EDITORIAL

Guillain-Barré Syndrome

Some of the neurological diseases are common causes of disability and death world wide. Among them, stroke and degenerative diseases are of importance. But the acute conditions like infection and vascular phenomenon usually carries good prognosis whereas chronic illnesses like motor neuron disease and Parkinson's disease give rise to more disability. Guillain-Barré syndrome (GBS) is one of the commonest causes of acute symmetrical flaccid paralysis of limbs in much of the world.[1,2] The basic pathogenesis of this disease is the involvement of nerve roots by antigen and antibody mediated reaction. The disease is often preceded by an antecedent, either upper respiratory tract infection (URTI) or gastroenteritis by virus or bacteria. The antigens of these organisms have molecular mimicry with that of gangliosides of nerve roots. The antibody produced against the organism's antigen interacts with the gangliosides of nerve root and results either demyelination or axonopathy or both[3,4,5].

The diagnosis of GBS is usually straightforward on the basis of its triad of presentation like-acute symmetrical ascending paralysis of limbs, areflexia and albumino-cytological dissociation in CSF[6,7,8,9]. The electro-physiological diagnosis is useful in the early stage and in typing of GBS.

Under the description of GBS there are varieties of presentations like acute inflammatory demyeleniting polyradiculopathy (AIDP), acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN) and a mixed variety AIDP+AMSAN. The differentiation into those types is done on the basis of their presentations and prognosis. AIDP carries good prognosis and AMAN carries poor prognosis. The others are in between the two[10,11]. The commonest type of GBS in the western world is AIDP following URTI. But from the northern China and India the commonest variety reported is AMAN following gastroenteritis by *Campylobacter jejuni*[12,13]. In Bangladesh, it is anticipated that the predominant type of GBS may be AMAN, but there had been two case studies which showed conflicting results, AIDP in one and AMAN in the other. In Bangladesh, GBS is the commonest cause of polyradiculopathy in the hospital. No age or sex is immune, but the commonest age of involvement is young adults and male sex. It is observed that there is a little higher admission of GBS in summer season.[14]

There is no specific treatment of GBS. But the use of intravenous immunoglobulin[19] in the first week in selected cases may slow the progress of the disease and thus enhance recovery, shorten the hospital stay and minimize the need of a ventilator. The use of Inj. Methyl Prednisolone alone is not recommended and the combination of IVIg and Inj. Methyl Prednisolone also has no benefit. Plasmapheresis is another option but its benefit is like IVIg in addition to many limitations of its use. Physiotherapy is still the mainstay of treatment in all cases[15,16]. The prognosis of GBS is good, 80% recover, 10% remain disabled and 10% die[17].

Professor Quazi Deen Mohammad
Head, Department of Neurology
Dhaka Medical College, Dhaka.

(J Bangladesh Coll Phys Surg 2006; 24: 42-43)

References:

1. Leneman F. The Gullian Barre Syndrome: Definition, etiology and review of 1100 cases. Arch Intern Med 1996; 128: 139-49.
2. Griffin JW, Li CY, Ho TW. Gullian Barre Syndrome in northan China. The spectrum of neuropathological changes in clinically defined cases. Brain 1995; 118: 577-95.
3. Dowling PC, Cook SD. Role of infection in Gullian Barre Syndrome. Neurology (NY) 1978: 28; 686-90.
4. Hadden RDM, Karca H, Hartung HP. Preceding infaections, immune factors and outcome in Gullian Barre Syndrome. Neurology 2001; 56: 758-65.
5. Heigines RAC, Hudden RDM, Greson ND, Smith KJ. Pathogenesis of Gullian Barre Syndrome. J Neuro Immuno 1999; 100; 74-97.

6. Asbury AK, Amason BG, Karp HR, Mc Forlin DE. Criteria for diagnosis of Gullian Barre Syndrome. Ann Neurol 1978; 3: 565-6.

7. Asabury AK, Cornblath DR. Assessment of correct diagnostic criteria for Gullian Barre Syndrome. Ann Neurol 1990; 27 (suppl.): 21-4.

8. Marshal J. The Landry Gullian Barre Syndrome. Brain 1963; 86: 55-56.

9. Masucci EF, Kurtzkz JF. Diagnostic criteria for the Gullian Barre Syndrome: An analysis of 50 cases. J Neurol Sci 1971; 13: 483-501.

10. Albers JW, Donofrio PD, Mc Gonagle TK. Sequential electro-diagnostic abnormalities in acute inflammatory demyelinating polyradiculopathy. Muscle Nerve 1985; 8: 528-9.

11. McLyod JG Electrophysiological studies in the Gullian Barre Syndrome. Annals Neurol 1981; 9 (suppl): 20-7.

12. Ropper AH. The Gullian Barre Syndrome. New Eng J Med 1992; 326: 1130-6.

13. Rees JH, Soudain SE, Gregon NA. Campylobacter jejuni infection and Gullian Barre Syndrome. New Eng J Med 1995; 333: 1374-9.

14. Haque MA, Haque S, Rahman SA, Hossain S, Gogh PC, Begum T et al: Presentation of GBS-A prospective study in BIRDEM Hospital. Bang J Neurosci 1998; 14: 35-40.

15. Yelena LD, John TK, Jerry RM. Treatment approach for Gullian Barre Syndrome and chronic inflammatory demyelinating polyradiculopathy. Neurologic Treatment 2001; 19: 187-204.

16. Deborah M G Advances in management of Gullian Barre Syndrome. Curr Neurol Neurosci Rep 2002; 2: 541-48.

17. Carpo M, Redotti R, Allaria S, Lolli F, Muta S, Cavaletti G. Clinical presentation and outcome of Gullian Barre and related syndrome in relation to anti-ganglioside antibodies. J Neurol Sci 1968; 7: 581-92.