GUILLAIN-BARRE SYNDROME IN A PATIENT WITH DIABETIC COMA AS INITIAL MANIFESTATION

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ABSTRACT

We report a case of a 39-year-old man, with an acute inflammatory demyelinating polyneuropathy (AIDP), clinically with acute progressive and symmetrical motor weakness of the extremities, weakness of the bulbar and facial muscles. EMG test revealed primary demyelination of peripheric nerves, and CSF examination showed albumino-cytologic dissociation (albumines 2,1 g/l, cells 1/mm³). Patient recovered well after performing 5 consecutive plasma exchange therapy. A particularity in this case, is the diabetic coma, as initial clinic manifestation, followed by Guillain-Barre syndrome, well recovery after plasma exchange. Both diseases had probably the same etiology – an infectious trigger yet not identified.

Key words: Guillain-Barre syndrome, diabetic coma, infectious trigger, plasma exchange

INTRODUCTION

Guillain-Barre syndrome (GBS), is an acquired autoimmune disorder of peripheral nervous system characterized by weakness, usually symmetrical, evolving over a period of several days or more. The disease is characterized by rapidly progressing limb weakness and loss of tendon reflexes caused by impairment of transmission of nerve impulses to the muscles (1). There are several clinical subtypes of Guillain-Barre syndrome, with different pathological and electrophysiological characteristics (2): acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAM), acute sensory neuropathy (ASN), acute panautonomic neuropathy such as Miller Fisher syndrome (MFS), Fisher's syndrome/GBS overlap syndrome (2). Treatment with both intravenous immunoglobuline and plasma exchange reduces the time taken for recovery. Corticosteroids, which are effective in other acute and chronic autoimmune disorders have no effect in patients with GBS.

CASE REPORT

A 39-year-old man without medical antecedents admitted with diabetic comatose state in our emergency unit. At admission to our hospital the patient was somnolent,with limited cooperation and orientation, with Glasgow coma Scale (GSC) 12 (E3V4 M5). He presented marked Kussmaul's breathing, no focal neurological signs. Blood pressure was 100/65 mmHg, cardiac rhythm of 120 beats/min, body temperature was 37,2°C axillary. About 2 weeks before he had an upper respiratory infection; other chronic illness were not known.

Laboratory tests revealed: hyperglycemia (602mg/dl), bicarbonate 10 mEq/l, arterial blood gases: paO2 110mmHg, paCO2 22,5mmHg, pH 7,25, base excess -15,3mEq/l severe metabolic acidosis, urinary ketonic bodies. White blood cell count, red blood count, trombocyte count were normal. Usual blood biochemisty AST, ALT, CK, LDH were normal. Chest x-ray was normal. The diagnosis at the admission was diabetic ketoacidosic comatose state.

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Patient was treated in the intensive care unit, with infusions solutions (isotonic saline, and glucose 5% in addition to intravenous (IV) insulin, and slowly regained consciousness. In the second day, all the vital signs and laboratory became normal, and the patient was transported to the diabetic clinic.

After five days, the patient accused lumbar pain and progressive numbness and proximal lower limb weakness, followed by facial diplegia, impaired deglutition for liquids and solids. Neurological examination revealed bilateral peripheral facial palsy, dysphagia and dysarthria, global areflexia, and symmetrical weakness of lower limbs > upper limbs. Lumbar puncture with cerebrospinal fluid (CSF) examination showed markedly raised protein content, Pandy's test ++++, albumines= 2,1g/l, cell 1/mm3, glicorachia =141mg/dl.

Based on clinical signs (quadriparesis, global areflexia, bilateral peripheral facial palsy, dysphagia and dysarthria), and CSF examination with markedly albuminocytologic dissociation our diagnosis was acute polyradiculonevritis with ascendent evolution. The patient was transported to the neurologic intensive care unit.

The next day, he presents autonomic dysfunctions: sinus tachycardia, hypotension and respiratory failure. He required mechanical ventilatory support. The following 5 days there were performed 5 consecutive plasma exchange therapy with rapid restoration of respiration, facial paresis and deglutition. Patient was detubated after last plasma exchange therapy.

Laboratory tests for Campylobacter jejuni, HIV, hepatitis B and C were negative.

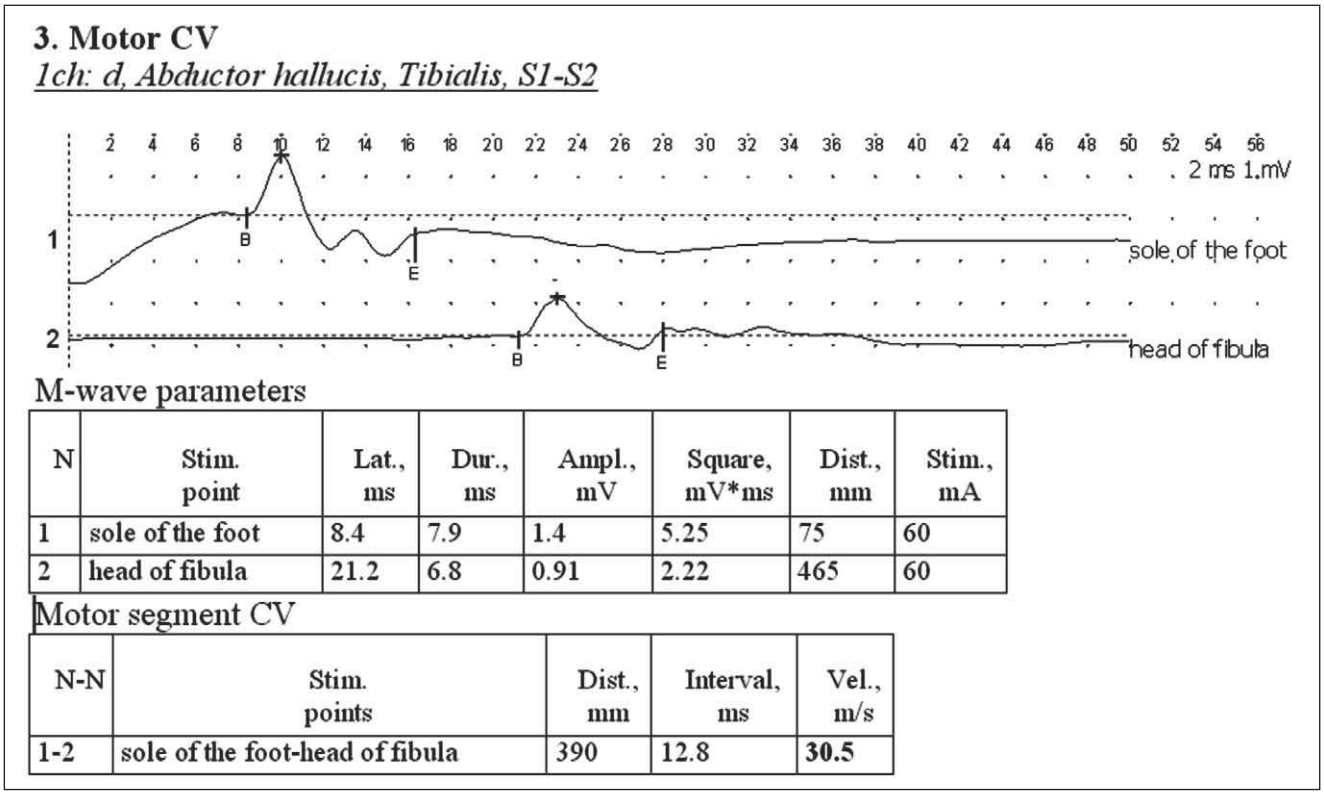
EMG test revealed primary demyelination of peripheral nerves of lower limb, (increased distal latencies, increased dispersion of signal and decreased MCV in proximal and distal parts of nerves and no sign of axonal degeneration) (FIGURE 1 and FIGURE 2).

The final diagnosis was acute inflammatory demyelinating polyneuropathy (AIDP). Our patient treatment comprised insulin, vitamins of group B (Milgamma N). The patient followed a rehabilitation program with physical therapy, and motor weakness recovered after 6 weeks.

He had residual hyporeflexia and diabetes needing an insulin replacing therapy.

DISCUSSION

Gillain-Barre sydrom (GBS) is an acute demyelinating disorder of the peripheral nervous system



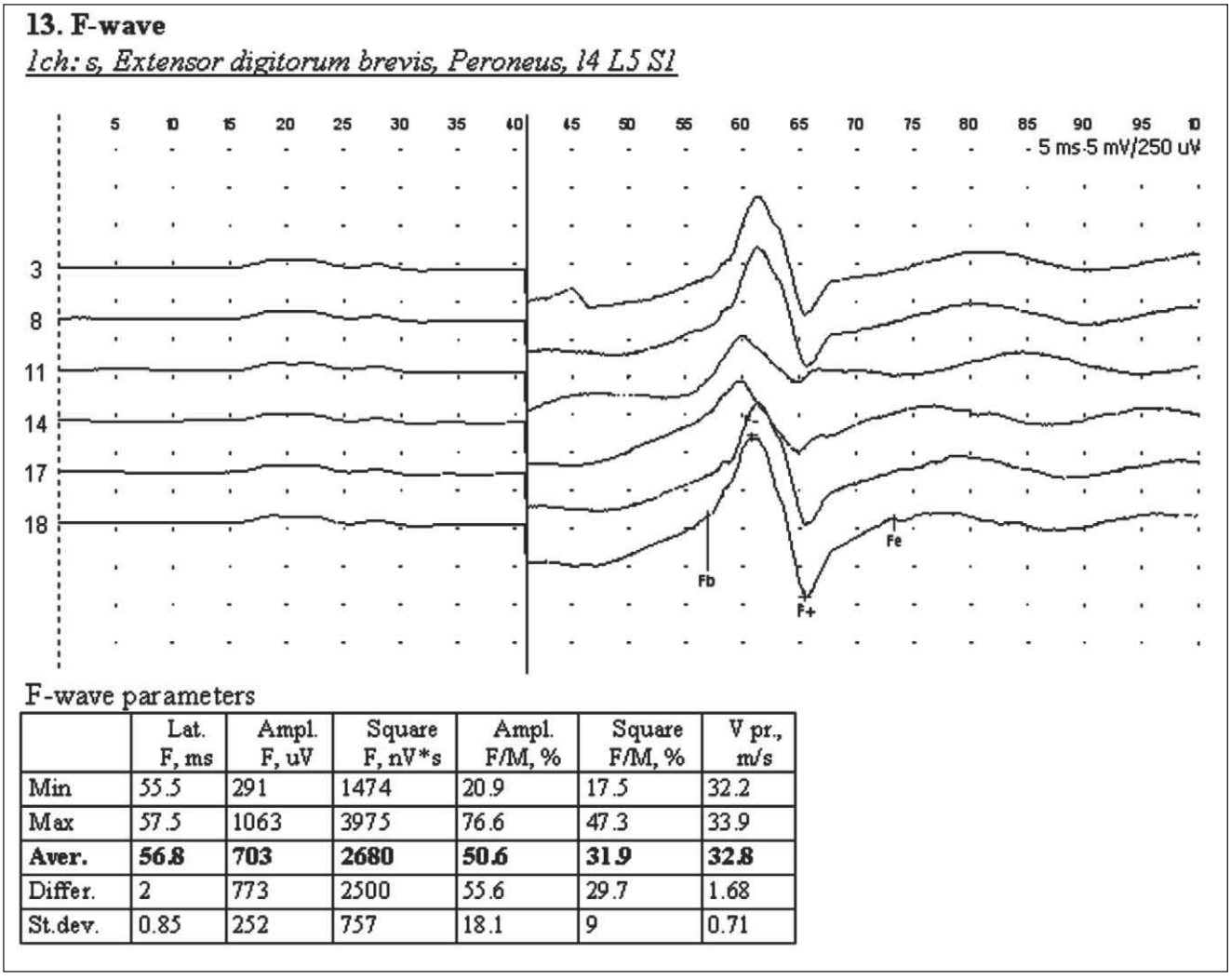
M-wave parameters

N	Stim. point	Lat., ms	Dur., ms	Ampl., mV	Square, mV*ms	Dist., mm	Stim., mA
1	sole of the foot	8.4	7.9	1.4	5.25	75	60
2	head of fibula	21.2	6.8	0.91	2.22	465	60

Motor segment CV

N-N	Stim. points	Dist., mm	Interval, ms	Vel., m/s
1-2	sole of the foot-head of fibula	390	12.8	**30.5**

FIGURE 1. *Motor conduction velocity on right tibialis. We obtained an increased distal letancy, decreased motor conduction velocity in distal segment (between sole of the foot and head of fibula) with a significant dispersion of signal suggesting a primary demielinating process.*



F-wave parameters

	Lat. F, ms	Ampl. F, uV	Square F, nV*s	Ampl. F/M, %	Square F/M, %	V pr., m/s
Min	55.5	291	1474	20.9	17.5	32.2
Max	57.5	1063	3975	76.6	47.3	33.9
Aver.	**56.8**	**703**	**2680**	**50.6**	**31.9**	**32.8**
Differ.	2	773	2500	55.6	29.7	1.68
St.dev.	0.85	252	757	18.1	9	0.71

FIGURE 2. *Proximal motor conduction velocity measured on left peroneus. F-waves recorded on peroneus nerve were inconstant and with prolounged letancies. The velocity calculated in proximal segments was decreased suggesting a poliradiculopaty.*

that results from aberrant immune response directed at peripheral nerves.

Only a few cases of acute polyneuropathy have been reported as complications of diabetic coma. We found on *Medline*,10 articles about the association between diabetes mellitus and Guillain Barre syndrome, both having an autoimmune physiopathology; in these articles there are presented 5 cases that have exactly the same course: onset with diabetic coma, 2-3 weeks after an acute infection, (diarrhea or upper respiratory infection) and then, within a week, the patients present an acute polyneuropathy; the evolution was favorable in all cases; diabetes needed an insulin replacing therapy for long time.

This association seems to be not just a random one; it is rather a single entity. These diseases could be triggered by a common event. "It is likely that in our two patients both auto-immune diseases were triggered by a common event." (Rouanet-Larriviere M. – 2000) (3). Niklasson B. in 1998 found that the incidence of Guillain-Barré syndrome and insulin-dependent diabetes mellitus, as well as the number of deaths caused by myocarditis, followed the fluctuations in numbers of bank voles; an environmental factor, such as an infectious agent, has been suggested for all three diseases. He hypothesized that Guillain-Barré syndrome, myocarditis, and insulin-dependent diabetes mellitus in humans in Sweden are caused by one or more infectious agents carried by small rodents; also, a group of novel picornaviruses recently isolated from these small rodents is being investigated as the possible etiologic agent. (4)

Our case report sustained the hypothesis that an infectious agent (yet not identified) could be a common trigger for both diseases, diabetes mellitus and Guillaine Barre syndrome.

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