Commentary

The first description of the disorder was provided by French doctor Jean Landry in 1859.[1] In 1916, Guillain *et al*.[2] diagnosed the condition in two soldiers and described an abnormal increase of protein production in cerebrospinal fluid and reduced osteotendinous reflexes. In 1956, neurologist, Miler Fisher described the variant that bore his name.[3] The incidence of Guillain–Barré syndrome (GBS) varies between 0.89 and 1.89 cases in 100,000 subjects/year. The male/female ratio is approximately 1.78. In two-thirds of cases, it is preceded by an infection in the upper respiratory system as described by Nanda or by gastrointestinal phenomena, more frequently diarrheal. The most commonly associated infectious agent is campylobacter jejuni followed by cytomegalovirus. Further infectious agents can be the virus Epstein–Barr, varicella-zoster, and Mycoplasma pneumoniae. At times, vaccinating some weeks prior to developing GBS is thought to be a triggering factor, as in cases recorded following mass immunization against virus A/NewJersey/1976/H1N1 "swine flu".[4] In their article "Twelfth cranial nerve involvement in Guillain–Barré Syndrome",[5] the authors underline number of fundamental characteristics of such pathology, emphasizing its seriousness and complications. The classic symptoms of the onset of GBS are numbness, tingling, weakness, and pain that generally begin in the lower limbs. Such symptoms can appear separately or together and gradually worsen. The main characteristic is a relatively symmetrical weakness to the lower limbs, which worsen progressively over a period of between 12 hours and 28 days, spreading in a craniocaudal direction, possibly also concerning areas, as this study illustrates, which are innervated by cranial nerves, before reaching a plateau. The patient present hypo-areflexia although around 10% of cases may present conserved or even increased reflexes. Its course is usually monophasic, although relapses may occur in about 7% of patients.[6] Upon reaching the peak of hyposthenia, approximately two-thirds of patients are unable to walk. In about 25% of patients, respiratory failure occurs and mechanical ventilator may be necessary, as in the case described. Internistic complications such as aspiration pneumonia, sepsis, pulmonary embolism, and gastrointestinal bleeding develop in around 60% of cases. The Miller Fisher variant, characterized by ataxia-areflexia-ophthalmoplegia, is most common in eastern Asia. Moreover, in these cases, infections often precedes the onset of the pathology. Some pathological conditions can mimic the Miller Fisher syndrome, so careful clinical evaluations must be carried out. The pathogenesis is of the disimmune type and it has by now well documented that molecular mimicry plays an important role. Among all others we recall the following autoantibodies: GQ1b and GT1a, associated to the Miller Fisher strain; GM1 and GD1a, associated to the axonal variant. The patient described presented positivity, among others, for GT1b: Said antibody is associated to the most serious forms, which often, as in the case illustrated, require mechanical ventilation.

Differential diagnosis concerns a wide variety of neurological pathologies. A detailed neurological examination with careful anamnesis localizes the problem on a peripheral nervous system level rather than on other levels, such as the brainstem, as can be hypothesized by the involvement of the XII NC described in the case report under examination. The authors correctly carried out an encephalon Nuclear Magnetic Resonance (NMR) to rule out other causes of the hypoglossal nerve.

Studies of nerve conduction are useful in confirming the presence of the polyneuropathy. It is interesting to note that the Brighton criteria,[7] designed to be used in situations of poor resources, does not consider such studies to be obligatory for diagnosis. On the basis of the studies of nerve conduction, GBS divides into two variants: demyelinizing and axonal. The two variants have important differences in geographical distribution; indeed the demyelinizing variant

represent about 90% of cases in Europe and North America, while the axonal variant is more frequent in China, Japan, Bangladesh, and Mexico. The case in question, study of nerve conduction showed severe demyelinization.

Rachicentesis is carried out in all patients with suspected GBS. A common error is to think that the cerebrospinal fluid examination must always show albumin-cytological dissociation. During the first week of illness, such fluid alteration is only present in approximately 50% of patients and rises to 75% in the third week.[8]

GBS therapy can be approached on a number of levels: immunotherapy and support therapy.

In the first instance we recall the efficacy of plasmapheresis especially when this is started within 2 weeks of the symptoms appearing, and endovenous administration of immunoglobulin. Support therapy is necessary to face all possible complications of an internal nature to which such patients are exposed. The case report described here clearly highlights how support therapy, in this case mechanical ventilation, is a fundamentally important element of managing the patient affected by GBS and how it can drastically change the patient's prognosis.

The case illustrated by Nanda *et al*., demonstrates many of the clinical and epidemiological characteristics of GBS in addition to the less frequent involvement of the XII CN, showing how such disease can be heterogeneous and how its diagnosis and more importantly its management can require multidisciplinary approach and represent a significant challenge for the clinician.

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