

with major anatomical difficulties, marked edema, or hematoma, a percutaneous technique may be facilitated by ultrasonography.⁵

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TO THE EDITOR: In their video on cricothyroidotomy, Hsiao and Pacheco-Fowler show a technique that is ideal for experienced physicians. However, in some circumstances, cricothyroidotomy may also be performed by paramedics and nurses in the prehospital setting or by relatively inexperienced physicians in the hospital. Many of these

providers do not have tracheal hooks and Trouseau dilators available to them.

In addition, a number of potentially catastrophic complications, such as time delays and placement of the tracheostomy or endotracheal tube in a false passage, are not mentioned in the video.^{1,2} Some studies have reported complication rates as high as 54% in the medical air-transport setting.³

We advocate the use of a gum elastic bougie, which is also called an endotracheal-tube introducer, to facilitate surgical cricothyroidotomy. This approach has been described by MacIntyre et al. as the "three-step" technique.⁴ Our experience in both the cadaver laboratory and prehospital setting indicates that this approach obviates the need for any equipment other than a tracheostomy or endotracheal tube, bougie, and scalpel.

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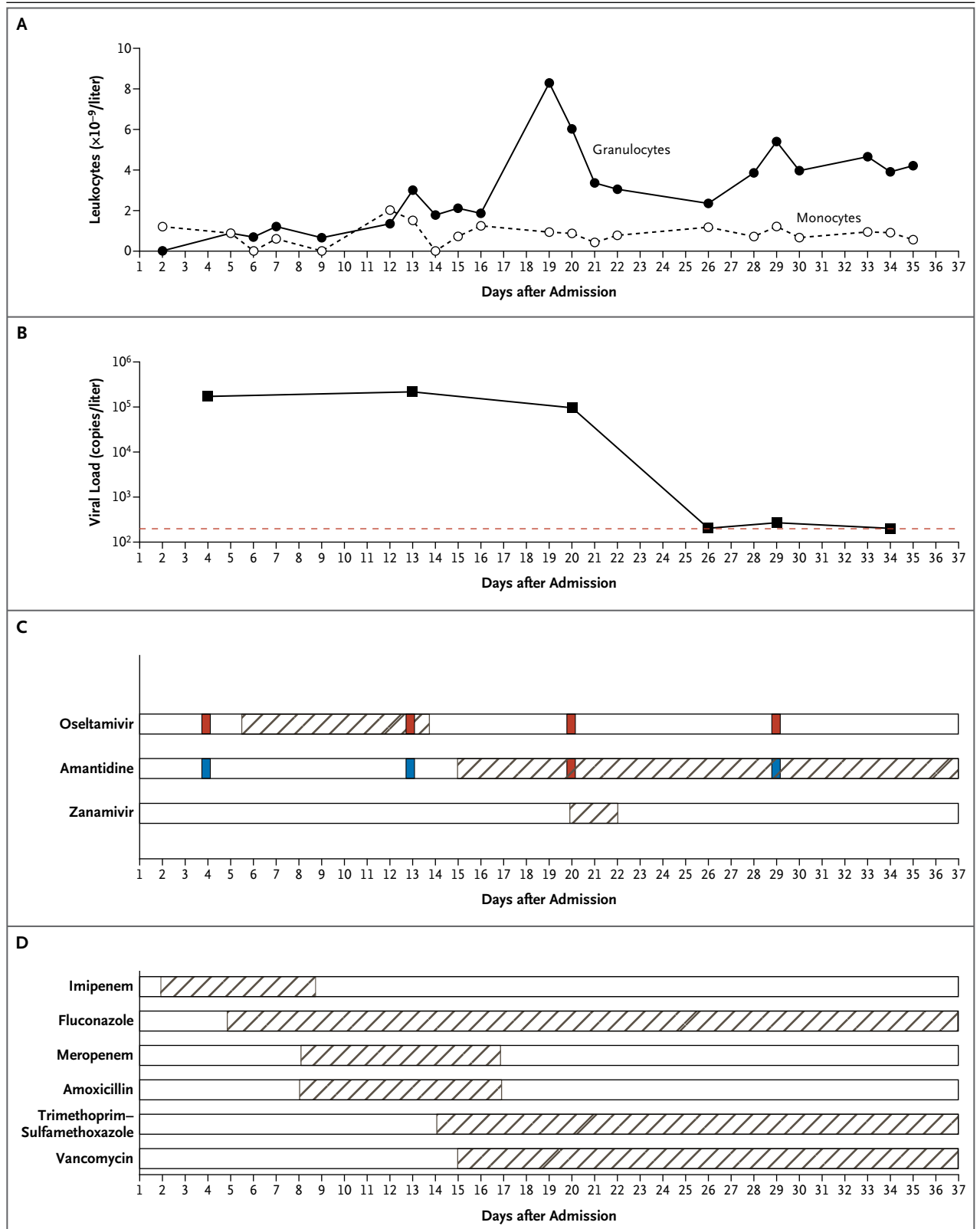
Fatal Oseltamivir-Resistant Influenza Virus Infection

TO THE EDITOR: The incidence of influenza A (H1N1) viruses that carry the neuraminidase H274Y mutation has increased by 30% this year in the Netherlands.¹ Influenza A (H1N1) viruses that carry this mutation are resistant to oseltamivir but remain sensitive to zanamivir.² However, these mutant viruses are considered to have attenuated pathogenicity.^{3,4}

A 67-year-old man who had received a diagnosis of chronic lymphocytic leukemia 3 years earlier was admitted to the hospital because of dyspnea, dry cough, and fever. One week before admission, he had received a course of cyclophosphamide, vincristine, and prednisolone chemotherapy. At admission, his white-cell count was 137,000 per cubic millimeter, with 99% lymphocytes and no neutrophils. Because of acute respiratory failure, empirical antibacterial therapy was

Figure 1 (facing page). Leukocyte Counts, Viral Loads, and Treatment during the Hospital Course in a Patient Infected with Influenza A (H1N1) Virus with the H274Y Mutation.

Panel A shows the patient's granulocyte and monocyte counts. The gradual increase in the granulocyte count was consistent with bone marrow recovery. Panel B shows the viral load in the respiratory specimens. The dashed red line indicates the lower limit of detection. Various therapeutic and empirical antiviral therapies, shown in Panel C, and antibacterial and antifungal therapies, shown in Panel D, were given to the patient at different intervals (shaded bars). The red portions of the bars in Panel C indicate detection of resistance mutations for either oseltamivir (neuraminidase H274Y) or amantadine (M2-channel L26F), and the blue boxes indicate detection of the wild-type genotype. The L26F resistance mutation in the M2 protein was detected only on day 20, whereas the H274Y mutation was present before and after oseltamivir was administered.



initiated, and mechanical ventilation was required by the second hospital day (Fig. 1). Computed tomography (CT) revealed patchy infiltrates in both lungs, and influenza A (H1N1) virus was detected in respiratory secretions. During the entire hospital course, no other respiratory pathogens were detected in bronchoalveolar-lavage specimens. The only other pathogens identified in blood cultures were *Candida albicans* and *Enterococcus faecium*, for which fluconazole and vancomycin were given.

Oseltamivir was administered for the influenza virus infection, beginning on the sixth hospital day, but it was discontinued on day 13 because sequence analysis revealed the H274Y mutation, and no decrease in the viral load was observed. In retrospect, the H274Y mutation was present in the specimen obtained before oseltamivir therapy was initiated. The patient's hospital record and his family indicated that he had had no contact with patients who had received oseltamivir. On day 15, amantadine was added to the patient's treatment regimen. Four days later, the neutrophil count increased, indicating bone marrow recovery. Mechanical ventilation was discontinued on day 20, and zanamivir by inhalation was initiated. However, respiratory failure occurred on day 22, mechanical ventilation was reinstated, and therapy with zanamivir was discontinued. On day 26, the influenza virus was no longer detectable. Because sequence analyses showed an amantadine-resistance mutation in the viral M2 protein (L26F) and zanamivir therapy had

been limited to three doses, clearance of the virus was probably due to recovery of the immune system. A second CT scan, obtained on day 28, revealed progression of the pulmonary infiltrates. Because of the poor prognosis, mechanical ventilation was discontinued on day 34. The patient died 3 days later.

It has been suggested that the H274Y mutation, which confers resistance to oseltamivir, leaves the influenza A (H1N1) virus severely compromised.^{3,4} However, the case we describe suggests that this oseltamivir-resistant virus can be pathogenic, at least in an immunocompromised patient.

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