


Reply

SIR—We appreciate the interest of Cheng and Khairullah in our case report about antiphospholipid syndrome related to acute cytomegalovirus (CMV) infection in an immunocompetent host [1]. Prieto et al. showed strong evidence that hepatitis C virus (HCV) infection is associated with the presence of antiphospholipid autoantibodies and a high incidence of thrombotic disorders in this group of patients [2]. Therefore, there is growing evidence that antiphospholipid autoantibodies related to viral infections could be pathogenic and are not only an epiphenomenon. However, there is some evidence that enveloped virus could have some procoagulant activities. For example, it has been shown that the CMV surface contains the necessary procoagulant phospholipid for coagulation enzyme complex assembly [3].

In addition, patients with cirrhosis secondary to HCV infection and thrombosis could have other procoagulant alterations that explain the thrombotic problems [4]. In this sense, the hypothesis of Cheng and Khairullah about probable pathways for anticardiolipin induction during viral infections should be considered and explored. Further studies to determine the pathogenic role of anticardiolipin antibodies in relation to various infections are warranted.

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References


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Isolated Detection of Cryptococcal Polysaccharide Antigen in Patients with Cryptococcosis

SIR—Manfredi et al. recently highlighted the importance of CSF cryptococcal antigen assay for the early diagnosis of cryptococcosis in patients with AIDS [1]. However, the isolated detection of cryptococcal polysaccharide in CSF is not a new finding. In our series from northern India [2], cryptococcal meningitis was diagnosed in 11 patients by use of isolated cryptococcal antigen assay (Crypto-LA test; International Biological Labs, Cranbury, NJ). Simultaneous efforts to isolate Cryptococcus neoformans from the same CSF samples were unsuccessful. The antigen titer was >8 in all 11 cases. Ten patients presented with symptoms and signs of chronic meningitis. The remaining patient had no signs of meningeal involvement and presented with pyrexia of unknown origin. Six of 11 patients were immunocompromised because of underlying illness, but none of the patients had AIDS.

Because of the known specificity of cryptococcal antigen assay, antifungal therapy was instituted for all of the patients, eight of whom responded well. The other three patients died of their illnesses. One patient was autopsied, and yeast cells with capsules were identified in meningeal tissue. The cross-reaction of Trichosporon antigen and Cryptococcus antigen is known [3]. However, the rarity of disseminated Trichosporon infection in northern India led us to believe that all of these patients had cryptococcosis; this could even be confirmed by autopsy in one case. Therefore, cryptococcal antigen assay should be included as part of the routine diagnostic protocol for all patients with chronic meningitis.

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References


Reply

Sir—Chakrabarti and Gupta present important observations regarding isolated detection of the polysaccharide antigen of Cryptococcus neoformans from the CSF of 11 non-HIV-infected patients with CNS cryptococcosis in northern India, although in three of 11 cases, direct microscopic study of the CSF revealed the organism [1]. Unfortunately, at the time that we submitted our report [2], we were not aware of Chakrabarti and Gupta’s report [1]; the journal in which their study was published is not available in our country and is not cited in the major international indexes.

The testing of serum and CSF for cryptococcal antigen is used increasingly as a highly sensitive and specific screening technique for high-risk immunocompromised patients, most specifically those with advanced AIDS [3, 4]. Although the presence of cryptococcal antigenemia without detection of fungi from any site represents an emerging issue because of repeated observations [3] (Powderly [4] already stated in 1993 that isolated cryptococcal antigenemia should be regarded as a “new clinical entity”), an early diagnosis of CNS cryptococcosis made solely on the grounds of CNS positivity for antigen remains rare [1, 2], although it should be carefully considered in the management of this opportunistic infection, whose early recognition may be hampered by mild and atypical clinical features and slow progression.

The data reported by Chakrabarti and Gupta are largely consistent with ours and above all underline that isolated antigen detection, and to devise guidelines for appropriate treatment of this condition.

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References


Thrombotic Thrombocytopenic Purpura and Cytomegalovirus Infection in an Immunocompetent Adult

Sir—We read with interest the recent article on severe cytomegalovirus (CMV) infection in immunocompetent patients by Edleston et al. [1]. On rare occasions, CMV infection may also give rise to thrombotic microangiopathy [2]. Recently, we treated a patient with thrombotic thrombocytopenic purpura (TTP) that arose during the course of a primary CMV infection; to our knowledge, this is the first case of TTP reported in a CMV-infected patient.

A 30-year-old woman was referred to our hospital because of purpura, fever (temperature, 39°C), and jaundice. Two days before presentation, she had noticed the appearance of purpuric lesions on her feet that spread to her thighs. There were no other clinical signs on admission.

Blood test results were consistent with mechanical hemolytic anemia and included the following: hemoglobin, 75 g/L; haptoglobin, markedly decreased; schistocytes, 8%; direct antiglobulin test, negative; total bilirubin, 55 mg/L (unconjugated, 34 mg/L); platelets, $10 \times 10^9$/L; WBCs, 11.6 $\times 10^9$/L (neutrophils, 55%; lymphocytes, 30%; and no atypical forms). There were no hemocoagulation abnormalities. Renal function was not altered. Results of blood chemistry evaluation revealed evidence of hepatic cytolysis: aspartate aminotransferase (AST), 136 IU/L; alanine aminotransferase (ALT), 154 IU/L (normal values <30 IU/L). The alkaline phosphatase level was 176 IU/L (normal level, 20–80 IU/L), and the γ-glutamyl transpeptidase level was 327 IU/L (normal level, <37 IU/L). Blood and urine cultures were negative.