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The Rehabilitation of Patients with Severe Guillain-Barré Syndrome

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Summary

Twenty four of more than 800 patients admitted to our centre presented with a severe Guillain-Barré Syndrome. The length of hospitalisation, duration of illness, treatment, and the rehabilitation course of these patients are discussed. Suggestions are made concerning the necessity to obtain maximal rehabilitation for these patients.

Key words: *Guillain-Barré Syndrome, Rehabilitation; Outcome.*

Introduction

The Guillain-Barré Syndrome (GBS) is a polyradiculitis resulting from acute demyelination of the peripheral nervous system and/or the cranial nerves. The exact etiology is unknown with conflicting evidence supporting viral, bacterial, or autoimmune causes (Merrit, 1973; Zweiman, 1983). The GBS usually follows a non-specific viral infection of the upper respiratory or alimentary systems. It can occur after immunizations (Kaplan *et al.*, 1980) or in conjunction with oncologic disease; men and women being equally affected.

The clinical picture of paralysis progresses symmetrically beginning in the lower limbs and ascending to and sometimes including the cranial nerves. Distal sensory changes or ataxia (Fisher, 1956) may occur accompanied by muscular or articular pain (Rapper and Shahabi, 1984). Additional findings include: pyrexia, tachycardia, hypertension, elevated hematocrit, and elevated spinal cord fluid protein. Nerve conduction studies show a marked slowing of conduction velocity in all peripheral nerves with the lower extremities more affected (Brown, 1984). Complete recovery usually occurs in most of the cases, but in a small minority, the paralysis can be permanent (Adams and Victor, 1981). We present 24 cases of the GBS who were treated in our rehabilitation department.

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Patients

From 1974 until 1986, more than 800 new spinal cord injured patients and more than 150 patients with non-traumatic spinal cord lesions were admitted to our centre. Only 24 of these patients, 15 men and 9 women, were diagnosed as having the GBS and were admitted following initial hospitalisation in various neurological or internal medicine departments. All in-patient and out-patient records of these patients were reviewed. Fourteen patients were of European origin, 9 patients were of Mediterranean origin and 1 patient was an Arab. The time interval from the onset of the febrile episode until the appearance of the paralysis ranged between 3 hours to 3 weeks. Table I shows the distribution of these prodromal illnesses. Fifty per cent of all the patients suffered from an upper respiratory infection prior to the onset of the GBS. Table II shows the

Table I Infectious diseases preceding the onset of the Guillain-Barré Syndrome

Disease	Number of patients	%
Upper respiratory infections (flu, tonsilitis, pharyngitis)	12	50
Infectious Mononucleosis	1	4
Measles or German measles	2	8
None	9	38

Table II Seasonal distribution of onset of Guillain-Barré Syndrome

Months	Number of patients	%
January – March	13	54
April – June	5	28
July – September	5	28
October – December	0	0

distribution of the seasonal occurrence of the GBS with most of the cases occurring in the winter-spring period. Table III summarises the clinical picture of this patient cohort. All patients suffered from paresthesiae, but only one third complained of pain or respiratory difficulty. Fifty per cent had ascending

Table III Summary of clinical findings

Clinical findings	Number of patients	%
Parasthesias	24	100
Weakness	12	50
Cranial nerve paralysis	12	50
Assending paralysis	23	96
Respiratory difficulties	9	38
Pain	8	33
Incontinence	5	21

Table IV Summary of diagnostic laboratory data

Laboratory findings	Number of patients	%
Elevated spinal fluid protein	15	63
Lymphocytosis	1	4
Elevated herpes virus titer	2	8

paralysis with cranial nerves involved. Table IV shows the laboratory data pertinent to the diagnosis of the GBS. The possible presence of a compromised immunologic system in 8 patients prior to the onset of the GBS is noted by the presence of arthritis, previous GBS in 1 patient, allergies, etc. Pharmacological therapy included: 21 patients who received corticosteroid therapy, 2 patients vitamins, and one patient had plasmaphoresis (Richard 1985; Greenwood 1984). In all the patients there were no apparent benefits as measured by dramatic, sudden improvement in their physical condition. Table V shows the duration of hospitalisation in our rehabilitation centre. Most patients were discharged within 3 months with the remainder being hospitalised up to 1 year. The final neurological outcome is summarised in Table VI.

Table V Duration of hospitalisation

Hospitalisation	Number of patients	%
Less than 3 months	13	54
3 – 6 months	8	33
6 – 12 months	3	13

Table VI Final neurological picture

Final status	Number of patients	%
Tri or tetraplegia	4	16
Para or monoplegia	9	38
Ataxia	4	16
Recovered	7	30

Discussion

As is shown in our data, 50% of the patients had a prodromal upper respiratory tract infection shortly before the onset of the GBS (Gortner 1984; Roman 1978). Since most of these viral infections occur during the winter and the spring, it is little wonder that the incidence of GBS was greatest during this time of the year. In our patient cohort there was no apparent relationship between the GBS and the presence of any other diseases previous to the onset of the GBS. In the 8 patients whose medical history revealed a previous infection (poliomyelitis, previous GBS, arthritis, etc.) we found nothing to indicate any special relationship i.e. abnormal blood count, increased gamma globulin on plasma elec-

tro-phoresis etc. between the GBS and the other disease entity. The number of our patients treated given any type of specific pharmacological treatment for the GBS (vitamins, plasmaphoresis) is much too small for any significant conclusions to be drawn.

The clinical course of our patients was typical of the classical variety of the GBS with distal paresthesiae and ascending paralysis. In only 1 patient was the paralysis descending. In the 30% of the patients with respiratory compromise these problems proved to be of life-threatening proportions, requiring tracheostomy in all cases. All of these 8 patients were transferred to us after having been weaned from the respirator in the intensive respiratory care facility. The tracheostomies were closed by gradually diminishing the size of the tracheostomy tube over a 2 week period and then removing the tube entirely. The 8% of our cases with either hypertension or cardiac arrhythmias correlates well with the reported literature. High spinal fluid protein levels were only found in 63% of the cases.

The vast majority of the patients who contract GBS recover spontaneously with little or no residual neurological findings. Patients with GBS who show minimal recovery after the onset of the paralytic symptoms or whose recovery is delayed for several weeks after the onset of the clinical picture, are referred to the rehabilitation centre for long term hospitalisation. This selection factor may well account for the fact that most of our patient cohort did not show a complete recovery. Our patient cohort is very small, but it seems as if the younger patients achieve a more complete recovery. In those patients with respiratory problems the initial problem is one of maintenance of respiratory support, and with the onset of recovery eventual weaning from respirators and then closure of the tracheostomies if possible. The patient is first weaned from oxygen to room air while still on the respirator. Then weaning from the respirator is done by gradually decreasing the number of respirations per minute which trigger the respirator, thus giving the patient the opportunity to breathe a few breaths on his own unassisted. This is gradually performed and then the respirator is discontinued. Total weaning required is between 1 to 3 months. Only then, depending upon the neurological picture and its rate of change, are the patients ready to proceed with the physical rehabilitation programme including ambulation training and training in activities of daily living. Obviously the duration of hospitalisation is a function of severity of the disease. As most of our patients, 17 out of 24, were discharged showing some residual neurological findings, the selectivity of these severely handicapped individuals dictates an extended communal rehabilitation approach. Adaptation of the home environment is sometimes necessary and provisions for long term follow-up with vocational evaluation, training and career changes are necessary. As the recovery of neurological function can be prolonged, post-discharge neurological examination is desired.

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