Intrathecal anti-CD20 efficiently depletes meningeal B cells in CNS autoimmunity.

Clinical trials revealed that systemic administration of B-cell-depleting anti-CD20 antibodies can hold lesion formation in the early relapsing-remitting phase of multiple sclerosis (MS). Throughout the secondary-progressive (SP) course of MS, pathogenic B cells may, however, progressively replicate within the central nervous system (CNS) itself, which is largely inaccessible to systemic anti-CD20 treatment. Utilizing the murine MS model of experimental autoimmune encephalomyelitis, we show that intrathecal (i.t.) administration of anti-CD20 alone very efficiently depletes meningeal B cells from established CNS lesions. In SP-MS patients, adding i.t. administration of anti-CD20 to its systemic use may be a valuable strategy to target pathogenic B-cell function.