

due emphasis was placed on the presence of MGUS in this patient. Critically, the history of invasive, recurrent pneumococcal infection, a well-established clinical feature of HIV infection, should have sufficed to alert the clinicians to the possibility of HIV infection.² Furthermore, it would have been interesting to know his lymphocyte count during convalescence, since patients with recurrent pneumococcal infection and HIV have low CD4 T-cell counts at presentation.²

Although a spectrum of immunoglobulin abnormalities, ranging from polyclonal hypergammaglobulinemia to MGUS, are well recognized in HIV infection, we do not believe that this case report constitutes sufficient evidence to warrant screening all young patients with MGUS for HIV infection. We would contend that this case highlights the importance of screening young patients with recurrent pneumococcal infection for HIV infection, rather than screening young patients with MGUS for HIV infection.

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1. Lu CM, Dezube BJ, Pantanowitz L. HIV infection masquerading as monoclonal gammopathy of undetermined significance. *N Engl J Med* 2003;349:1192-3.

2. Turett GS, Blum S, Telzak EB. Recurrent pneumococcal bacteremia: risk factors and outcomes. *Arch Intern Med* 2001;161:2141-4.

THE AUTHORS REPLY: We wholeheartedly agree with Drs. Aslam and Misbah that young patients with recurrent pneumococcal infection should be screened for HIV infection. However, their recommendation does not negate our recommendation that young people with MGUS also be screened for HIV infection, particularly those at increased risk for HIV infection. Indeed, had our patient been tested for HIV infection when his initial MGUS was detected, his recurrent pneumococcal infections might have been avoided. Therefore, we do not believe that undue emphasis was placed on his MGUS. In fact, the emerging literature appears to support our initial proposal to consider HIV infection as a cause of MGUS in young patients.¹ The only CD4 cell count obtained in this young man's case (9 per cubic millimeter) was on his final admission to the hospital. Before that, a CD4 cell count was not ordered because a diagnosis of HIV infection was not entertained.

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1. Pantanowitz L, Dezube BJ. Multiple myeloma and HIV infection — causal or casual coincidence? *AIDS Read* 2003;13:386-7.

Bulimia Nervosa

TO THE EDITOR: In Dr. Mehler's discussion of the physiological complications of bulimia nervosa (Aug. 28 issue),¹ he emphasizes the diagnostic usefulness of urinary electrolyte measurements and concludes that low urinary potassium and sodium concentrations are compatible with vomiting. The amount of potassium in gastric secretions is actually trivial, and the mechanism of hypokalemia in such persons is urinary potassium loss, which occurs as high distal delivery of bicarbonate enhances potassium secretion in the cortical collecting duct.²⁻⁴ In addition, there is an obligatory loss of sodium with bicarbonate after each bout of vomiting. Therefore, the urinary hallmarks of active vomiting are an elevated potassium concentration, a low urinary chloride concentration, and depending on the degree of urinary bicarbonate, sodium loss. Only urinary electrolyte measurements performed at a time that is

remote from the episode of vomiting and in the absence of distal bicarbonate delivery might show low urinary potassium and sodium concentrations.

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1. Mehler PS. Bulimia nervosa. *N Engl J Med* 2003;349:875-81.

2. Kassirer JP, Schwartz WB. Correction of metabolic alkalosis in man without repair of potassium deficiency. *Am J Med* 1966;40:19-26.

3. Carlisle EJ, Donnelly SM, Ethier JH, et al. Modulation of the secretion of potassium by accompanying anions in humans. *Kidney Int* 1991;39:1206-12.

4. Kamel KS, Ethier JH, Richardson RM, Bear RA, Halperin ML. Urine electrolytes and osmolality: when and how to use them. *Am J Nephrol* 1990;10:89-102.

THE AUTHOR REPLIES: Dr. Weinstein is concerned about the discussion of the role of urinary electro-

lytes in the diagnosis of bulimia. I agree with him that with vomiting, most of the potassium loss is in the urine as a result of metabolic alkalosis and the bicarbonate diuresis that ensues, resulting in loss of potassium as the accompanying cation. Rates of renal potassium secretion are further increased in the presence of an elevated aldosterone level secondary to volume contraction, causing worsening hypokalemia. My comment about the usefulness of finding a low urinary potassium level in a patient with bulimia was in reference to diarrheal states due to laxative abuse and to the differentiation of this mode of purging from vomiting, wherein the uri-

nary potassium level is elevated. However, there is an error in Table 2 of the article: urinary levels of potassium are indeed increased with excessive vomiting, not decreased, as printed.

The key points of the paragraph to which Dr. Weinstein refers remain the same — namely, that hypokalemia is highly specific for the diagnosis of bulimia in otherwise healthy young women and that patients with strict nonpurging anorexia nervosa should not have metabolic disturbances.

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Molecular Epidemiology of Tuberculosis

TO THE EDITOR: In their excellent review of the molecular epidemiology of tuberculosis, Barnes and Cave (Sept. 18 issue)¹ recommend that the Centers for Disease Control and Prevention (CDC) support newer, more rapid genotyping tests. In fact, the CDC is doing just that. This winter, two genotyping laboratories will use two polymerase-chain-reaction–based methods — mycobacterial-interspersed-repetitive-units (MIRU) analysis² and spoligotyping³ — for primary genotyping of *Mycobacterium tuberculosis* isolates. The laboratories will have the capacity to analyze isolates from every patient with culture-positive tuberculosis in the United States and will report results in less than two weeks. In combination, these methods are highly discriminatory. For isolates with identical MIRU and spoligotype patterns, a third genotyping method, IS6110-based fingerprinting,⁴ will be available, if needed, to provide further discriminatory power. We anticipate that rapid access to genotyping results will be a powerful tool for controlling and preventing tuberculosis.⁵

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1. Barnes PF, Cave MD. Molecular epidemiology of tuberculosis. *N Engl J Med* 2003;349:1149-56.

2. Mazars E, Lesjean S, Banuls AL, et al. High-resolution minisatellite-based typing as a portable approach to global analysis of *Mycobacterium tuberculosis* molecular epidemiology. *Proc Natl Acad Sci U S A* 2001;98:1901-6.

3. Kamerbeek J, Schouls L, Kolk A, et al. Simultaneous detection and strain differentiation of *Mycobacterium tuberculosis* for diagnosis and epidemiology. *J Clin Microbiol* 1997;35:907-14.

4. Van Embden JDA, Cave MD, Crawford JT, et al. Strain identification of *Mycobacterium tuberculosis* by DNA fingerprinting: recommendations for a standardized methodology. *J Clin Microbiol* 1993;31:406-9.

5. McNabb SJN, Braden CR, Navin TR. DNA fingerprinting of *Mycobacterium tuberculosis*: lessons learned and implications for the future. *Emerg Infect Dis* 2002;8:1314-9.

TO THE EDITOR: The review article on the molecular epidemiology of tuberculosis by Barnes and Cave is excellent. We do, however, want to point out that more than 95 percent of the new cases of tuberculosis in the world occur in developing countries, especially in the 22 high-burden countries.¹ The lack of studies on the dynamics of tuberculosis in these settings with the use of new molecular techniques is alarming.

We advocate a major effort to establish a collaboration in tuberculosis research between developed and developing nations in order to shed light where it is most needed. Molecular epidemiology can be an important tool for understanding the current tuberculosis pandemic and for influencing the design of future policies in global tuberculosis control.

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1. Global tuberculosis control: surveillance, planning, financing. WHO report 2003. Geneva: World Health Organization, 2003. (WHO/CDS/TB2003.316.)