Arq Neuropsiquiatr 1997;55(2):315-318

GUILLAIN-BARRÉ SYNDROME AND HEAD TRAUMA

CASE REPORT

GABRIEL R. DE FREITAS*, MARCOS R.G. DE FREITAS**, MYRIAM C.L. FERREIRA***

ABSTRACT - The authors report the case of a 29 years old male patient presenting classical Guillain-Barré syndrome following head trauma. Only one other similar case is described in the literature. Head trauma as a precipitating event of the disease is discussed.

KEY-WORDS: Guillain-Barré syndrome, polyradiculoneuropathy, head trauma.

Síndrome de Guillain-Barré e traumatismo crânio-encefálico: relato de caso

RESUMO - Os autores relatam o caso de um paciente de 29 anos que apresentou síndrome de Guillain-Barré clássica após traumatismo crânio-encefálico. Citam o único caso semelhante descrito na literatura e discutem o trauma como fator desencadeante desta polirradiculoneuropatia.

PALAVRAS-CHAVE: síndrome de Guillain-Barré, polirradiculoneuropatia, traumatismo crânio-encefálico.

The Guillain-Barré syndrome (GBS) is an acute or subacute inflammatory polyneuropathy. The classic description of the syndrome was first made in 1916 by Guillain, Barré and Strohl[10]. The most frequently encountered clinical picture[1] consists of relatively symmetric muscular weakness that begins distally in the lower limbs. In a few days, it begins to evolve more proximally, affecting more proximal muscles such as those of the trunk, neck and face. Paresthesias are common, although objective alterations or deficits in sensibility are usually discrete or absent. Hypotonia and absence of deep reflexes are also seen. The cerebrospinal fluid (CSF) reveals in the majority of cases an elevation of protein and a normal cell count. Eletroneuromyography is of the demyelinating type with reduction of motor conduction velocity, the presence of 'conduction block' in the motor nerves and elevation of the F-wave latency[1]. Variant forms of GBS are less commonly seen, presenting with: severe ataxia and sensory loss; isolated weakness of the arm and oropharynx ; bilateral weakness of facial muscles with distal paresthesias; exclusively autonomic nervous system disturbance; the axonal form of GBS; and the Miller-Fisher syndrome which consists basically of ophthalmoplegia, ataxia and deep areflexia[10]. The cause of GBS is unknown[1]. However in two thirds of cases it follows respiratory or gastrointestinal infection in approximately one to three weeks. Other preceding factors include surgery, viral infection (cytomegalovirus, Epstein-Baar virus, human immunodeficiency virus - HIV), bacterial infections (*Campylobacter jejuni*, *Mycoplasma pneumoniae*) lymphoma and immunization.

Department of Neurology, Hospital Universitário Antônio Pedro, Universidade Federal Fluminense (UFF), Niterói: *Resident; **Chairman; ***Assistant Physician. Aceite: 11-dezembro-1996.

Dr. Gabriel Rodríguez de Freitas - Rua Gastão Ruch 16/1402 - 24220-100 Niterói RJ - Brasil.

The authors report a classic clinical case of GBS which followed head trauma in association with subarachnoid hemorrhage. They discuss head trauma as the triggering factor and mention the only similar case report described in all of the literature.

CASE REPORT

MAC, a 29 years old, white male patient, single, was admitted to the Emergency of the Antônio Pedro University Hospital on April 22nd, 1996 presenting alteration in state of conscience and vomiting. He had been beaten one week before, receiving several blows to the head. The patient was alcoholic and made regular use of marijuana and cocaine through inhalation. There was nuchal rigidity (++/4+) and his Glasgow score was calculated at 13 (3 + 4 + 6). A computed tomography (CT) brain scan revealed subarachnoid hemorrhage. A lumbar puncture showed bloody CSF. The patient later began to show weakness in the four limbs and, on April 25th, was admitted to the Reposal Ward of this same hospital. He was fully alert and oriented. The mucous membranes were dry. His blood pressure was 160x120 mmHg; pulse was 94/min, breath rate was 22/minute and he was apyrexial. The heart, lungs, and abdomen were normal. There was no peripheral edema. His neurological examination revealed a flaccid, distal and proximal tetraparesia along with facial diplegia and deep areflexia. His superficial as well as deep sensibilities were preserved, with bilaterally indifferent cutaneous plantar reflex response. He also presented bilaterally fotoreactive isocoria. All remaining brain stem reflexes revealed no abnormalities. Extrinsic ocular motricity was preserved. There were no radicular signs nor was there nuchal rigidity. The optic fundi were normal. Blood chemistry and hemogram findings are shown in Table 1. A chest X-ray disclosed clear lungs and a normal heart. On the same day, the patient went into respiratory failure so an orotracheal tube was inserted and mechanical ventilation was initiated. The following day, he presented arterial hypotension (60x0 mmHg). Fluid reposition and vasoactive amines were administered intravenously. An eletroneuromyography was performed which revealed a demyelinating pattern (Table 2). Laboratory evaluation of CSF revealed three cells with a protein level of 200 mg/dL. On the 28th April, the patient presented various febrile peaks, a tachycardia of 120/minute and diffuse rales on pulmonary auscultation. An hemogram, blood chemistry, blood cultures were then carried out, along with the inception of an antibiotic regimen consisting of clyndamicine with gentamycine. Athelectasia of the inferior two thirds and lobar consolidation, both of the left lung, were observed on the radiographs of the chest taken on the same day. His white blood cell count was 11,600/mm3, with approximately 74% neutrophils and 27% band forms. The patient was transferred to the Intensive Care Unit. On April 30th, he went into cardiorespiratory arrest from which he was resuscitated. Hours later he suffered another cardiorespiratory arrest this time not responding to manoeuvers used. All three blood cultures yielded *Staphylococcus aureus*, sensitive to the antibiotics prescribed. The ELISA and Western Blot tests for HIV were both negative. Serological tests for cytomegalovirus, Epstein-Barr virus and *Campylobacter jejuni* were not made.

Due to the history of trauma, the body was sent to the Institute of Legal Medicine. There the spinal cord and the peripheral nerves are not studied routinely. The Institute's official report revealed a grayish abrasive 48x25 mm lesion located in the left occipital lobe and a subarachnoid haematoma in the left cerebral hemisphere. Upon transverse slicing, the encephalic tissue showed disseminated linear hemorrhage in a 'dotted' pattern. The cause of death was stated as being cranial contusion associated with both meningeal and encephalic hemorrhage.

Table 1. Results of the first routine laboratory tests em blood.

Biochemistry	Hematology	Platelets(mm³)	White-cell count (mm³)
Sodium (mmol/L) 138	Ht (%) 60.2	203000	11500
Potassium (mmol/L) 5.0	Hb (mg/dL) 18.0		Neutrophils 87%
Urea Nitrogen (mg/dL) 45	MCV (μm³) 93		Band forms 5%
Creatinine (mg/dL) 0.6	MCH (μm³) 31.9		
Glucose (mg/dL) 110	CHCM (μm³) 34.4		

Ht, hematocrit; Hb, hemoglobin; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration.

Table 2. Nerve conduction.

Nerve	Latency (msec)	Amplitude (mv)	NCV (m/sec)	F-Wave (msec)
		MOTOR		
L Median distal	5.9 (n<4)	5.7 (n>3.5)	nr	34.2 (n<31)
proximal	11.5 (n<8.8)	5.1(n>3.5)	45.5 (n>51)	nr
L Ulnar distal	5.6 (n<3.4)	8.5 (n>2.8)	nr	nr
proximal	11.0 (n<7.5)	7.9 (n>2.7)	45.0 (n>54)	nr
L Fibular distal	10.7 (n<5.5)	3.1 (n>2.5)	nr	nr
proximal	19.0 (n<12.9)	1.1 (n>2.5)	38.0 (n>40)	nr
L Tibial distal	12.0 (n<6.0)	1.8 (n>2.9)	nr	nr
proximal	23.0 (n<15.1)	1.2 (n>2.5)	38.8 (n>41)	nr
		SENSORIAL		
L Median	5.7 (n<3.5)	24μv (n>19)		
L Ulnar	0 (n<3.1)	0 (n>18)		

NVC, nerve velocity conduction; L, left; nr, not realized; n, normal.

DISCUSSION

The patient was admitted due to post-traumatic subarachnoid hemorrhage which had been confirmed by CT, lumbar puncture and, finally, autopsy. During his stay at the hospital, he presented with a clinical picture which corresponded to the classic form of GBS. Such a clinical suspicion was confirmed by eletroneuromyography. The CSF evaluation could well have been misleading, since the increased concentration of protein could have easily been explained by subarachnoid hemorrhage. The clinical and eletroneuromyographic picture of the patient fulfilled perfectly the diagnostic criteria for the typical form of GBS according to Asbury and Cornblath[3].

The literature is filled with factors considered to act as 'triggers' in the GBS[1,2,7,10]. Approximately two-thirds of cases occur following a simple, trivial infection, usually viral in nature. The viruses involved are the Epstein-Barr virus, the cytomegalovirus, the other herpesviruses and the HIV. Some cases of GBS have been tied to hepatitis A, hepatitis B, rubella, mumps, influenza A, influenza B, Coxsackievirus and parvovirus[2]. Among bacterial agents involved, *Campylobacter jejuni* and *Mycoplasma pneumoniae* have been the most mentioned. Few reports have appeared with typhoid fever, listeriosis, brucellosis, tularemia, tuberculosis, leptospirosis, malaria and other bacterial infections[2]. *Campylobacter jejuni* is frequently associated with variant or even severe forms of the disease. Vaccination, surgery, epidural anesthesia and drugs, including gold, D-penicilamine, thrombolytic agents and zimelidine have all been associated with some cases[2]. The latter drug, an anti-depressant, has been removed from European drug market after being held responsible for GBS in 13 patients[6]. There are also reports of so-called "symptomatic" GBS, when it is seen as a manifestation of an underlying disease such as systemic lupus erythematosus, Hodgkin's disease and sarcoidosis. A significant number of the above mentioned associations have been well proven clinically in randomized case-controlled studies[2].

Although surgeries, including neurosurgeries[2], are implicated to cause the GBS, there is only one sole case reported of the disease occurring after head trauma[5]. Duncan and Kennedy described

the case of a patient at age 61 who presented with clinical and eletroneuromyographic findings compatible with GBS. He had a parieto-frontal contusion and a small right-sided subdural haematoma that not deemed neurosurgical intervention. The flaccid tetraparesia reached its nadir in four days. Five plasmapheresis sessions were carried out and after a few days, gradual improvement was noted.

Presently, it is believed that the cellular as well as the humoral immunities both have a role in the physiopathogenesis of the GBS[7]. Cellular and humoral immunities are altered by head trauma. Two recent reports[8,9] show that severe head injury is associated with depressed cell-mediated immunity. These patients are at increased risk for infection and have anergy to common antigens. One of these reports[8] reveal on the other hand an increase in B-cells and immunoglobulin levels immediately after the injury.

More sensible tests demonstrate the presence of anti-myelin antibodies in the majority of patients with GBS. There are reports that confirm the existence of elevated serum and CSF levels of basic myelin protein in patients who were submitted to neurosurgeries or suffered head trauma[5]. It is possible that in our case and in the one described by Duncan and Kennedy, head trauma may have induced the synthesis of anti-myelin antibodies.

Another possible mechanism is the stress caused by infections, surgery or trauma activating some therefore latent or subclinical process. There are reports that describe the reativation of the GBS in patients who had a previous history of the disease once they were submitted to surgical procedures[4].

In regard to the reported case, it is hard to affirm whether there actually exists a cause-and-effect relationship between these two entities or if it is simply mere coincidence. The authors believe that further studies regarding the complex physiopatogenesis of the GBS coupled with extensive epidemiological research will clarify such a singular and peculiar association.

Acknowledgment - The authors thank Dr. Peter Salem Junior for his helpful assistance in preparing this manuscript.

REFERENCES

1. Adams RD, Victor M. In: Principles of neurology; Ed 5. New York: McGraw-Hill, 1993:1117-1169 (Diseases of peripheral nerves).
2. Arnason B, Soliven B. Acute inflammatory demyelinating polyradiculoneuropathy. In Dyck PJ, Thomas PK, Griffin JW, Low PA, Poduslo JF. Peripheral neuropathy; Ed 3. Philadelphia: WB Saunders, 1993:1437-1497.
3. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990;27:S21-S24.
4. Borit A, Altrocchi PH. Recurrent polyneuropathy and neurolymphomatosis. Arch Neurol 1971;24:40-49.
5. Duncan R, Kennedy PGE. Guillain-Barré syndrome following acute head trauma. Postgrad Med J 1987;63:479-480.
6. Fagius J, Osterman PO, Siden A, Wiholm BE. Guillain-Barré syndrome following zimelidine treatment. J Neurol Neurosurg Psychiatry 1985;8:65.
7. Hughes RAC. Guillain-Barré syndrome and chronic inflammatory demyelinating polyradiculoneuropathy. In Asbury AK, Thomas PK. Peripheral nerve disorders 2. London: Butterworths, 1985:175-204.
8. Meert KL, Long M, Kaplan J, Sarnaik AP. Alterations in immune function following head injury in children. Crit Care Med 1995;23:822-828.
9. Quattrocchi KB, Miller CH, Wagner FC Jr, DeNardo SJ, DeNardo GL, Ovodov K, Frank EH. Cell-mediated immunity in severely head-injured patients: the role of supressor lymphocythes and serum factors. J Neurosurg 1992; 77:694-699.
10. Ropper AH. The Guillain-Barré syndrome. N Engl J Med 1992;326:1130.