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SOME OBSERVATIONS ON THE GULLAIN - BARRE SYNDROME IN SURABAYA

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Since the eradication of poliomyelitis, Guillain-Barre syndrome (GBS) remains one of the most crippling neurological diseases. Although many scholars have written about it, treatment remains symptomatic with a mortality rate of 25% (5). The opinions are divided among neurologist regarding the value of treatment with corticosteroids and in one of the recent textbooks it is still written, that the value of treatment with corticosteroids is uncertain.

GBS is frequent in tropical countries, but reports from these countries on this disease are seldom, probably because of the hopelessness of this syndrome.

This is a report of 67 patients seen in Surabaya at the division of neurology, University of Airlangga, school of medicine and the private offices of the author. In this report the author further presents the result of a new form of treatment used in University Hospital in Surabaya.

MATERIALS AND METHODS

As criteria for selection the author used the criteria as mentioned by Mc. Farlan: (4)

1. The paralytic illness may follow a non-specific infection, but there is no preceding or accompanying illness of a type known or thought to be associated with polyradiculo-

neuropathy.

2. Sensory impairment may occur, but is less severe than motor impairment.
3. Diffuse lower motor neuron paresis is usually rapid in onset, often ascending, usually symmetrical, may be proximal and distal or both.
4. There are ten or fewer white blood cells per cubic millimeter in the cerebro-spinal fluid (CSF).
5. There is protein of 60 mg in CSF or higher.

Between the first of January 1957 until 1st January 1973, 67 patients were seen at the out-patient department of the University Hospital or at the private offices of the author. They were admitted in the University Hospital or the private hospital but were all examined by the author, except during the years of 1962 and 1963 when the author was in the United States.

In all patients, besides a neurological examination a complete blood count, differential count, platelet count and urinalysis was performed. Since the diagnosis was based on the CFS findings, in all patients a lumbar puncture was performed. Patients with a positive serologic reaction for syphilis were excluded from this study.

Clinical Findings.

The age and sex of the patients were as follows:

Table I: Age and sex from patients with Guillain-Barre syndrome in Surabaya

Age	male	female	total number
0 – 10	7	6	13
11 – 20	9	9	18
21 – 30	11	12	23
31 – 40	4	3	7
41 – 50	3	2	5
51 – 60	0	1	1
All ages	34	33	67

Seasonal incidence can be seen in table II

Table II: Seasonal incidence from patients with GBS

Month	No cases	Season	Total
January	13	rainy	37
February	12		
March	9		
April	3		
May	2	dry	8
June	1		
July	1		
August	2		
September	2	rainy	22
October	4		
November	9		
December	9		
			67

63 patients stated that they had an infection before the paralysis started. The variety of illnesses preceding the polyradiculoneuropathy may be seen in table III.

Table III: Preceding illness

Type of illness	No. of cases
Upper respiratory infection	34
Gastroenteritis	12
Fatigue, headache	9
Muscle aches	8
None	4
	67

Initial complaints.

The patients complaints at the first interview can be seen in table IV.

Table IV. Initial complaints of patients with GBS.

Symptom	legs	legs and arms	arms	Total number
Paresthesias and weakness	17	12	7	36
Neck pain	1			2
Motor weakness	12	9	5	29
			Total	67

Most patients were seen within 14 days after onset of the disease.

Neurological signs

The findings on the initial neurologic examination are given in Table V.

Table V. Initial neurological findings in GBS

Sign	facial	arm/legs	neck	respiratory	trig.
Weakness	333	67	37	12	77
Tonus — lowered		67	37	—	
Neg. reflex tendon		67			
Anaesthesia—glove—stocking		67			

Spinal fluid findings.

Since the diagnosis is based on the CSF findings, the spinal fluid was examined by all patients. The findings can be seen in table VI.

Table VI. Cerebro spinal fluid findings in 67 patients with GBS

Signs	60–80 mg%	80–100 mg%	more than 100 mg%
Protein — total	29	27	11
Cell — count	always lower than 10/mm ³ , mostly lymphocytes		
Colloidal gold all abnormal:	first zone	8	
	second zone	52	
	third zone	7	

Time relations:

In 63 patients the illness developed 7–21 days after a preceding illness. The peak of the illness was reached in 6–30 days and most patients began to recover 14–17 days after the peak in 56 pat. The typical course therefore was progression for two–three weeks, stabilization for two and then recovery. This is the rule in the patients not treated with intrathecal dexamethasone. (1, 6)

Therapy:

As noted before, the opinions on steroid treatment are divided. (3) The author noted however,

that the corticosteroids usually were given orally or Intramuscularly. The author therefore decided to use the intrathecal route, because by giving it intrathecal, the local concentration of steroid in the spinal, roots should be increased during several hours (similar to in spinal anaesthesia). Most investigators now regard the GBS as a primary demyelinating disorder similar to allergic encephalomyelitis. (7). The usefulness of steroids in the last disease is long known.

Since 1971, the author offered this form of therapy to all patients with GBS. 11 patients agreed: 5 mg of dexamethasone was given every day

Table VII. Comparison of patients treated with dexametasone – intrathecal and without.

	dexamethasone intrathecal	only support. therapy
Average days in hosp.	14 – 30 days	60 – 90 days
Neurological residual	only paraesthesias (leg and arm)	leg weakness in 60%
Protein value in CSF	lower than 30 mg %	when dismissed in 60% still above 60 mg %

by lumbar-injection intrathecally during ten days. When the protein value in the spinal fluid was lower than 30 mg%, the injections were stopped, otherwise a second series of injections was given. The results of this form of therapy may be seen in table VII.

DISCUSSION

As seen in the first table, there were as many males as females. Mc. Farland (4) found the same ratio, but Wiederholt (6) twice as many male persons. The age distribution showed that GBS was most frequent in the first, second and third decade.

The seasonal incidence showed, that GBS was frequent in the rainy season. The table on preceding illness, showed that an upper respiratory infection, was one of the most frequent disease preceding the neuropathy.

The initial complaints were mostly paresthesias combined with motor weakness (53.7%). 29 patients (43.3%) complained of weakness alone. In the majority of the patients the complaints were centered on the legs.

The neurological findings on examination showed, that the common picture was a flaccid paralysis of all extremities, with sensibility disturbances at the distal ends.

The spinal fluid showed a disturbance of the total protein value and of the colloidal gold.

Although the opinions are divided regarding the use of corticosteroids in GBS, it become apparent from a review of the literature, that:

1. GBS is regarded by many scholars as a primary demyelinating disorder similar to allergic encephalomyelitis and experimental allergic neuritis. (EAN (7)).
2. Steroids inhibit the appearance of lesion in allergic encephalomyelitis.
3. Corticosteroids were used orally or intramuscular in GBS, while the rational way of giving steroids locally in slight dosage should be the intrathecal route, similar like in spinal

anaesthesia.

The results in 11 patients treated with intrathecal dexametasone showed, that the number of days spent in the hospital was shortened, because the beginning of the recovery state in GBS was accelerated, a fact which was already suggested by Wiederholt. (6). Table VII proved too, that the neurological residual signs were mild in the patients treated with intrathecal dexametasone compared with those who received only supportive therapy. All the protein values were definitely low in the intrathecal treated cases.

7 patients, who took dexametasone orally, but did not show any improvement, were switched to the intrathecal form of therapy, with much success.

In the 11 patients treated with intrathecal injections no side effects were encountered. It should however be emphasized, that when giving a drug intrathecally, the strict precautions of sterility should be taken and that disposable needles and syringes should be used.

Although Fishman (2) mentioned objections, when giving intrathecal therapy for the purpose of reaching the cerebrum or optic nerves, it seems that from the results mentioned above, that intrathecal therapy with the objective of the spinal roots has still a place in the neurological armamentarium.

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MIGRAINE, RELATED HEADACHES AND PSYCHOTROPIC MEDICATION

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These observations result from previous experience with headache problems in a headache clinic in Switzerland, and from brief experience with headache problems in Malaysia, for little more than one year (by far not enough time for a statistical survey).

I have read two recent papers from this region: The Diagnosis and Management of Headache, by Loong Si Chin, and Complicated Migraine, by T.G. Loh and J.C. Chawla. I would like to comment upon a few concepts from these articles which may be representative of the interpretation of headache problems at an academic level in this region.

"Complicated migraine", say Loh and Chawla, "is not well known.....it is associated with protean ...neurological features...".

Migraine has been described by laymen from ancient Summerian and Egyptian times, and by Western medical authors (as far as their writings extend) from the second century A.D., when the term was first introduced as "hemicrania" by Galenos of Pergamon, the father of Western medicine.

The best known variant of migraine is that hemicrania which is further characterized by preceding scintillating scotoma and concomitant nausea: the megrim, or sick headache, which had been described by Galenos and, as "heterocrania", by his precursor, Aretaios, wherefore it is often known as "classical migraine". In 1878, Galezowski gave it the self-explanatory name of ophthalmic migraine.

What Anglo-Saxon authors call "hemiplegic migraine", that is, a paroxysmal headache with transient features akin to some or all of the symptoms of stroke, had been named "migraine

accompagnee" by Charcot, the founder of modern neurology, in 1887. The majority of cases of "complicated migraine," can rightly be classified as migraine accompagnee.

The very rare condition of paroxysmal periodic headache with extraocular palsy was first identified by P. J. Moebius in 1884, and it was named "ophthalmoplegic migraine" by Charcot in 1890.

The migraine variant with the highest frequency of attacks which occur once to many times a day within bouts or "clusters" of one to two months is known in medical literature by at least twelve different names (such as: Horton's Neuralgia, Migranous Neuralgia, Erythroprosopalgia, etc.). In the last decades, American and Scandinavian authors have increasingly preferred "cluster headache", again a self-explanatory term.

On a sound historical basis, migraine and its variants can be classified as follows: common migraine, ophthalmic migraine, migraine accompagnee, ophthalmoplegic migraine, and cluster headache. There are other terms which have found less ready acceptance, such as dysphrenic migraine, characteristic migraine, basilar artery migraine. There is another, more comprehensive classification: the list of the ad hoc committee on classification of headache of the National Institute of Neurological Diseases and Blindness of the U.S.A., 1962, which includes facial migraine under a separate heading. However, the simple classification as proposed above takes care of the practically important forms of migraine, and allows us to get beyond the impression of "protean neurological features" and to sort out the "(infinite) variety of migraine" (Friedman) in terms compatible with contemporary literature.

In Anglo-Saxon literature, "Tension Headache"