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Human Immunodeficiency virus and Guillain 'Barre' Syndrome in intensive care unit patients

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SUMMARY

Among 155 medical admissions to the intensive care unit during the period 1989 to 1990, 16 patients had Guillain-'Barre' Syndrome (GBS), five of whom ere HIV positive. Out of the five cases, three had manifested *herpes zoster* and one had TB. The impact of HIV infection o GBS is discussed.

INTRODUCTION

The global picture of tuberculosis (TB) appears to be changing dramatically for the worse as a result of the human immunodeficiency virus (HIV) pandemic. there is evidence that most of the increase in the number of TB cases attributable to HIV results from reactivation as cell-mediated immunity declines.[1]

further, HIV infection s related to immunological status. Guillain-'Barre' Syndrome (GBS) is believed to have an immunological basis. Therefore, it is reasonable to suppose that in the presence of HIV infection, the course of GBS may be changed.

Out of a total of 155 medical patients admitted to the Intensive Care Units (ICUs) of the Harare Group of Teaching Hospitals (Harare and Parirenyatwa Teaching Hospitals), during a two-year period (1989 to 1990), with respiratory failure, 16 (9,7 pc) had GBS. This study was performed to find out if there exists an association between HIV infection and GBS.

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MATERIALS AND METHODS

The case records of 16 patients admitted to ICU requiring mechanical ventilation for severe GBS were reviewed. The information was obtained from the patient's notes and the ICU 24-hour chart.

The following information was extracted from the records: 1. Age; 2. Sex; 3. History of antecedent infection; 4. Duration of stay in ICU; 5. Complications; 6. HIV status; and 7. Outcome.

RESULTS

The ages of the patients ranged between 13 and 51 years with a mean age of 31 years and standard deviation of 12,9. There was a predominance of females, 11 out of 16 as noted in Table I. Five (31 pc) of the patients were positive for HIV infection.

Table I: Age and Sex Distribution

Age Group	No of Patients Male	Female
10–19 Yrs	1	2
20–29	1	5
30–39	0	4
40–49	0	0
50–59	3	0
Total	5	11

Table II: HIV Status

Antecedent Infection	HIV Status Positive	Negative	Not know
Herpes Zoster	3		
Tuberculosis	1		
Nil	1	8	3

Table III: Duration Of Stay in ICU

Duration	No. of Patients Discharged	Died
0–20 days	2	3
21–40 days	6	2
41–60 days	0	0
61–80 days	2	1

Average duration was 31 days.

Three (60 pc) of the five HIV positive patients had *herpes zoster* while one had TB prior to the onset of GBS (Table II). The majority of the survivors (six out

of ten) spent between 21 to 40 days in the ICU (Table III). The average stay in ICU (including the patients who died and those who were discharged) was 312 days. Ten of the 16 patients were discharged from ICU, while six died, resulting in a mortality rate of —37,5 pc. Three of the patients who died developed cardiovascular complications, while one had tracheo-oesophageal fistula (Table IV).

Table IV: Complications and outcome

Complication	Outcome	No. of Patients/pc
Nil	Discharged	10 (62,5)
Cardiac arrhythmias	Died	3
Tracheo oesophageal fistula	Died	1 (37,5)
Not documented Died	1	

DISCUSSION

Guillain 'Barre' Syndrome, also known as ascending polyneuritis or acute inflammatory demyelinating polyneuropathy, was described by Guillain, Barre and Strohl in 1916. However, the severe variant of the syndrome had been described by Landry in 1859.

In two thirds of the cases, an infectious episode, often an upper respiratory tract infection, usually precedes the onset of the neurologic syndrome by one to three weeks subsiding before the onset of the syndrome.[2] GBS has also been reported to follow influenza vaccination, surgery and is thought to have an immunological basis.[3] Osuntokun and Agbebi concluded that antecedent infection may play a major role in producing antigenic stimulation of antibody against nervous tissue.[4]

The clinical criteria for this diagnosis of GBS include progressive motor weakness affecting at least two limbs and loss of reflexes.[2] The muscle weakness usually starts in the legs, progresses upwards at a variable speed but reaches its peak before the fourth week. Milk sensory deficits may occur and cranial nerve involvement is sometimes seen. In all of the patients in this study, the diagnosis of GBS was made by the admitting physician before the patient was referred and admitted to ICU and the usual criteria were used.

In the series reported here, there of the five HIV positive patients had *herpes zoster* prior to the onset of GBS. In the light of this finding, it is fair to suggest that *herpes zoster* virus and HIV infection should be included in the group of viral infections associated with Guillain-Barre Syndrome. Prospective studies involving larger series are required to confirm the association.

Perronne *et al*[5] reported that in their series of 50 patients with HIV infection, in 23 of them the testing had been prompted by the presence of *herpes zoster.*

Varicella in patients infected with HIV is more severe in its manifestation and often fatal.[6] It is interesting to note that in our series two patients who died out of the five patients who had GBS and HIV infection had *herpes zoster* prior to the manifestation of the syndrome.

Clinical Course: GBS is a monophasic polyneuropathy from which many patients ultimately recover satisfactorily.[7] In the benign form of the disease, recovery is excellent and is complete in 80 to 90 pc of patients while a small subset of patients have residual disability.[2] Osuntokun and Agbebi concluded that the good prognosis makes it imperative to control respiration if bulbar or thoracic paralysis is present.[4] the severe end of the clinical spectrum of the Guillain 'Barre' Syndrome is characterised by nearly complete paralysis of somatic muscles including the respiratory muscles. All of the sixteen patients had the severe form of the syndrome. A decreasing vital capacity together with bulbar weakness, with difficulty in swallowing and coughing, is a dangerous combination leading to aspiration and acute respiratory failure. Artificial ventilation becomes necessary when the vital capacity shows a progressive decrease to less than 1 litre of predicted value.[8]

The vital capacity was not determined in our patients prior to artificial ventilation. The decision to ventilate was made on clinical grounds and poor arterial blood gas results.

Second to respiratory failure, the commonest cause of death i GBS is cardiac arrhythmias.[8] This was noted in three of the six patients who died. The arrhythmias are frequently associated with autonomic abnormalities. Demyelination of some of the fibres of the vagus may occur.[9]

Early in the disease, sinus tachycardia and persistent hypertension are frequently observed. Badycardia and hypotension are seen particularly following vagal stimulation from tracheal suction.[8] The mechanism of death in a patient with excessive or paroxysmal autonomic activity is not well understood.

It is possible that brain stem dysfunction is responsible for the abnormalities, that this leads to alterations in cardiac output or rhythm and thereby results in sudden death.[10]

The milestones of recovery are, return of respiratory muscle function and spontaneous breathing. A vital capacity greater than 1 to 1,5 litres is usually required before satisfactory spontaneous ventilation is achieved.

At this point, abdominal paradoxical movement disappears, signifying the return of adequate diaphragmatic activity. The sequence for weaning the patient from artificial ventilation is commenced guided by satisfactory arterial blood gas results.

In this series, ten of the sixteen patients recovered and were discharged from the ICU after an average stay of four and half weeks.

CONCLUSION

Out of 16 patients with GBS, three had *herpes zoster* and HIV infection. There appears to be evidence that HIV, in itself, is associated with GBS and TB and worsens the prognosis.

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