an initial brainstem attack sharing chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) features. After steroid weaning, MOG-seropositive longitudinally extensive transverse myelitis (LETM) involving the conus appeared, but in absence of brainstem lesions.[1] A diagnosis of CLIPPERS is difficult in this clinical picture.

Brainstem punctate and curvilinear enhancements, a characteristic radiologic finding of CLIPPERS, may conceal several diseases such as glioma, primary CNS lymphoma, lymphomatoid granulomatosis, primary CNS vasculitis, and multiple sclerosis. Except for glioma, all of these diseases initially respond to high doses of steroids, and some could have a relapsing-remitting course in absence of immunosuppressive therapy. However, unlike CLIPPERS, the brainstem is not systematically affected at each relapse and lesion distribution does not remain concentrated in the pons.[2–4]

The Symmonds et al. case highlights a possible pathophysiologic connection between CLIPPERS and demyelinating diseases. Recently, a postmortem analysis performed in a patient sharing all CLIPPERS features revealed the classical perivascular lymphohistiocytic infiltrates but also perivenular demyelinating lesions (as seen in acute disseminated encephalomyelitis).[5] Since perivenular demyelinating lesions were found in only one CLIPPERS patient, it is unlikely that CLIPPERS is a primary demyelinating disease. However, as suggested by the authors, CLIPPERS may induce immunization against MOG antigen.

Author Response: Mkael Symmonds, M. Isabel Leite, Ursula G. Schulz, Oxford, UK: We agree with the important points that Drs. Taieb and Labauge raised in response to our recent report.[1] Our patient presented with clinical and radiologic features consistent with CLIPPERS, even though the subsequent episode of LETM involving the conus had not been previously reported as part of this disease spectrum. While CLIPPERS can relapse, typically with recurrence of brainstem inflammatory features, this does not form key diagnostic criteria and the diagnosis could be consistent with a single episode.

As discussed, the differential diagnosis of CLIPPERS is wide and many of these alternatives will clearly define themselves in time. The histopathologic findings of demyelination in an isolated case at postmortem provides interesting additional support for the hypothesis that CLIPPERS may not be a distinct entity, but rather have a range of underlying etiologies. Our case raises the possibility that anti-MOG antibodies may be causal in some CLIPPERS cases, although we cannot exclude the possibility that CLIPPERS itself causes immunization against MOG epitopes.

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3. Limousin N, Praline J, Motica O, et al. Brain biopsy is required in steroid-resistant patients with chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS). J Neurooncol 2012; 107:223–224.
4. Ferreira RM, Machado G, Souza AS, et al. CLIPPERS-like MRI findings in a patient with multiple sclerosis. J Neurol Sci 2013;327:61–62.
5. Moreira I, Cruto C, Correia C, et al. Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS): postmortem findings. J Neuropathol Exp Neurol 2015;74:186–190.

CORRECTION

Pseudo-Foster-Kennedy syndrome with optic nerve compression by the gyrus rectus

In the Neuro*Image* "Pseudo-Foster-Kennedy syndrome with optic nerve compression by the gyrus rectus" by N. Desai et al. (*Neurology*® 2015;85:385), there is an error in the correspondence address. The note at the bottom should read "Correspondence to Dr. Rucker: janet.rucker@nyumc.org." The authors regret the error.

Author disclosures are available upon request (journal@neurology.org).

Pseudo-Foster-Kennedy syndrome with optic nerve compression by the gyrus rectus

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