was a chronic alcoholic and an occasional smoker with a history of pneumonia & chickenpox in the past. He was apparently well with no comorbidities. He started complaining of limb pain initially below the knee. He developed numbness of hands and feet followed by weakness of bilateral lower limbs. The weakness then progressed to proxymal and distal upper limbs. The patient was unable to get up from the bed and he had difficulty in taking food & holding objects. He was admitted in a local hospital on 10/10/13.Nerve conduction velocity (NCV) study was carried out which showed radical neuropathy with absent 'F' waves in the lower limbs. Patient was diagnosed to have GBS and serology showed HIV (ELISA) positive. He had one dose of IVIG 0.4 mg/kg (20mg) and was referred to a super speciality tertiary care hospital for further management. Initial lab results shown in table 1.Patient had hyponatremia corrected with added salt in the diet. He was started on Nitrofurantoin 100mg TID in view of urinary tract infection and Gabapentin 100mg BD for peripheral neuropathy. Nerve conduction study (NCS) showed bilateral lower limb motor axonal neuropathy. A physical medicine and rehabilitation consultation was sought which advised for daily physiotherapy. Full body physiotherapy was recommended with gait training, Hamstring +quadriceps strengthening exercises and Ardmore physical therapy (ARDM) to all 4 limbs. He was also given sit ups in chair and motor re-education to ankle and foot dorsiflexors. Another 4 doses of IVIG 0.4mg/kg (20mg) was administered. He started on antiretroviral drug Anzavir-r (Atazanavir sulphate 300mg+Ritonavir 100mg with Tenofovir + Emtricitabin). Patient made a steady recovery and became clinically & hemodynamically stable at the time of discharge. He was able to ambulate with assistance and was advised to continue physiotherapy. Now proximal muscles of lower limbs have less than 3 power and both feet have less than 3 powers for foot and ankle dorsiflexors.

DISCUSSION

- Neurologic manifestations of acute HIV infection

include aseptic meningitis, encephalitis, myelopathy, various peripheral neuropathies and rarely AIDP or GBS [2] .HIV associated GBS can be associated with more frequent recurrent episodes than GBS alone. GBS is found to be an early manifestation of HIV Infections .In a prospective study of 32 consecutive patients with GBS 27 tested positive for HIV [3].

- The cardinal clinical features are progressive symmetric muscle weakness accompanied by absent or depressed deep tendon reflexes. Weakness usually starts in the legs and varies from mild difficulty with walking to nearly complete paralysis of all extremity, facial, respiratory and bulbar muscles. Pain typically located in back and extremities can be a presenting feature. Parasthesia in hands and feet accompany the weakness in more than 80% of patients.

- For long term management of neuropathic pain *Tricyclic antidepressants, Tramadol, gabapentin, carbamazepine or pregabalin* can be given. Both paroxysmal hypertension and orthostatic hypotension are frequent due to autonomic dysfunction. Episodes of severe hypertension can be treated with *labetolol, esmolol or nitroprusside*.

- The main modalities of GBS treatment are *plasmapheresis and IVIG*. Plasmapheresis costs nearly 62,000(for 3 sessions) where IVIG costs Rs.80, 000 for 1 dose (20mg). Plasmapheresis is less costly than IVIG.

- *Plasma Exchange* can remove circulating antibodies and compliment soluble biologic respond modifiers. *IVIG* provide antiidiotypic antibodies modulating expressions and function of Fc receptors, activation of complement and production of cytokines, interfere with activation and effector function of T and B cells.

- The acute phase Rehabilitation include an individualized program of gentle strengthening involving isometric, isotonic, isokinetic, manual resistive and progressive resistive exercises. Rehabilitation emphasizes proper limb positioning, posture and orthotics.GBS is a disease having good prognosis for more than 80% patients assuring complete recovery within few months to a year.

- The characteristic CSF finding in GBS is one of albuminocytologic dissociation namely an elevated CSF protein level with a normal WBC count. Therefore pleocytosis in a patient whose clinical history is consistent with GBS should prompt an evaluation for HIV infection.

- It is important for physicians to be aware of this relationship and to obtain a detailed history at the time of presentation focusing on potential risk factors for HIV infection.HIV testing should be conducted in patients who have risk factors for HIV infection, evidence of generalized lymphadenopathy on physical examination or leukocytosis on CSF analysis.

- Our patient clinical improvement following treatment with IVIG supports earlier reports of HIV positive patients who improved after IVIG therapy [4].

- Early initiation of highly active antiretroviral therapy (HAART) irrespective of CD4 count in patients with HIV and any target organ damage due to HIV is mandatory, in this patient HAART was initiated as soon as possible after initial treatment with IVIG.

- An increased risk of renal failure for people with HIV has been associated with a low CD4 cell count, a high viral load and treatment with some antiretroviral drugs, especially *tenofovir* and the protease inhibitors *atazanavir, indinavir and lopinavir*. Data from a large study into the safety of anti-HIV drugs suggest that the antiretrovirals *tenofovir* (*Viread*, also in *Truvada, Atripla and Eviplera*), *atazanavir (Reyataz) and lopinavir/ritonavir (Kaletra)* each have an independent association with a decline in kidney function. Regular monitoring of kidney function is recommended in patients taking antiretroviral drugs and dosage adjustment should be done as per the creatinine clearance.[8][9]

- Prophylaxis against opportunistic infections should be administered to all HIV-infected patients with a CD4 count of <200 cells/μL or a history of oral thrush.

- HAART and HIV-related medications (opportunistic infection prophylaxis) can have various drug interactions, resulting in altered drug levels and potential adverse effects. Regular monitoring of serum potassium is recommended to avoid hyperkalemia in patients on trimethoprim, losartan and

Table 1

Test	Result	Reference Range
WBC	9340 cells/μL	4400-11300 cells/μL
Absolute lymphocyte count	622 cells/ μL	1662-5370 cells/μL
T lymphocytes (CD3+)	86.7%	55-82%
T lymphocytes (CD3+) absolute count	539 cells/μL	1072-3890 cells/μL
Helper/inducer T lymphocytes(CD3*/CD4*) %	8.1%	27-57 %
Helper/inducer T lymphocytes(CD3*/CD4*)	50 cells/μL	562-2692 cells/μL

propranolol.

- In addition, initiation of HAART in a newly-diagnosed HIV patient may allow for activation of an underlying opportunistic infection (i.e. cryptococcosis, tuberculosis or PCP) secondary to an immune reconstitution inflammatory syndrome (IRIS) resulting in acute respiratory failure and other conditions that necessitate intensive care.

- As clinical outcome of this type of patients depends on prompt use of multidrug therapy with zero drug interactions, a clinical pharmacist's role is very important in helping the physician to achieve the desired therapeutic targets.

CONCLUSION

The two major treatment modalities under disease modifying therapy viz. *plasmapheresis* and *IVIG* are found to be equivalent in improving outcome [5].The choice between plasma exchange and IVIG is dependent on local availability, patient preferences, risk factors and contraindications. Small volume plasma exchange can be used with equal efficacy due to constraints. Low side effect profile, ease of administration, reduction in length of hospital stay and low cost of procedures make IVIG more preferable over plasmapheresis. Treatment shortens the time to walk independently by 40-50%.In the absence of disease modifying treatment most patients show continued progression for upto 2 weeks, followed by a plateau phase of 2 weeks and then recovery of function over several weeks to months.

Even with treatment approximately 5-10% of patients have prolonged course with very delayed and incomplete recovery and 5 % die despite intensive care. Poor prognostic factors include age over 40 years, history of preceding diarrhoeal illness, requiring ventilator support, high anti-Gm I titre and poor upper limb muscle strength strength[6][7].

This case highlights the importance of having a high degree of suspicion for HIV infection in patients who present with GBS.

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