

University of Groningen

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Published in:
Annals of Internal Medicine

DOI:
[10.7326/0003-4819-153-6-201009210-00014](https://doi.org/10.7326/0003-4819-153-6-201009210-00014)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2010

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):
Opstelten, W., van Essen, G. A., & Hak, E. (2010). Barriers to Herpes Zoster Vaccination. *Annals of Internal Medicine*, 153(6), 418-418. DOI: 10.7326/0003-4819-153-6-201009210-00014

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COMMENTS AND RESPONSES

Barriers to Herpes Zoster Vaccination

TO THE EDITOR: Hurley and colleagues (1) showed that the major barriers that keep physicians from using the herpes zoster vaccine are the cost concerns of their patients and their own worries about freezer storage of the vaccine. These barriers may have resulted in low vaccine uptake. With respect to these findings, we would like to make some comments based on our prospective questionnaire study in the Netherlands (2). In our primary care study, we assessed the willingness of elderly patients to accept free herpes zoster vaccination simultaneously with their annual influenza vaccination. The participating primary care physicians were equipped free of charge with adequate storage facilities. In all, only 39% of the invited patients accepted the herpes zoster vaccination, whereas 76% accepted the flu vaccination. The major determinants of nonadherence with the herpes zoster vaccination were perceived lack of recommendation by the physician, unwillingness to adhere to the physician's advice, and the perception of low risk for herpes zoster. These results show that removing the issues of cost and freezer storage space, which Hurley and colleagues identified, may not lead to desirable rates of herpes zoster vaccination. To increase the acceptance of vaccination, more information is needed for both patients and physicians about the effect of herpes zoster in elderly persons and about the efficacy and cost-effectiveness of the vaccine. Furthermore, in Europe, a refrigerator-stable herpes zoster vaccine that can be stored at temperatures between 2 °C and 8 °C has been registered. This may end the need for freezer storage (3).

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Potential Conflicts of Interest: None disclosed.

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IN RESPONSE: We thank Dr. Opstelten and colleagues for their comments and agree that solving the financial and freezer storage issues we discovered would not remove all barriers to herpes zoster vaccine uptake in the United States. However, the results of our study and those of Opstelten and colleagues' study (1) differ in a few

important ways that make direct comparison problematic. First, Opstelten and colleagues' study was done in the Netherlands, and the herpes zoster vaccine was not recommended there at that time; therefore, physicians and patients were probably not familiar with it. Our study was done in the United States at a time when the herpes zoster vaccine had been recommended for almost 2 years. Second, health care systems and financing of care differ between the Netherlands and the United States in ways that may markedly affect population responses. Third, the study by Opstelten and colleagues (1) presents the patient perspective, whereas our study presents the provider perspective. Although we did not find the perceived lack of serious sequelae from herpes zoster and postherpetic neuralgia to be a major barrier to administering herpes zoster vaccine to providers, U.S. patients could perceive it as a larger barrier. The 2007 National Immunization Survey—Adult (2), which provides some insight into the patient perspective of herpes zoster vaccination in the United States, found that 77.8% of respondents who had not yet received the herpes zoster vaccine would accept it (hypothetically) if recommended by their physician. It would be interesting to do a study in the United States similar to that of Opstelten and colleagues, where patients would have the option of receiving the herpes zoster vaccine for free, rather than theorizing on what patients might do if offered it, as in the National Immunization Survey—Adult survey.

We agree with Dr. Opstelten and colleagues that greater awareness of herpes zoster vaccine in patients may lead to greater acceptance. Patients in the United States are probably less aware of the herpes zoster vaccine than of other routinely recommended vaccines because the manufacturer has had supply constraints and, as a result, the vaccine has not been actively promoted in the United States. Dr. Opstelten and colleagues also note that a refrigerator-stable herpes zoster vaccine (3) has been registered in Europe, which is another crucial issue. Besides the herpes zoster vaccine, the other varicella-containing vaccines have been available in international markets in refrigerator-stable formulations (4). Hopefully, these vaccines will become available in the United States, because the requirement for freezer storage is an important barrier to all vaccine programs.

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Sixteen years of global experience with the first refrigerator-stable varicella vaccine (Varilrix). *BioDrugs*. 2008;22:387-402. [PMID: 18998756]

Important Differences in Measurement of Fetuin-A

TO THE EDITOR: Parker and colleagues (1) found no significant association between fetuin-A levels and mortality in a cohort with prevalent coronary artery disease. Our understanding of fetuin-A biology is greatly undermined by the lack of agreement among different methods of measurement. It is therefore particularly important that method descriptions are unambiguous. The authors comment that their nephelometric fetuin-A assay “uses the same high-specificity antibody as commercially available enzyme-linked immunosorbent assays.” It is unclear to which commercially available assays they are referring—the 2 most widely used commercial kits not only use different antisera but also use a sandwich format with 2 different antibodies that recognize spatially distinct molecular epitopes. This statement implies a degree of equality with other methods that we have not found to be the case (2). The authors justify use of this method by referring to a study (3) in which use of the same fetuin-A assay showed an association between fetuin-A levels and mortality in patients receiving hemodialysis. However, it does not necessarily follow that this method is powered to detect significant associations in other settings, associations that may be masked by a lack of specificity to the biologically relevant fraction.

The poor agreement between commercial assays for fetuin-A does not represent a mere analytical nuance but has critical implications for observations derived from their measurements. For example, in a cohort of patients with mild-to-moderate chronic kidney disease, we showed that fetuin-A was an independent predictor of progressive aortic stiffness (4), a vascular variable known to be predictive of cardiovascular events and mortality. This relationship was lost, even in simple bivariate correlation, by measuring fetuin-A with an alternative assay. Our experiments suggest that the lack of agreement in both chronic kidney disease as well as non-chronic kidney disease settings reflects variation in antibody specificity for different glycosylated forms of fetuin-A (2). Although Parker and colleagues correctly acknowledge that “results may differ with other assays,” it remains a relatively unappreciated point that differences in antibody specificity to different modified forms of the same protein (a heterogeneous mixture) can mask potentially important associations. In the case of fetuin-A, have the investigators considered further study using alternative methodology?

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Potential Conflicts of Interest: None disclosed.

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IN RESPONSE: We appreciate Drs. Smith and Holt’s insightful comments about the importance of assay characteristics and agree that associations between particular measurements and outcome might be influenced by the assay. Moreover, test specificity and reliability might depend on factors present in the participants and specimens being evaluated.

We used a fetuin-A assay developed by our group in Aachen, Germany, which is not commercially available. This assay has been described in greater detail in our prior articles (1) and was used initially to show the associations of fetuin-A with mortality in patients with end-stage renal disease (2). We evaluated the nephelometric method for low fetuin-A serum measurement in a side-by-side comparison with immunoblot analysis to exclude cross-reactivity of the antibodies with other serum proteins and proteolytic fragments of fetuin-A. We calculated final serum fetuin-A concentrations by regression analysis of a serial dilution curve obtained from standard serum. For comparison and reliability testing, we prepared a control solution of purified serum fetuin-A powder (Boehringer Mannheim, Mannheim, Germany, and Dade-Behring, Marburg, Germany). For both methods, we used the polyclonal rabbit antihuman fetuin-A antibody that does not cross-react with fetuin-B.

In regard to similarity in findings with other groups and assays, we have shown that higher fetuin-A levels were associated with the metabolic syndrome (1) and diabetes mellitus by using this assay. Moreover, we have shown that lower fetuin-A levels were associated with aortic stenosis, an association that was present only in participants without diabetes (3). These data are similar to findings from Ford and colleagues (4) using the BioVendor fetuin-A assay (BioVendor, Candler, North Carolina). The study found lower fetuin-A levels were associated with progression of arterial stiffness (potentially a consequence of greater arterial calcium deposition), an association that was also limited to persons without diabetes. Nonetheless, we are uncertain of the correlation between our assay and that from BioVendor. We agree that future studies with multiple assays may be useful to show their correlations to one another and to determine which has the strongest associations with arterial calcification, arterial stiffness, and cardiovascular disease events in different settings.

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Urine Drug Testing Is Still an Invaluable Resource for Primary Care

TO THE EDITOR: We commend Starrels and colleagues (1) for their excellent systematic review and wholeheartedly agree that more rigorous studies are needed to determine the roles of treatment agreements and urine drug testing (UDT) to reduce misuse of prescription opioids. The authors could have emphasized, however, that in addition to reducing opioid misuse, for which they found weak supportive evidence, UDT plays an invaluable role in detecting abuse of and addiction to prescribed opioids, illicit drugs, and nonprescribed controlled substances, as well as diversion of prescribed opioids. The abuse of prescription opioids in the United States has reached epidemic proportions. Heit and Gourlay (2), in their accompanying editorial, point out that the number of new nonmedical users of prescription opioids has equaled or exceeded the number of new users of marijuana in recent years. The provenance of the opioids and the consequences of their use were left unstated. As prescriptions for opioid analgesics have increased dramatically in recent years, there have been parallel increases in emergency department mentions, hospitalizations, and deaths related to these medications. In fact, rates of poisoning mortality involving opioid analgesics have been exponentially higher than those involving heroin and cocaine (3). It should be sobering to all clinicians that most of the opioids destined for nonmedical use in the United States originate from valid physician prescriptions (4). Reducing misuse of prescription opioids remains a vexing problem and depends on multiple patient, physician, and system factors. However, the utility of UDT in the detection of opioid-related problems is unequivocally supported by data, such as the seminal study by Katz and colleagues (5), which shows that UDT is perhaps the most important surveillance technique for detecting illicit drug use in long-term opioid analgesic therapy.

Our concern is that the article by Starrels and colleagues, although highlighting the dearth of quality evidence supporting the role of UDT in reducing opioid misuse, devotes only a single sentence to its role in detecting opioid and other substance use disorders. As a result, it may provide yet another pretext for most primary care physicians who prescribe opioid analgesics but never perform drug testing (6) to continue not testing, thereby wasting valuable

opportunities to identify and address drug-related problems before they end in tragedy.

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Potential Conflicts of Interest: None disclosed.

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IN RESPONSE: We agree with Dr. Reisfield and colleagues that UDT is a valuable tool for monitoring patients receiving long-term opioid therapy for chronic pain, but our endorsement of its use in primary care is tempered by its limitations. Urine drug testing can detect use of illicit or nonprescribed drugs that is not disclosed by patients, which is essential to managing the risks associated with prescribed opioids. When ordered and interpreted correctly, UDT can also detect nonuse of prescribed opioids. However, although UDT detects these 2 types of aberrant behavior, current evidence does not support the claim that it can detect the clinical diagnoses of opioid use disorders (that is, abuse and dependence) or the crime of diversion (that is, selling or giving away). Indeed, Katz and colleagues' article (1), referenced by Dr. Reisfield and colleagues, found that UDT is effective in detecting "behaviors suggestive of inappropriate medication use." To detect the more serious outcomes of abuse, dependence, or diversion, serial UDT results need to be interpreted over time along with data from other sources, including patient interviews; physical examination; pill counts; and monitoring of patient behaviors, such as requests for early refills.

Although we highlight gaps in the scientific literature about UDT, we agree that the public health threat posed by opioid misuse is significant and that UDT may be useful for detecting proximal outcomes (inappropriate drug use or nonuse). However, a study of 80 primary care physicians by Reisfield and colleagues (2) showed that even those who performed UDT had poor knowledge of how to interpret the results. Accurate interpretation of UDT results requires an understanding of the type of assay ordered, major and minor

opioid metabolic pathways, expected drug detection times, and potential causes of false-positive and false-negative results. Misinterpretation of these results can harm the physician-patient alliance or patient well-being. For example, we are aware of physicians who inappropriately discontinued synthetic opioids (that is, fentanyl) because they thought the negative results of an immunoassay-based UDT for natural opiates (that is, morphine, codeine) signified evidence of diversion. We recommend that physicians become better educated about the use and limitations of UDT (3) and consult their laboratory toxicologist with questions about which test to order and how to interpret abnormal results. We also encourage researchers to continue to investigate best practices regarding UDT and other risk management strategies for patients who are prescribed long-term opioids.

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Potential Conflicts of Interest: None disclosed.

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Guidelines and Conflicts of Interest

TO THE EDITOR: Guyatt and colleagues (1) group financial conflicts of interest with intellectual conflicts of interest. I find this puzzling. Financial conflicts of interest are of concern because they do not coincide with and are not accountable to research or clinical aims. But "intellectual conflicts of interest," according to Guyatt and colleagues, are to be found in persons with substantive knowledge, engagement, or research investment in the question at issue (indeed, according to the authors' view, they themselves would be disqualified from primary authorship of any future expert guideline on intellectual conflicts of interest). But those are precisely the people whose opinions I want to hear.

The issue here is the misguided notion that evidence-based medicine is the view from nowhere (2), a wholly objective enterprise where evidence is weighed and summarized without contamination by opinion. Evidence is never considered this way (3). Evidence-based medicine depends on those who have thought-out, well-

informed opinions. It couldn't be done without intellectual conflicts of interest.

Better to define what makes intellectual content worthy of exclusion (is it based on faulty data, suspicious borrowings, or retracted publications?) than to try to eliminate what makes intellectual discussion possible: the strongly held opinions and long-term investment of experts.

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Potential Conflicts of Interest: None disclosed.

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TO THE EDITOR: In the aftermath of the scandals over clinical guidelines written by experts with major conflicts of interest, Rothman and colleagues (1) suggested that authors of guidelines of professional medical associations be free of any financial conflicts of interest. Guyatt and colleagues (2) have devised a complex solution to put investigators with conflicts of interest back into action. Experts with important financial conflicts can collect and interpret evidence, but only panel members who are conflict-free can be involved in developing the recommendations for a specific question. Guyatt and colleagues say that investigators with conflicts of interest may offer unique insights into the clinical context and evidence. No data, however, substantiate the claim that these experts are better qualified in certain respects than those who chose to have no financial ties. Indeed, Guyatt and colleagues anticipate that collection and interpretation of findings may be potentially biased by conflicts of interest but rely on methodologists, who have little or no clinical understanding of the issues under discussion, for balancing the final outcome. How will these methodological heroes circumvent subtle manipulations and the unavoidable external pressures?

Rothman and colleagues (1) deserve credit for raising the issue of intellectual conflicts of interest, which are part of the more general problem of nonfinancial sources of conflict (3). However, the authors do not set specific thresholds for all sorts of conflicts of interest. I have outlined criteria for establishing the presence of a substantial conflict (3), based on the work of Krimsky and colleagues (4): being an employee of a private company, being a regular consultant or on the board of directors of a company, being a stockholder of a company related to the field of research, or owning a patent directly related to the published work. Occasional consultancies, grants for performing investigations, or receiving honoraria or refunds in specific occasions would not be a source of substantial conflict. Similarly, there are several steps for setting a threshold to and addressing nonfinancial conflicts of interest in medical research (3).

Are guidelines necessary? Guyatt and colleagues argue that without them, clinicians would have to undertake their own reviews. I agree that spending nights performing meta-analyses for deciding what to prescribe the next morning would be unrealistic. But re-

views, case management articles, and updates were available in the medical literature well before guidelines became fashionable. In journals that claim independence, guidelines should be reserved for investigators without substantial conflicts of interest (3). Actually, the problem of guidelines and conflicts of interest is only part of the general problem of professional medical organizations. I have outlined some suggestions for fostering creativity and independence in professional medical organizations (3), such as complete change of leadership and inclusion of physicians who are not experts in the specific field. Unless profound changes occur, professional medical organizations may no longer have the credibility for issuing guidelines. What credibility is left (and it is not much) should not be put at risk, because when trust goes, so does the healing power of physicians (5).

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Potential Conflicts of Interest: None disclosed.

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IN RESPONSE: Dr. Berger wants to hear the opinions of authors with intellectual conflicts of interest. We agree, which is why we ensure the active engagement of these experts in the process of gathering, summarizing, and interpreting the evidence used in our guidelines.

Dr. Fava implies that we should be recruiting experts without financial conflicts of interest to our guideline panels. In areas of investigation in which most world leaders are free of financial conflicts, this may be a good idea. However, in several areas, including thrombosis, many of the international authorities have financial conflicts. Excluding them from gathering and interpreting the evidence would result in lower-quality guidelines.

Dr. Fava suggests that our methodologist chapter editors have "little or no clinical understanding of the issues under discussion," which shows insufficient respect for the capacity of intelligent scientists to, after detailed review and discussion of the evidence with leaders in the field, understand the relevant clinical issues. Nevertheless, we agree that clinical expertise is important for optimum understanding. That is why, among our 13 methodologist chapter editors, we have recruited 10 specialists in internal medicine, 1 surgeon, and 1 primary care physician, and why the 3 methodologist editors with ultimate responsibility for the guidelines are also specialists in inter-

nal medicine. Other organizations (the World Health Organization [1], the Allergic Rhinitis and Its Impact on Asthma guideline panel [2], and the World Allergy Organization [3]) have successfully placed major responsibility on nonexpert clinician-methodologists in recent guidelines.

Dr. Fava notes that we did not provide specific thresholds for all sorts of conflicts of interest and cites guidelines from his own and others' work. We do have detailed criteria; however, the word count limitations of the article prevented their presentation in the *Annals*, but they are available for anyone interested (e-mail, guyatt@mcmaster.ca).

Dr. Fava challenges the need for practice guidelines. The popularity of recommendations to guide practice attests to their necessity. Formal structured processes of recruiting panelists with methodological and content expertise; summarizing and interpreting evidence regarding desirable and undesirable consequences of alternative management strategies; summarizing and interpreting evidence regarding patients' values and preferences; specifying the preferences underlying recommendations; and developing and applying rules for participation in decision making that consider financial and intellectual conflicts of interest are certain to result in higher-quality recommendations. Investigation addressing the effect of financial and intellectual conflicts and associated guideline policies could inform what is certain to be an ongoing debate.

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CLINICAL OBSERVATION

A Rare Cause of Cardiac Ischemia: Systemic Lupus Erythematosus Presenting as the Hyperviscosity Syndrome

Background: The hyperviscosity syndrome can cause cardiac ischemia by impairing microcirculation. This syndrome with cardiac ischemia can be caused by erythrocytosis, leukocytosis, hypercholesterolemia, or paraproteinemia (1). The hyperviscosity syndrome from paraproteinemia more commonly results from the monoclonal

Table. Laboratory Values at Admission and Serial Measurements of Cardiac Enzymes and Plasma Viscosity

Admission Hematology Panel	Value
Leukocyte count (range), $\times 10^9$ cells/L	4.5 (4.5 to 11.0)
Hemoglobin level (range), g/L	97 (120 to 160)
Hematocrit (range), %	28.4 (37 to 48)
Platelet count (range), $\times 10^9$ cells/L	117 (150 to 350)
Leukocyte differential (range), %	
Lymphocytes	46 (24 to 44)
Monocytes	13 (2 to 11)
Neutrophils	39 (40 to 70)
Immature granulocytes	0.2 (0.0 to 0.1)
Eosinophils	1.3 (1 to 4)
Basophils	0.2 (0.0 to 2.0)
Reticulocytes	1.5 (0.5 to 1.8)
Prothrombin time (range), s	15.5 (9.6 to 11.5)
Activated partial prothrombin time (range), s	33.0 (22.8 to 33.3)
International normalized ratio (range)	1.5 (0.9 to 1.1)
ESR (range), mm/h	>130 (4 to 25)
C-reactive protein (range), nmol/L	0.29 (0.0 to 0.48)
Admission Complete Metabolic Panel	Value
Sodium level (range), mmol/L	131 (135 to 148)
Potassium level (range), mmol/L	4.0 (3.5 to 5.1)
Chloride level (range), mmol/L	104 (96 to 109)
Bicarbonate level (range), mmol/L	23 (21 to 31)
Glucose level (range)	
mmol/L	6.4 (3.33 to 5.49)
mg/dL	116 (60 to 99)
Blood urea nitrogen level (range), mmol/L	8.6 (2.5 to 7.9)
Creatinine level (range)	
mmol/L	97.24 (44.2 to 106.1)
mg/dL	1.1 (0.5 to 1.2)
Calcium level (range)	
mmol/L	2.1 (2.1 to 2.6)
mg/dL	8.5 (8.4 to 10.5)
Phosphate level (range)	
mmol/L	1.6 (0.87 to 1.5)
mg/dL	5.1 (2.7 to 4.5)
Magnesium level (range)	
mEq/L	0.85 (0.65 to 1.0)
mg/dL	1.7 (1.3 to 2.0)
Total protein level (range), g/L	129 (60 to 82)
Albumin level (range), g/L	24 (35 to 53)
Aspartate aminotransferase level (range), U/L	82 (0 to 31)
Alanine aminotransferase level (range), U/L	41 (0 to 31)
Alkaline phosphatase level (range), U/L	43 (30 to 120)
Time of Serial Relative Plasma Viscosity Ratio Measurement	Ratio (Plasma-Saline)
Admission	16.0
24 h (after first plasmapheresis)	6.0
2 d	6.0
3 d (after second plasmapheresis)	2.7
5 d	2.9
9 d	3.6
10 d	3.7

gammopathies of multiple myeloma and Waldenström macroglobulinemia and less commonly from the polyclonal gammopathies of rheumatoid arthritis, the Sjögren syndrome, chronic hepatitis, and

Table—Continued

Time of Serial Cardiac Enzyme Measurement	Value
Admission	
CK-MB fraction, $\mu\text{g/L}^*$	—
CK index, %†	—
Troponin I level, ng/mL‡	0.10
14 h	
CK-MB fraction, $\mu\text{g/L}^*$	12
CK index, %†	4
Troponin I level, ng/mL‡	0.14
18 h (after first plasmapheresis)	
CK-MB fraction, $\mu\text{g/L}^*$	14
CK index, %†	4
Troponin I level, ng/mL‡	0.13
3 d	
CK-MB fraction, $\mu\text{g/L}^*$	15
CK index, %†	5
Troponin I level, ng/mL‡	0.08
5 d (after second plasmapheresis)	
CK-MB fraction, $\mu\text{g/L}^*$	18
CK index, %†	5
Troponin I level, ng/mL‡	0.14

CK-MB = creatine kinase-MB; ESR = erythrocyte sedimentation rate.

* Range, 0 to 7.

† Range, 0 to 3.

‡ Range, 0.00 to 0.06.

some rare lymphoproliferative disorders (2). To the best of our knowledge, only 3 case reports (3–6) have described the hyperviscosity syndrome in systemic lupus erythematosus (SLE), and only 1 of these reports (6) described hyperviscosity as the initial manifestation.

Objective: To describe cardiac ischemia from the hyperviscosity syndrome in a patient with previously undiagnosed SLE.

Case Report: We accepted transfer to our cardiac intensive care unit of a 47-year-old African-American woman with chest pain and dyspnea whose physicians suspected the acute coronary syndrome. Her presentation was atypical because she had recurrent chest pain and dyspnea for 2 months but new onset of left ankle pain, dizziness, and blurry vision. Two months before transfer, her physicians diagnosed anemia, cardiomyopathy with an ejection fraction of 0.35, biventricular hypertrophy, pulmonary hypertension, and polyclonal IgG gammopathy of undetermined origin.

On admission to the intensive care unit, she seemed weak and tired. She was afebrile and normotensive, with a heart rate of 80 beats/min. She reported scalp alopecia and a 100-lb weight loss during the past year. On examination, we found clear lungs, a loud P2, a tender left ankle without swelling or erythema, intact extraocular movements, and normal visual acuity. Electrocardiography revealed normal sinus rhythm, no chamber enlargement, and nonspecific T-wave inversions. The phlebotomist noted the patient's blood to be "thick," because the initial blood samples clotted. Initial laboratory studies included elevated troponin I (0.10 ng/mL) and paraprotein (nonalbumin protein, 10.5 g/dL) levels. The patient's plasma viscosity ratio was 16.0 (plasma-saline), and we initiated emergency plasmapheresis for the hyperviscosity syndrome.

Although our patient had ankle pain, she did not have the morning stiffness or joint pain characteristic of rheumatoid arthritis. She also did not have the keratoconjunctivitis sicca and xerostomia characteristic of the Sjögren syndrome. An inguinal lymph node biopsy with flow cytometry ruled out lymphoproliferative disorders.

She had positive test results for antinuclear antibodies (ANA) (titer, 640), rheumatoid factor, anti–double-stranded DNA (anti-dsDNA), antiribonucleoprotein antibodies, and anti–68-kD antibodies. Her C3 and C4 levels were within normal limits. We diagnosed SLE on the basis of her alopecia, anemia, positive ANA results, and positive anti-dsDNA results.

Her physicians discharged her from the hospital 10 days later with prescriptions for dexamethasone, warfarin, and sulfamethoxazole–trimethoprim in addition to her admitting medications. She did not take her medications as they were prescribed; developed worsening headaches, dizziness, and difficulty sleeping 6 months later; and was admitted to another hospital for recurrent hyperviscosity syndrome. Her condition is currently maintained successfully with prednisone therapy.

Discussion: Patients with the hyperviscosity syndrome can present with constitutional, neurologic, and cardiovascular symptoms (2). Symptoms are not expected until plasma viscosity increases above a plasma–saline ratio of 4.0:5.0. Immediate plasmapheresis reduces the risk for neurologic complications, and 75% of patients with plasma viscosities greater than 5.0 have reductions in neurologic and other symptoms with plasmapheresis (3). Our patient required 2 sessions of plasmapheresis to reduce plasma viscosity to 2.7.

Non–ST-elevation cardiac ischemia can occur in the acute coronary syndrome, subendocardial ischemia, coronary artery dissection, coronary artery vasospasm, coronary embolism, and the hyperviscosity syndrome. We believe that hyperviscosity was the sole cause of our patient’s cardiac ischemia, because she had a relatively mild troponin I elevation and cardiac enzyme levels and

symptoms returned to normal when plasmapheresis reduced the viscosity ratio (Table).

Conclusion: Our patient had cardiac ischemia caused by a hyperviscosity syndrome from the paraproteinemia of SLE.

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Potential Conflicts of Interest: None disclosed.

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