Pediatric Neurology: Chapter 120. Acute polyradiculoneuritis: Guillain-Barré syndrome (Handbook of Clinical Neurology)

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Guillain–Barré syndrome (GBS) is an acute, immune-mediated, postinfectious polyneuropathy with symmetrical ascending weakness, diminished deep tendon reflexes, and nonspecific sensory symptoms. CSF protein is raised with normal or only slightly elevated cell count. Based on electrophysiological and pathological findings, a demyelinating variant (acute inflammatory demyelinating polyneuropathy, AIDP) and an axonal variant (acute motor axonal neuropathy, AMAN) can be differentiated. Molecular mimicry with common epitopes between infective agents and peripheral nerves is discussed as an important pathophysiological principle. The symptoms progress for a mean of 10 days (up to 4 weeks) and after a plateau of 1–2 weeks remit spontaneously. At the height of the disease 60% of children are unable to walk and 10–15% need artificial ventilation. Treatment with plasmapheresis and intravenous immunoglobulins (IVIG) has been proven in placebo-controlled studies in adults with severe disease to speed up recovery significantly. In children, mostly open studies have shown similar treatment effects, although their spontaneous course is frequently less severe. Children with GBS should be treated with IVIG when they have lost the ability to walk, or when they are still deteriorating significantly and are expected to lose the ability to walk. The long-term prognosis is more favorable than that in adults. Whereas 25% of patients maintain mild neurological symptoms and signs, disability in the long term is very rare and usually due to complications such as myelitic involvement or chronic inflammatory demyelinating polyneuropathy (CIDP).

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