EDITORIAL

Guillain- Barre` Syndrome: Critical Care of Paediatric Patients

Peripheral neuropathies of various aetiologies occur in children. Their courses could be acute, subacute or chronic. Many a times it is difficult to reach to a causative diagnosis. Infections like diphtheria, HIV, Cytomegalovirus, Epstein-Barr virus, Campylobacter jejuni have been found to be linked with childhood polyneuropathy[3]. Some also been shown to be inflammatory or associated with certain drugs and malignant conditions as well as been hereditary too[1].

Guillain- Barre Syndrome (GBS) is also referred to as acute inflammatory or post-infection neuropathy, acute inflammatory demyelinating ploy-radiculo-neuropathy and Laudry-GBS[1]. It is the most common cause of acute polyneuropathy. In GBS, an infection commonly of the upper respiratory tract or gastro-intestinal tract preceed the onset of the paralysis by 1-3 weeks. The related organisms induce antibody response against peripheral nerves.

Typical GBS is a monophasic illness with symmetrical ascending weakness affecting both proximal and distal muscles. Hallmark features include ascending paralysis and areflexia, paresthesia and muscle pain. The sensory involvement is usually minimal, deep tendon reflexes are lost early in the course of the illness; cranial nerve involvement especially of the Facial nerve os fairly common[2]. Autonomic involvement can cause wide fluctuations of the blood pressure, cardiac arrhythmias and bladder dysfunctions.

In GBS, paralysis may progress rapidly from hours to days, most patients reaching the maximal deficit by two weeks; respiratory failure occurs in up to 30% affected[2]. In young children severe pain mimicking encephalo-myelitis may signify onset of GBS[3].

Diagnosis of GBS is based mainly on clinical grounds. CSF study shows increased protein (1-3 gm/dl) with only a few cells (albumino-cytologic dissociation) and normal sugar content. Spinal MRI, nerve conduction study and nerve biopsy may help in diagnosis. Nerve conduction study demonstrates either axonal or demyelinating neuropathy, axonal ones having the worse prognosis for recovery[3]. Acute paralytic illness like botulism, spinal cord compression, muscular disease and Myasthenia should be excluded.

All children with suspected GBS should be hospitalized, essential supportive therapy given and monitored for respiratory status, blood pressure and cardiac arrythmias. Special expertise in medical and nursing care should be involved in management of such cases[2]. With worsening conditions patients should be referred to special centers having additional facilities for intubation and artificial ventilation such as intensive care units (ICU / PICU). Patients should be put to ventilator early if deterioration noted.

Intravenous gamma globulin (IVIG) administered early within the first two weeks reduces duration and severity of paralysis[1]. Prior to administration of IVIG, serum IgA level of the patient should be measured as in an occasional patient with congenital IgA deficiency has been noted to manifest severe allergic reactions. Plasma exchange has proven to shorten disability in GBS and steroids found to be no value. Prolonged ventilations as required in some patients. To get recovered in GBS takes weeks to months, more severely affected ones are left with residual weakness, fatigue is common, and some 15% die or are left disabled.

To summarize, patients whose conditions suggest GBS should be very closely monitored, meticulous supportive treatment given; as soon as condition deteriorates should be transferred to an intensive care facility (ICU/PICU). The study reported in this issue on on 214 paediatric patients with GBS emphasized the importance and prognosis in a well supported tertiary care hospital.

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