Zhou et al state that “recent evidence-based recommendations for the management of patients with herpes zoster recommend using antiviral therapy to decrease the incidence of PHN,” citing an article for which I am a co-author.1 This article stated that antiviral therapy should be used to treat patients with herpes zoster and that it had an impact on acute neuritis. Although the guidelines concluded that the use of antiviral therapy may have an effect on “chronic pain,” this therapy was not sufficient to uniformly prevent PHN. As noted here, the FDA reached a similar conclusion.

The goal of management of herpes zoster is to accelerate healing, prevent complications, and decrease pain—both acute pain and PHN. With the evidence available from existing antiviral studies, these end points have been achieved with the exception of prevention of PHN. The management of PHN is of critical importance for individuals who have herpes zoster. Consideration of combination therapy at the onset of disease, particularly in patients with severe acute pain, needs to be further evaluated.4 Future treatment strategies may prevent or provide more significant relief for this potentially devastating complication.

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RESEARCH LETTER

Rhabdomyolysis Associated With 2009 Influenza A(H1N1)

To the Editor: Rhabdomyolysis, which has been associated with infections from various strains of influenza,1 requires prompt clinical suspicion and targeted clinical management to prevent renal failure. We are not aware of prior reports of rhabdomyolysis in patients with 2009 influenza A(H1N1)—induced respiratory failure.

Report of a Case. A 28-year-old woman presented in June 2009 with 1 week of shortness of breath, muscle aches, and fevers. Two days before presentation, she had been prescribed moxifloxacin and hydrocodone/acetaminophen for presumed pneumonia without any significant improvement. She denied any significant medical history or regular medication use prior to her illness, had a family history of sickle cell trait, and smoked 2 to 3 cigarettes daily for 1 year. On examination, she was obese (body mass index, 40; calculated as weight in kilograms divided by height in meters squared), febrile (body temperature, 38.3°C), tachycardic, and tachypneic, with a digital pulse oximetry saturation of 80% on room air.

Her admission laboratory tests included white blood cell count, 2800/µL (normal, 3500-11 000/µL); hematocrit, 45% (normal, 36%-46%); platelet count, 128 × 10³/µL (normal, 140-450 × 10³/µL); potassium, 3.6 mEq/L (normal, 3.5-5.0 mEq/L); ionized calcium, 4.16 mg/dL (normal, 4.64-5.28 mg/dL; to convert to mmol/L, multiply by 0.25); phosphorus, 2.0 mg/dL (normal, 2.5-4.8 mg/dL); serum creatinine, 1.9 mg/dL (normal, 0.5-1.2 mg/dL); lactic dehydrogenase, 1875 U/L (normal, 112-220 U/L); and creatine kinase level, 27 820 U/L (normal, 13-156 U/L). The urinalysis revealed 3+ hemoglobin and 7 red blood cells per high-power field; myoglobin was not assayed. A chest radiograph revealed diffuse bilateral alveolar infiltrates.

Within a few hours of her presentation, the patient was intubated and mechanically ventilated for hypoxemic respiratory failure. She received broad-spectrum antimicrobial agents, including 5 days of oseltamivir, 75 mg twice daily, for suspected 2009 influenza A(H1N1) pneumonia. Real-time reverse-transcriptase polymerase chain reaction (RT-PCR) of nasopharyngeal smear performed at the Santa Clara County Public Health Department was negative for influenza A. Fiberoptic bronchoscopy revealed extremely erythematous and friable mucosa with bloody bronchoalveolar lavage (BAL) fluid that grew influenza A, identified as 2009 H1N1 by the California Department of Public Health laboratory. Treatment with intense hydration (22 L over 96 hours) and intravenous sodium bicarbonate was initiated for suspected rhabdomyolysis, following which there was a progressive reduction in creatine kinase levels (FIGURE). Her creatinine level remained normal. A search for other etiologies of rhabdomyolysis was unrevealing. The patient was ex-

Figure. Creatine Kinase in Patient With 2009 Influenza A(H1N1)

Normal range for creatine kinase is 13 to 156 U/L.

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tubated on hospital day 15 and discharged home 3 days later. She provided consent for publishing this case report, which was approved by the Santa Clara Valley Medical Center human subjects review committee.

Comment. Rhabdomyolysis may occur because of genetic defects, trauma, exertion, muscle hypoxia, body-temperature changes, metabolic and electrolyte disorders, drugs and toxins, and bacterial or viral infections. Although the incidence of rhabdomyolysis associated with viral pneumonia is not clearly defined, a case series of 63 patients with influenza pneumonia showed 9.5% had rhabdomyolysis.

Although the disease spectrum of 2009 H1N1 is unclear, it may have a greater propensity for muscular inflammation than other viral infections or seasonal influenza serotypes. A recent report showed mild to moderate elevation of creatine kinase levels in 62% of patients with 2009 H1N1 pneumonia and respiratory failure. However, the true incidence of rhabdomyolysis in 2009 H1N1 and the mechanisms underlying its pathogenesis remain unknown. It has been presumed that viral invasion, viral toxin, or cytokine may induce myonecrosis causing rhabdomyolysis.

Although real-time RT-PCR has advanced the ability to diagnose 2009 H1N1, a negative nasopharyngeal specimen may not rule out a lower respiratory tract infection, as was seen in this patient. In patients with pulmonary involvement, samples such as BAL may improve the diagnostic yield.

In the setting of a worldwide pandemic, physicians should be aware that rhabdomyolysis may present as a complication in a critically ill patient with 2009 influenza A(H1N1)—induced respiratory failure.

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Study concept and design: Ayala, Kagawa, Wehner.

Acquisition of data: Ayala, Wehner, Tam.

Analysis and interpretation of data: Ayala, Wehner, Upadhyay.

Drafting of the manuscript: Ayala, Kagawa, Upadhyay.

Critical revision of the manuscript for important intellectual content: Ayala, Kagawa, Wehner, Tam, Upadhyay.

Study supervision: Kagawa, Wehner, Upadhyay.

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CORRECTIONS

Inaccurate Statement: In the Clinical Crossroads entitled “A 70-Year-Old Woman With Shingles” published in the July 1, 2009, issue of JAMA (2009;302[1]:73-80), the sentence on page 76, right column, first full paragraph, that stated “A Cochrane review of antiviral treatment for preventing PHN reported no benefit of valacyclovir at 4 or 6 months after rash onset.5” should have stated, “A Cochrane review of antiviral treatment for preventing PHN reported no benefit of acyclovir for reducing the incidence of PHN; there was insufficient evidence to determine the efficacy of other antivirals.”

Incorrect Wording in a Table: In the Clinical Crossroads entitled “A 52-Year-Old Woman With Obesity,” published in the September 9, 2009, issue of JAMA (2009;302[10]:1097-1104), incorrect wording occurred in Table 2 on page 1101. Under the column heading “Additional Findings” for Buchwald et al., 2004, the data labeled “mean estimated weight loss” should have been labeled “mean percentage of excess weight loss” for both types of surgery.