

To the editor:

Central nervous system (CNS) disease in Abelson virus–induced leukemia

In the original description and isolation of the murine Abelson leukemia virus, we described a “massive degree of meningeal involvement” as a characteristic of the disease process.^{1,2} As in the Bcr/Abl murine bone marrow transduction and transplantation model, the latent period to tumor appearance was very short. We suggested that the meningeal disease could serve as a model for testing chemotherapy.

Herbert T. Abelson

Correspondence: Herbert T. Abelson, Department of Pediatrics, University of Chicago, 5841 South Maryland Ave, MC 1051 (C-120), Chicago, IL 60637; e-mail: habelson@peds.bsdu.uchicago.edu

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To the editor:

ADAMTS13 and thrombotic thrombocytopenic purpura–hemolytic uremic syndrome

We read with interest the paper by Vesely et al concerning ADAMTS13 (a disintegrin-like and metalloprotease with thrombospondin type 1 repeats) activity in relation to presenting features and clinical outcomes of patients suffering from thrombotic thrombocytopenic purpura–hemolytic uremic syndrome (TTP-HUS).¹ We would like to make a few comments.

First, in regards to the etiology, we are surprised that in a large prospective cohort of 142 patients, only 2 patients were HIV positive. In a recent retrospective review of 55 adults admitted to our renal intensive care unit (ICU) between 1990 and 1998, 18 suffered HIV infection.² It was the main factor influencing the in-hospital mortality and the requirement for hemodialysis. HIV-1 infection has been reported to account for up to 30% of TTP-HUS.³ In a similar fashion, a 1.4% incidence of TTP-HUS was reported in patients with acquired immunodeficiency syndrome.⁴ It is known that this incidence of HIV-related TTP-HUS decreased until the introduction of highly active antiretroviral therapy (HAART).⁴ The registry enrolled all patients from January 1, 1989, to December 31, 2001, thus including a period before the HAART era. There is no explanation for this low number of HIV-positive patients.

Concerning the treatment, Vesely et al argue that the standard practice is to treat all adult patients with plasma exchange. This conviction is based mainly on the result of a large prospective study reporting therapeutic plasma exchange to be superior to plasma infusion.⁵ In this study, however, the volumes of plasma administered in the plasma infusion group were low compared with those of the therapeutic plasma exchange group. A previous report suggests that high-dose plasma infusion may be as effective, less cumbersome, and less invasive than therapeutic plasma exchange.⁶ The efficiency of high-dose plasma infusion (> 30 mL/kg per day) has been confirmed in a larger and more recent study.⁷ We think that the use of therapeutic plasma exchange is of limited interest in the case of bone marrow transplantation–associated TTP-HUS. A recent review reports a low response rate (45%) and an overall low survival (18%) in

this special form of TTP-HUS.⁸ After correcting high blood pressure and blocking the renin-angiotensin system, drugs such as tumor necrosis factor α (TNF- α) antagonist, defibrotide, or nitric oxide donors may be superior to plasma exchange.⁸

We are surprised by the rate of death that, except for pregnancy/postpartum-related TTP-HUS, is between 40% (bloody diarrhea) and 100% (stem cell transplantation).¹ In a recent study of 30 patients admitted to the ICU for management of TTP-HUS (mean simplified acute physiology score II [SAPS II] 37.3 ± 18.7 ,⁹ 57% under mechanical ventilation), only 27% of patients died.¹⁰ Of most importance is that 9 of 30 patients were infected with HIV, one of the main factors influencing the prognosis in multivariate analysis in our report (odds ratio [OR], 20.3; range, 2.5-167.5; $P = .0002$).² The death rate of TTP-HUS patients is especially high in patients with moderate deficiency or normal levels of ADAMTS13 activity (respectively, 61% and 35%). Is it possible to conclude that plasma exchange is of poor efficacy in the case of hemolytic and uremic syndrome, and of value in the case of thrombotic thrombocytopenic purpura detected by a severe deficiency (< 5% activity)¹¹ of ADAMTS13? This hypothesis was suggested in a recent review by Tsai.¹² We believe that adjuvant treatments such as corticosteroids and antihypertensive agents are of great importance in hemolytic uremic syndrome. Finally, we agree that ADAMTS13 deficiency will not detect all patients with TTP-HUS who may respond to treatment.

François Vincent, Marie-Alyette Costa, and Eric Rondeau

Correspondence: François Vincent, Service de Néphrologie A, Hôpital Tenon, 4 Rue de la Chine, 75020 Paris, France; e-mail: fmcsvncnt@aol.com

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