Neurocritical Care

ISSN 1541-6933/06/5:251 ISSN 1556-0961 (Online)
DOI: 10.1385/Neurocrit. Care 2006;05:251

Letter to the Editor

Response By the Authors:

We thank Dr Czartoski and Dr. Becker for their interest in our report *(1)*. Although cerebrovascular complications are frequent in pneumococcal meningitis, with an incidence of more than 20% for arterial complications *(2)*, no study has systematically characterized these complications. In this context the suggestion of an early vasculitic process being distinct from a more chronic vascular inflammation seems hypothetical. In our report, vasculitis was certainly an early complication of pneumococcal meningitis. The patient developed focal neurological symptoms 3 days after the onset of meningitis, with two hypodensities in the right posterior arm of the internal capsula. The prolonged course of vascular inflammation and the relapse after steroid reduction that we observed may be caused by the delayed initiation of the immunosuppressive treatment.

We agree that the occurrence of persistent postinfectious vasculitis might be related to a host susceptibility, but the nature of the infectious pathogen is certainly also critical. It is noteworthy that vasculitic complications are observed more frequently in pneumococcal meningitis *(3)* and that the benefit of steroids seems to be greatest in patients with pneumoccocal disease *(4)*.

Neurological complications in pneumococcal meningitis are extremely various and difficult to predict. A critical issue is to identify patients susceptible to development of severe vascular complications and who would benefit from aggressive and prolonged immunosuppression. Leppert et al. *(5)* previously reported that cerebrospinal fluid levels of matrix metalloproteinase-9 (MMP-9) were higher in patients with long-term neurological deficits. MMPs play a key role in the extravasation of inflammatory cells in the central nervous system and may be directly implicated in the pathophysiology of meningitic vasculitis. In our case report, the patient clinical evolution and the vascular process correlated with MMP-9 levels. Thus, MMP-9 could be a marker of vascular inflammation and help to identify and monitor patients who could benefit from aggressive and prolonged immunosuppression. Further studies are needed to confirm this hypothesis.

References

1. Pugin D, Copin JC, Goodyear MC, Landis T, Gasche Y. Persisting vasculitis after pneumococcal meningitis. Neurocrit Care 2006; 4: 237–240.
2. Kastenbauer S, Pfister HW. Pneumococcal meningitis in adults: spectrum of complications and prognostic factors in a series of 87 cases. Brain 2003;126:1015–1025.
3. van de Beek D, de Gans J, Spanjaard L, Weisfelt M, Reitsma JB, Vermeulen M. Clinical features and prognostic factors in adults with bacterial meningitis. N Engl J Med 2004;351:1849–1859.
4. de Gans J, van de Beek D. Dexamethasone in adults with bacterial meningitis. N Engl J Med 2002;347:1549–1556.
5. Leppert D, Leib SL, Grygar C, Miller KM, Schaad UB, Hollander GA. Matrix metalloproteinase (MMP)-8 and MMP-9 in cerebrospinal fluid during bacterial meningitis: association with blood-brain barrier damage and neurological sequelae. Clin Infect Dis 2000;31:80–84.

***Correspondence and reprint requests to:**

Yvan Gasche
Division of Critical Care Medicine
Geneva University Hospital
24, Rue Micheli-du-Crest,
CH-1211,
Geneva, Switzerland.

E-mail:
yvan.gasche@medecine.unige.ch

Humana Press