CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 27-2024: A 24-Year-Old Man with Pain and Dyspnea

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PRESENTATION OF CASE

Dr. Meridale V. Baggett: One hundred years ago, a 24-year-old man was admitted to this hospital because of fever, cough, and shortness of breath.[1]

The patient had been well until 3 days before this admission, when malaise, generalized weakness, headache, and back pain developed. During the next 2 days, his symptoms persisted, and he remained at home and slept in bed for most of the day. On the day before this admission, he began to have fever, dry cough, and chills that caused him to shake considerably and "hunched him up in a knot." He was unable to get out of bed. He took aspirin at a dose of 10 grains (648 mg) every 4 hours, which led to decreased severity of the headache and back pain. On the day of this admission, the patient awoke with difficulty breathing and chest pain just below the xiphoid process. The pain increased with deep breathing and coughing. He sought evaluation at this hospital.

On examination, the rectal temperature ranged from 39.5°C to 40.8°C, the heart rate from 92 to 145 beats per minute, and the respiratory rate from 28 to 58 breaths per minute. The patient appeared acutely ill and nervous. He was shivering despite being wrapped up in several blankets. His breathing was rapid, shallow, and labored. He had frequent paroxysms of racking cough, which caused excruciating pain under the lower sternum. The cough was productive of pinkish, thick, slightly purulent sputum.

On palpation, the apical impulse of the heart was felt in the fifth intercostal space, 8 cm to the left of the sternum along the midclavicular line. There was no enlargement of the heart on percussion. On auscultation, the heart sounds were rapid and regular, and there was a soft systolic murmur at the apex that did not radiate. The breath sounds were diminished at the right side of the back, from the lower third of the scapula downward, but had no definitive dullness or change in character. No evidence of rales or a friction rub was noted.

The throat was somewhat injected, and the tonsils had been surgically removed. A healed surgical scar was present on the left side of the abdomen, at the site of

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a previous hernia repair. The abdomen was not distended or tender. The skin was dry and hot to the touch. The white-cell count ranged from 3700 to 14,500 per microliter, with neutrophil predominance (79%). Blood cultures showed no growth.

Dr. Jo-Anne O. Shepard: A posteroanterior chest radiograph (Fig. 1) showed bilateral patchy consolidations, previously described as "mottled dullness," that were most confluent in the right upper lobe and the left lower lobe. This finding is suggestive of pneumonia. There was also fullness in the left hilar region, which is suggestive of lymphadenopathy. The presence of a left pleural effusion cannot be ruled out.

Dr. Kathy M. Tran: Adhesive plaster was applied to the patient's chest, and he was admitted to this hospital. On the second hospital day, the shortness of breath and chest pain on inspiration continued. The cough became productive of purulent sputum and blood. W.H. Smith performed a physical examination. The patient appeared very ill and held his abdominal muscles rigid while coughing. A systolic apical bruit was detected, along with dullness at the right lung base. Small hyperemic papules were seen on the left palm and the right index finger. Smith noted a "serious outlook" for the patient.

On the third hospital day, the sputum became much more purulent. On examination, increased dullness was noted on percussion of the left lower back. Increased tactile fremitus was also noted. Loud bronchial breath sounds and very few rales were heard at the left side of the back, from the lower third of the scapula downward. Slight dullness was present on percussion of the right lower back, and there were distant breath sounds and occasional medium rales.

On the fourth hospital day, the patient continued to be very ill. He gradually grew weaker, and that evening, he died.

A diagnosis was made.

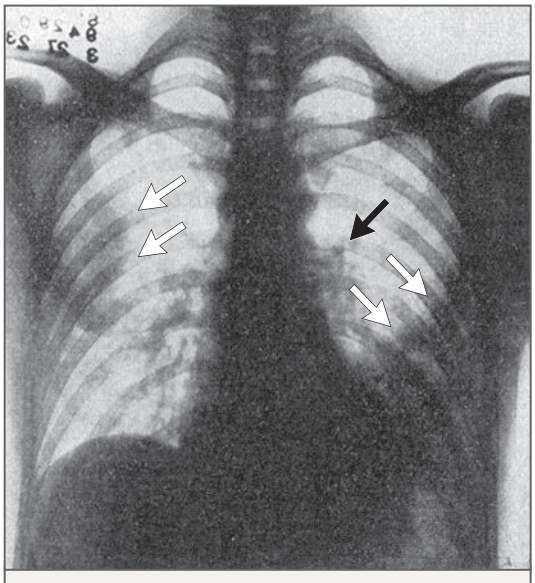

Figure 1. Chest Radiograph.

A posteroanterior chest radiograph obtained in 1923 shows bilateral patchy consolidations, previously described as "mottled dullness," that are most confluent in the right upper lobe and the left lower lobe (white arrows). Fullness in the left hilar region (black arrow) is also visible.

DIFFERENTIAL DIAGNOSIS

Dr. Rochelle P. Walensky: This 24-year-old man presented in March of 1923 with the acute onset of fever, chills, myalgias, shortness of breath, and pleuritic chest pain. His symptoms and signs are consistent with a respiratory viral infection such as influenza, possibly complicated by a bacterial superinfection. Given the proximity of his presentation to the influenza pandemic of 1918, influenza is the most likely diagnosis in this patient's case.

Although the signs, symptoms, and complications of influenza today are similar to those first noted in the 1918 pandemic, the intervening decades have brought important scientific discoveries, including the identification and isolation of influenza virus, rapid diagnostic tests, effective antiviral therapy, and the development of surveillance systems and vaccination programs. Looking back on the history of influenza offers the opportunity to reflect on what we have learned and how we can prepare for future pandemics.

1918 INFLUENZA PANDEMIC

During the 1918 influenza pandemic, the first case of influenza in the United States was diagnosed in a U.S. Army cook, stationed at Fort Riley in Kansas, on March 4, 1918.[2] During the next month, a physician in Haskell County, Kansas, named Loring Miner diagnosed 18 cases of severe influenza, including 3 that led to death. Miner reported his findings to the U.S. Public Health Service.

There was no public health response to Miner's report of these illnesses. Historians have observed that at the time, 4 years into World War I, the government was not transparent about the outbreak or its gravity. John Barry, who wrote the book *The Great Influenza*, commented in a 2020 interview on this lack of transparency: "The government lied. They lied about everything. We were at war, and they lied because they didn't want to upend the war effort. You had public health leaders telling people this was just the ordinary flu by another name. They simply didn't tell the people the truth about what was happening."[3] As a neutral party in World War I, Spain was the first country to publish newspaper articles related to influenza — which explains how this severe and novel viral infection, despite having first been documented by Miner in the United States, became known as the "Spanish flu."

From September through December 1918, an estimated 300,000 Americans died from influenza, a toll equating to 10 times the number of U.S. deaths from any cause during the same period in 1915. Influenza spread rapidly in the United States as soldiers, whether with symptomatic or asymptomatic infection, moved toward eastern transportation hubs after deployment. The disease was also disseminated through the frontline trenches in Europe, with more than 500 million people affected worldwide and 100 million deaths.[2]

In 1918, palliative therapies were limited and included aspirin and opium. The only therapeutic intervention with any suggestion of success was transfusion of blood from recovered patients to newly infected patients — what we now know as convalescent plasma therapy. Surveillance was limited to word of mouth. Workforce shortages in health care were exacerbated by the military deployment of more than one third of U.S. doctors and nurses. Although vaccines for cholera, typhoid, plague, and smallpox existed at the time, there was no vaccine for influenza because the pathogen had yet to be identified.

IDENTIFYING THE VIRUS

For years, the causative agent of the "Spanish flu" was thought to be Pfeiffer's bacillus (now known as *Haemophilus influenzae*), which had been found in the sputum of many — but not all — affected patients. Its absence was usually attributed to the nature of the bacteria as being fastidious and notoriously difficult to culture. However, because the bacteria had not been uniformly present, work to definitively identify the causative pathogen continued — and Pfeiffer's bacillus was later found to be the causative agent of a common bacterial superinfection.

The influenza virus was identified in 1933 by Wilson Smith and colleagues, who obtained throat washings from people with influenza, passed them through a bacteria-impermeable membrane, and then exposed ferrets to the bacteriologically sterile filtrate.[4] After 2 days of incubation, the exposed ferrets had symptoms similar to those occurring in humans with influenza. The article reporting these findings also mentioned that previous infection was "invariably" protective against reinfection with the same virus. A few years later, one of Smith's colleagues, Charles Stuart-Harris, contracted influenza after an infected ferret "sneezed violently at close range whilst being examined."[5] The virus isolated from Stuart-Harris was then used to infect a previously uninfected ferret. In the article describing these findings, the authors noted, "it is quite conceivable that a case of laboratory infection might be the starting point of an epidemic."[5]

DEVELOPING THE FIRST VACCINE

Once the influenza virus had been identified and isolated, work turned swiftly to producing a vaccine. In 1936, Frank Macfarlane Burnet was the first to show that the influenza virus grew easily in fertilized hen eggs, a method for vaccine production that is still in use today.[6] The first influenza vaccine was developed in 1940 by Thomas Francis and Jonas Salk. Given the devastating effects of influenza on the U.S. military in World War I, it is not surprising that the first people to be vaccinated in the United States, in the early 1940s, were Army soldiers. By 1942, there was evidence that such vaccination yielded protection, and the first influenza vaccine was approved for civilian use in 1946.[7] And vaccination was effective: the incidence of influenza among unvaccinated people was 10 to 25 times the incidence among those who were vaccinated.[8]

EARLY SURVEILLANCE EFFORTS

Surveillance for influenza disease activity and specific strains of the virus became critically important in guiding public health responses

and vaccination programs. Because influenza is a worldwide disease, both domestic and global surveillance systems are necessary.

The U.S. Communicable Disease Center (CDC) — the agency now known, under the same acronym, as the Centers for Disease Control and Prevention, for which I served as director from 2021 to 2023 — was founded in 1946 with a focus on studying outbreaks of diseases such as malaria, typhus, and smallpox. Five years later, the CDC created the Epidemic Intelligence Service specifically to provide training for the investigation of disease outbreaks. In 1954, the CDC established its first influenza surveillance system and began making periodic reports about disease activity.[9]

The World Health Organization (WHO) formed the Global Influenza Surveillance and Response System in 1952.[6] Collaboration between this system and the Global Initiative on Sharing All Influenza Data established surveillance for influenza around the world. The WHO designated the CDC as a collaborating center for surveillance, epidemiology, and control of influenza in 1956.[10]

1957 INFLUENZA PANDEMIC

The 1957 influenza pandemic originated in southern China in early 1957, and within 2 months, the virus had spread throughout China.[11] However, China was not yet part of the WHO and did not inform the rest of the world.[12] The new subtype of the virus (influenza A virus subtype H2N2) resulted from a mutation in an avian influenza strain in wild ducks that recombined with a human influenza strain.[13]

The warning of a developing pandemic eventually came from Maurice Hilleman, a U.S. microbiologist at the Walter Reed Army Institute of Research, who read an article in the *New York Times* in April 1957 about thousands of cases of influenza in Hong Kong.[14,15] After acquiring a sample of the virus from U.S. Navy doctors working in Japan, Hilleman identified the strain and began cautioning about a potential pandemic. In June 1957, this influenza virus reached the United States through infected military personnel returning from Asia.[16] The first wave of "Asian flu" in the United States peaked in October 1957, mostly affecting school-age children. The second peak more heavily affected older adults and was associated with higher mortality.

Recognizing early that this strain had pandemic potential, Hilleman shared samples with pharmaceutical companies to begin creating strain-specific vaccines, which became available in September 1957.[14] The U.S. Surgeon General recommended universal vaccination and was showcased in public-service announcements and commercials encouraging the public to get vaccinated.[17] However, the government response to build a vaccine and corresponding vaccination program was limited. Ultimately, 30 million people were vaccinated — only approximately 18% of the U.S. population at that time.[14]

The 1957 influenza pandemic caused 1 to 4 million deaths worldwide; in the United States, there were 20 million documented infections and 116,000 deaths.[14] It is estimated that the availability of an effective vaccine before the spike in February 1958 saved more than 1 million U.S. lives.[14] The decision by the Eisenhower administration to leave a vaccination program up to private solutions is now considered by many as "manifestly inadequate."[18]

1968 INFLUENZA PANDEMIC

The first case in the 1968 influenza pandemic also occurred in China, in July 1968.[19] Within 2 weeks after the first report, 500,000 cases had been documented in Hong Kong.[20] The new virus subtype (influenza A virus subtype H3N2) was the result of a genetic mutation that facilitated human-to-human transmission, but there was enough similarity to other influenza strains (N2) that previous exposure offered partial protection. Although the "Hong Kong flu" was highly transmissible, it resulted in a milder illness than that seen in the 1918 and 1957 pandemics. At an October 1968 meeting of the CDC Advisory Committee on Immunization Practices, the available bivalent and polyvalent influenza vaccines were recommended for use until a strain-specific vaccine was released. A monovalent vaccine ultimately became available, but only after the pandemic peak that year.[20] Approximately 1 million deaths occurred worldwide, including 100,000 in the United States; mostly older people died.[21]

2009 INFLUENZA PANDEMIC

In the 2009 influenza pandemic, the first case was identified in Mexico in March 2009, with the first death occurring there in April.[22] The

new virus subtype (influenza A virus subtype H1N1) — a triple reassortment from bird, swine, and human viruses — was known early on as the "swine flu."[23] Worldwide, the 2009 influenza pandemic affected between 700 million and 1.4 billion people, causing 284,000 estimated excess deaths.[24] The United States had 60 million cases, 274,000 hospitalizations, and 12,500 estimated deaths from influenza.[25] That year, approximately 41% of the U.S. population received the seasonal influenza vaccine, but the seasonal vaccine did not protect against H1N1.[26] The monovalent H1N1 vaccine became available in the United States in October 2009, around the same time that the number of U.S. cases peaked.[27] However, only 27% of the U.S. population received the monovalent vaccine. Of note, approximately 40% of school-age children were vaccinated, with the higher coverage attributed to school vaccination programs.[26] Although such programs were successful at reaching children, no mechanisms were in place to deliver widespread vaccinations to adults.

CURRENT STATE OF INFLUENZA SURVEILLANCE

Today, the CDC has developed an extensive network for domestic influenza surveillance. The four pillars of influenza surveillance are laboratory testing, as well as monitoring of outpatient cases, hospitalizations, and deaths (Fig. 2).

Global Surveillance

The Global Influenza Surveillance and Response System includes 144 national influenza centers, in more than 114 countries, that conduct year-round influenza surveillance; the CDC is one of these centers. Many centers send influenza virus isolates to the WHO for antigenic and genetic characterization, just as laboratories in the United States send isolates to the CDC. The 40-year collaboration between the CDC and China has been a critical part of global health security and diplomacy.[28]

Surveillance during and after Covid-19

During the coronavirus disease 2019 (Covid-19) pandemic, the CDC developed the Respiratory Virus Hospitalization Surveillance Network (RESP-NET) dashboard to collect data on Covid-19, influenza, and respiratory syncytial virus infection.[29] The dashboard is derived from the National Syndromic Surveillance Program platform,[30] which receives data from nearly 80% of emergency departments and from 6500 health care facilities, covering all 50 states. Such data collection is resource intensive, and therefore, pathogens for which there are medical countermeasures, such as vaccines or treatments, are prioritized for surveillance. Now that the Covid-19 public health emergency is over, the CDC lacks the authority to compel the reporting of hospitalization and bed-capacity data; reporting of such data to the National Healthcare Safety Network is voluntary.[31]

CURRENT STATE OF INFLUENZA VACCINATION

Influenza vaccines are created by the WHO twice per year.[32] In the past 20 years, average reported effectiveness has ranged from 40 to 55%.[33] Vaccination coverage is arguably even more important than effectiveness. In the United States, annual vaccination coverage for influenza is approximately 50% but varies according to age; nearly 75% of people older than 65 years of age are vaccinated each year. Vaccination coverage also varies by state. One of the most important predictors of vaccination in adults is insurance status.[34] Almost 50% of people with public or private health insurance are vaccinated against influenza each year, as compared with only 15% of those without insurance. The Vaccines for Children program has had success in closing this gap in the pediatric population.[35] A similar Vaccines for Adults program has been proposed in the President's budget, in both 2023 and 2024, to assist with vaccination coverage for more than 25 million uninsured adults, but this program has yet to be funded.[36]

IMPLICATIONS FOR THE NEXT INFLUENZA PANDEMIC

Remarkable scientific progress has been made since the 1918 influenza pandemic. The causative virus has been identified and investigated. Vaccines have been produced to provide protection, and scientific, technological, and logistic innovations have improved the speed of vaccine development. Strengthened surveillance systems, although still limited, provide earlier warnings of a possible next pandemic. What has been stagnant since 1918 is political will, engagement in social contracts, and the capacity for high-fidelity communication. Surveillance data are reported voluntarily, without bipartisan

commitment to standardize reporting and extend data authorities to support these systems. There is a lack of commitment in communities to take actions that protect others. And there are communication and media challenges that foster fearmongering, controversy, and propagation of misinformation and disinformation. As John Barry wrote in *The Great Influenza*, "Society cannot function if it's every man for himself. By definition, civilization cannot survive that."[37]

My diagnosis for the patient presented in this case is influenza with bacterial superinfection, causing pneumonia and possible empyema. My diagnosis for our country is this: despite the remarkable scientific advances in the identification and understanding of influenza, little progress has been made in the social challenges that continue to limit the impact of our scholarly progress. Failing to learn the lessons of previous pandemics has left us with consistently fractured health care, public health, and vaccine distribution systems. Huge strides were made in recognizing and addressing these fractures during the Covid-19 pandemic, and we must leverage that learning for the future health of our country and the world.

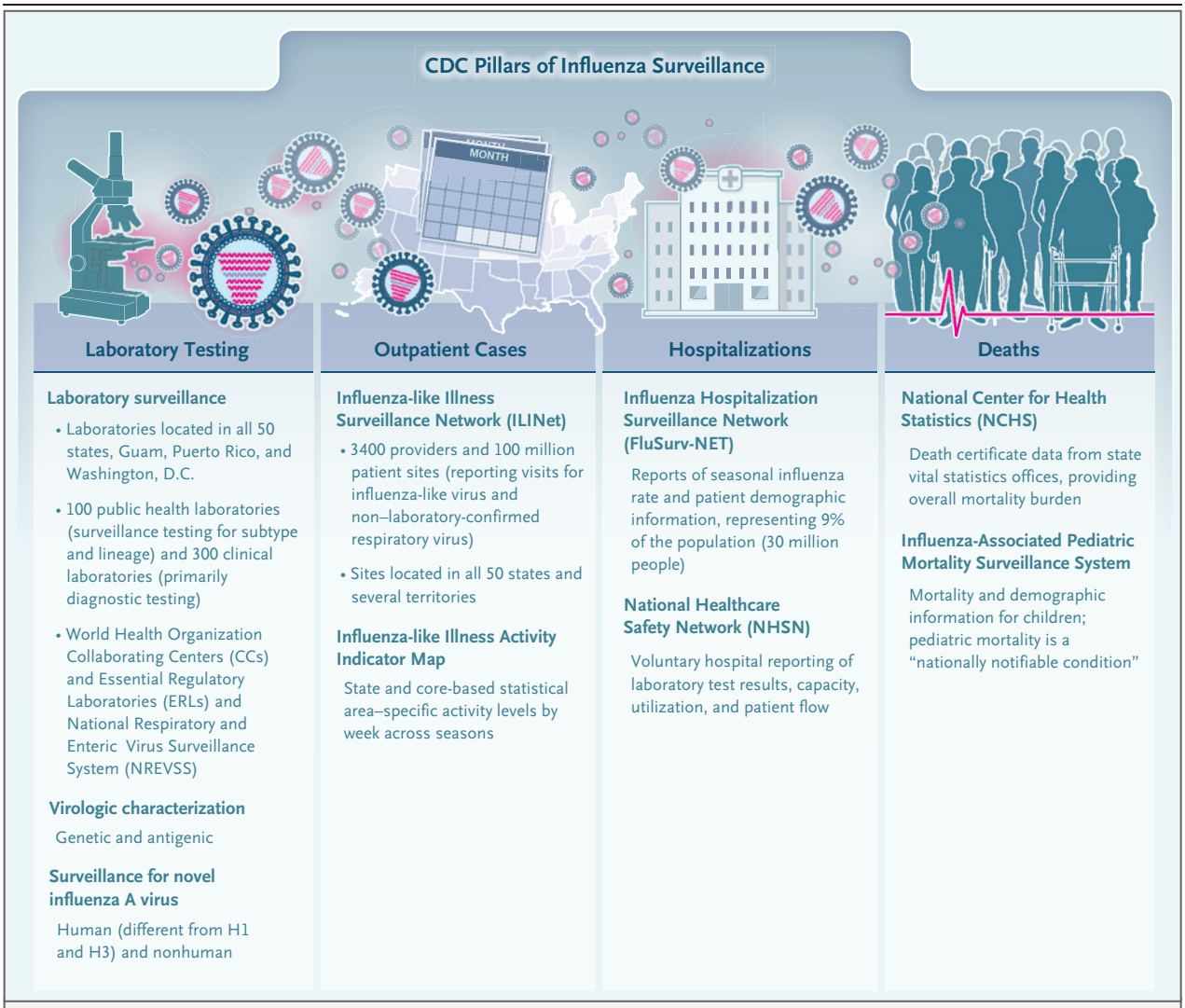

Figure 2. CDC Pillars of Influenza Surveillance.
CDC denotes Centers for Disease Control and Prevention.

CLINICAL IMPRESSION

Dr. David M. Dudzinski: It is a historic privilege to stand on the shoulders of a luminary of our hospital such as Richard Cabot and to present his impression from 1923. Cabot's clinical impression was bronchopneumonia with purulent bronchitis, as well as possible empyema on the left side of the chest. Furthermore, he thought that the bronchopneumonia was most likely due to influenza, noting that the sequence typically starts with diminished breathing and without rales.

CLINICAL DIAGNOSIS

Bronchopneumonia with purulent bronchitis and possible empyema, most likely due to influenza.

DR. ROCHELLE P. WALENSKY'S DIAGNOSIS

Influenza pneumonia with bacterial superinfection and possible empyema.

PATHOLOGICAL DISCUSSION

Dr. Dennis C. Sgroi: One hundred years ago, the autopsy for this patient was a gross examination only. The chest cavity contained 150 ml of thin purulent exudate in the right and left pleural cavities. The parietal and visceral pleurae of the lungs were coated with a fibrinopurulent exudate. The trachea and bronchi were described as blackish red, with thick, velvety mucosae, on top of which was a "fibrinous dirty grayish … so-called diphtheritic membrane." The lungs were described as having an area of purple-red tissue and focal patchy areas of various sizes that were frankly purulent, semifluid, and in some areas, gelatinous. The purple-red area probably indicates vascular congestion. The patchy purulent areas are consistent with an infectious process that is centered around the bronchioles. The gelatinous areas show frank necrosis. The final diagnosis was bronchopneumonia, most likely due to influenza.

FINAL DIAGNOSIS

Bronchopneumonia, most likely due to influenza.

This case was presented at the Case Records of the Massachusetts General Hospital Centennial Celebration Conference.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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