Outbreak of Guillain-Barré syndrome associated with water pollution

NAIF ABDULLAH SLIMAN

British Medical Journal, 1978, **1**, 751-752

Summary and conclusions

Sixteen cases of Guillain-Barré syndrome occurred in the third week of a diarrhoea epidemic caused by water pollution in El-Sult, Jordan. Of 30 000 people exposed to polluted water, 5000 developed diarrhoea, 74 typhoid, and 30 infectious hepatitis. Thirteen of the 16 patients with Guillain-Barré syndrome had been mildly affected by diarrhoea 8-24 days before the onset of peripheral neuropathy. Paralysis progressed rapidly, reaching a peak in one to five days, and recovery began three to seven days after the start of the most severe symptoms. All but four patients had recovered completely after one year. Rapid progress of paralysis and a delayed interval between maximum weakness and start of recovery were both associated with poor prognosis.

Introduction

In January 1976 an epidemic of diarrhoea occurred in El-Sult, a rural town of 30 000 inhabitants, 30 km west of Amman, Jordan. One week previously (23 December) a routine weekly sample of the drinking water showed heavy contamination with *Escherichia coli* (most probable number >16/100 ml). The drainage system was found to be leaking into the water supply, and within the first week of January 1976 the leakage was stopped.

On 2 January 1976 many patients with acute diarrhoea were notified to the medical authorities. During the first week of January 2546 cases were notified, and by the end of the third week the number had risen to 5000. Presumably many more cases escaped detection because patients either treated themselves or their symptoms were mild. Stool culture was negative except in 16 cases, in which shigellae were grown.

Faculty of Medicine, Jordan University Hospital, Amman, Jordan
NAIF ABDULLAH SLIMAN, MRCP, physician and assistant professor

Three weeks after the onset of the outbreak a typhoid epidemic began, reaching its peak in the fifth week. *Salmonella typhi* was isolated from 74 blood samples in 328 cases investigated. During the fifth week of the diarrhoea epidemic about 30 cases of infectious hepatitis appeared.

During the third week after the diarrhoea outbreak several patients with peripheral neuropathy presented at the El-Sult Hospital. The clinical picture in all cases was identical, which was consistent with Guillain-Barré syndrome. In an attempt to centralise the management of these cases the health authorities decided to have them admitted to the Jordan University Hospital; within a week 16 patients had been admitted, and are described here. No further cases appeared. No cases of poliomyelitis occurred in El-Sult.

Present series

Six of the 16 patients were male and 10 were female (table I). Ages ranged from 3-63 (mean 24.4) years. In all cases a full history was taken and clinical examination performed with particular reference to the nervous system. Investigations included complete blood count, urine analysis (including porphyrins), stool culture, liver function tests, measurement of serum lead concentrations, serum protein electrophoresis, cold agglutination tests for *Mycoplasma pneumoniae*, Paul-Bunnell test, and chest radiography; results were normal in all cases. The cerebrospinal fluid (CSF) was examined on admission and again a few days later if the CSF protein concentration had been normal. Virological studies of serum (two samples at 10-day intervals) and CSF gave negative results.

CLINICAL FINDINGS

Thirteen patients had diarrhoea, which began at the start of the epidemic and lasted for 1-15 days (table I). No patient had severe enough diarrhoea to warrant hospital treatment, and only five needed treatment at home. The interval between the onset of diarrhoea and the start of paralysis was 8-24 days (table I). The three patients who had no diarrhoea were living in El-Sult during the epidemic, and many other members of their families had diarrhoea.

All patients presented with a sudden onset of loss of motor power of varying degree. Progress was rapid, the maximum weakness being reached within one to five days (table I). Decrease of muscle power and tone was symmetrical, varying from partial loss to complete

TABLE I—Clinical details of 16 patients who developed Guillain-Barré syndrome during a diarrhoea epidemic due to water pollution (cases 11, 12, and 14 had no diarrhoea)

Case No	Sex	Age (years)	Days in hospital	Duration of diarrhoea (days)	Interval between start of diarrhoea and onset of paralysis (days)	Time to maximum weakness (days)	Duration of peak paralysis before start of recovery (days)	Time to maximum recovery (days)	Degree of recovery (%)
1	M	18	15	3	10	3	5	106	100
2	M	65	13	10	24	5	3	22	100
3	F	50	13	10	22	2	10	120	100
4	F	13	37	1	9	2	9	365	100
5	F	25	120	1	8	1	27	365	60
6	M	16	10	2	10	5	3	40	100
7	F	23	64	1	9	1	12	365	70
8	F	3	5	15	22	4	10	60	100
9	F	13	17	6	12	4	10	150	100
10	M	12	17	3	9	4	6	60	100
11	F	44	15			5	10	120	100
12	F	15	27			2	8	150	100
13	F	13	55	1	18	2	13	365	80
14	F	18	24			2	6	210	100
15	M	23	40	7	24	3	7	120	100
16	M	50	45	1	8	1	8	365	80

paralysis. The arms were mainly affected in one patient, the legs in six, and the other nine patients were equally affected in both arms and legs. Both proximal and distal muscles were affected.

Superficial reflexes were intact in every patient. All tendon reflexes were absent in 11 patients and present in three. In the remaining two patients, tendon reflexes were present in the arms but absent in the legs in one, and absent only on the left side in the other. The three patients with intact deep reflexes recovered completely. Muscle wasting was seen only in the later stages of the disease in the four patients who did not recover within the observation period (20 months).

TABLE II—Relation between duration of progressive paralysis and time taken for maximum recovery in 16 patients with Guillain-Barré syndrome

No of cases	Duration of progressive paralysis (days)	Rate of recovery (%)	Time to maximum recovery (days)	
			Mean	Range
3	1	70	365	365
5	2	96	242	120-365
2	3	100	113	106-120
3	4	100	90	60-150
3	5	100	60	22-120

None of the patients had cranial nerve lesions on admission, but nine days later one developed bilateral lower motor neurone facial palsy, which resolved within three weeks with corticotrophin injections, though the limbs had not regained full power even after 20 months. All patients complained of pain in the legs and, to a less extent, in the arms, but sensations (including pain, touch, temperature, vibration, and position senses) were intact in all patients. There were no signs of lesions in the central or autonomic nervous system, though most patients complained of hot, sweaty palms.

LABORATORY FINDINGS

Only three tests gave positive results.

Lung function tests showed slight reduction in forced vital capacity and forced expiratory volume in one second, which gradually returned to normal. (The main values were 90% of the mean predicted values.) No one had respiratory distress.

CSF—No cells were found in the CSF, but protein was moderately increased (0.5-1.5 g/l (50-150 mg/100 ml)) in all cases. CSF sugar and electrolyte concentrations were normal and culture grew no pathogens.

Electromyography and nerve conduction velocity—Sampling of either the abductor pollicis brevis or anterior tibial muscles showed no

TABLE III—Effect of duration of interval between peak paralysis and start of recovery on outcome in 16 patients with Guillain-Barré syndrome

No of cases	Interval from time of peak paralysis to start of recovery (days)	Time to recovery (days)	Degree of recovery (%)
5	3-6	88	100
8	7-10	251	97.5
3	11-27	265	70

spontaneous activity, and on volition a few motor units were seen to be polyphasic and M-shaped. Otherwise the muscle-action potential was of normal duration and amplitude (interference pattern was reduced). The motor conduction velocity ranged between 50 and 60 m/s. These changes were compatible with a neural lesion such as demyelination of nerve roots.

PROGRESS AND TREATMENT

Ten patients were chosen randomly for treatment with intramuscular corticotrophin, initially 60 U/day for a week, then 40 U/day and 20 U/day during the second and third weeks respectively. The other six patients were given only symptomatic treatment. Outcome

in both groups was essentially similar. Patients were in hospital for between five and 120 days (table I). Twelve patients were discharged when they could walk unaided. The other four (three female and one male) were still crippled on discharge, though they were improving. Both the duration of progressive paralysis before recovery (table II) and the interval from maximum paralysis to start of recovery (table III) were found to influence prognosis.

Assessment of the patients after one year showed that 12 had recovered completely (table I), while four were still severely disabled, but slowly improving. No patient had relapsed at reassessment 20 months after the onset of the disease. The four severely disabled patients still could not walk without crutches.

Discussion

Since the decline of poliomyelitis the most commonly encountered acute or subacute disabling polyneuropathy has been the Guillain-Barré syndrome.¹ This acute, diffuse post-infective disease of the nervous system affects the spinal roots and peripheral nerves, and occasionally the cranial nerves.² Criteria for diagnosing Guillain-Barré syndrome have been defined by Osler *et al.*³ It may be an inflammatory disorder due to allergy and hypersensitivity, possibly mediated by an unidentified allergen, though some cases might be due to an unidentified virus infection.² Antecedent infection, usually viral and affecting the respiratory tract,⁴ has been identified in more than half the reported cases,⁵ though herpes zoster,⁴ mycoplasmal infections,⁶ glandular fever,⁷ and chickenpox⁸ have also been implicated.

Although Guillain-Barré syndrome may follow gastro-intestinal upsets,⁷⁻⁹ no case has been reported after diarrhoea due to water pollution. All but three patients in the present series suffered from diarrhoea due to water pollution and developed acute polyneuropathy within 8-24 days from the start of the diarrhoea. Paralysis progressed rapidly, reaching its peak within one to five days. Cases reported elsewhere progressed at a much slower rate. After between three and 27 days from the onset of the most severe symptoms (table I) patients started to recover.

Two factors were found to influence prognosis: firstly, the duration of the progress of the paralysis, and, secondly, the interval between peak paralysis and start of recovery. When paralysis progressed rapidly, recovery was slow and often incomplete (table II). This is contrary to the findings of Ravn¹⁰ and Pleasure.¹¹ A short interval between maximum paralysis and start of recovery indicated a better prognosis (table III), which is consistent with the conclusion of Eberle *et al.*¹²

I thank Dr A Kurdi, KHMC, for his help in investigating these patients; Professor S Hilali, of the Faculty of Medicine, Jordan University, for his help in writing this paper; and Dr Colin Ogilvie, of Broadgreen Hospital, Liverpool, for his encouragement and valuable advice.

References

- ¹ British Medical Journal, 1975, **3**, 190.
- ² Brain, W R, *Diseases of the Nervous System*, p 814. London, Oxford University Press, 1969.
- ³ Osler, L D, and Siddell, A D, *New England Journal of Medicine*, 1960, **262**, 964.
- ⁴ Melnick, S C, and Flewett, T H, *Journal of Neurology, Neurosurgery and Psychiatry*, 1964, **27**, 395.
- ⁵ *Recent Advances in Clinical Neurology*, ed W B Mathews, p 267. Edinburgh, Churchill Livingstone, 1975.
- ⁶ Steele, J C, *et al*, *Lancet*, 1969, **2**, 710.
- ⁷ Marshal, J, *Brain*, 1963, **86**, 55.
- ⁸ Melnick, S C, *British Medical Journal*, 1963, **1**, 368.
- ⁹ Campbell, A M G, *Proceedings of the Royal Society of Medicine*, 1958, **51**, 157.
- ¹⁰ Ravn, H, *Acta Neurologica Scandinavica*, 1967, **43**, suppl No 30, p 77.
- ¹¹ Pleasure, D E, Mishner, K C, and Engel, W K, *Science*, 1969, **166**, 524.
- ¹² Eberle, J, *et al*, *Journal of Pediatrics*, 1975, **86**, 356.

(Accepted 5 January 1978)