Infections and the Guillain-Barré syndrome

It has long been textbook knowledge that in most patients an infective illness precedes the development of the Guillain-Barré syndrome (GBS). About two thirds of patients report symptoms of an upper respiratory tract or gastrointestinal tract infection preceeding the disease by 1–4 weeks. Many diverse infectious agents have been incriminated as triggers of this acute polyradiculoneuropathy but only few larger scale and case controlled studies, so important in a disease with a low incidence of 1–2/100 000, have been published. Based on this evidence, infections with the gram negative enteropathogen Campylobacter jejuni, cytomegalovirus (CMV), Epstein-Barr virus, and Mycoplasma pneumoniae are precipitants of GBS whereas other infections occur no more often in this neuropathy than in controls. The agent most commonly associated with GBS is Campylobacter jejuni. In most parts of the western world, the frequency is around 33%, whereas in China and Japan, this figure is around 45–60%. CMV has been identified as the predominant viral cause of an infective illness preceding GBS, present in 10–15% of patients. Association with Epstein-Barr virus infection and infection with Mycoplasma pneumoniae seem to be less common, occurring in around 8%–10% and 5%, respectively. These findings are certainly of epidemiological interest but they may also provide us with important insights into the pathogenesis of this immune mediated neuropathy and may explain some aspects of the emerging heterogeneity of this disorder. A study of the clinical and electrophysiological features of patients with CMV associated GBS that were enrolled in the Dutch GBS trial comparing IVIG with plasma exchange, first indicated that in this subgroup of patients with GBS the disease seems to follow a different clinical pattern with more frequent involvement of cranial nerves and severe sensory loss. Another feature that would separate CMV related GBS from GBS not preceded by this viral infection is the presence in a particular glycolipid in CMV related GBS although, unlike in C jejuni associated GBS, their causal relation seems obscure. Whether the occurrence of GM2 antibodies also in acute CMV infection without neuropathy recently reported by Yuki and Tagawa in their large scale study clouds this issue or implies a requirement for the presence of specific individual host susceptibility factors also remains to be determined.

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