CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 36-2021: A 22-Year-Old Man with Pain and Erythema of the Left Hand

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PRESENTATION OF CASE

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Dr. Marisa L. Winkler: A 22-year-old man was admitted to this hospital because of pain and rapidly spreading erythema of the left hand.

The patient had been well until the day of admission, when he awoke with pain and swelling of the left hand that involved the distal interphalangeal joint of the second finger and the proximal interphalangeal joint of the fourth finger. Over a period of several hours, the pain progressed and bullae began to form. He began to have pain with movement of the second and fourth fingers, and the bullae turned dark purple; these changes prompted the patient to present to the emergency department of this hospital.

In the emergency department, the patient reported pain of the left hand that worsened with movement of the second and fourth fingers. There was no lethargy, fatigue, headache, dyspnea, cough, or pain elsewhere. The patient had no notable medical history and had been well before the day of presentation.

On examination, the patient appeared well. The temperature was 38.6°C, the blood pressure 126/63 mm Hg, the heart rate 101 beats per minute, the respiratory rate 18 breaths per minute, and the oxygen saturation 100% while he was breathing ambient air. There were two violaceous, tender bullae on the dorsal aspect of the left hand — one on the distal interphalangeal joint of the second finger and one on the proximal interphalangeal joint of the fourth finger, each measuring 7 mm by 3 mm (Fig. 1). In addition, there was nontender, streaking erythema spreading across the dorsal aspect of the left hand, the volar aspect of the left forearm, and the medial aspect of the left upper arm into the axilla. There was no axillary lymphadenopathy.

On further examination of the left arm, the soft-tissue compartments were soft and compressible, without evidence of crepitus. Finger flexion and extension were intact but limited by pain and swelling in the second and fourth fingers. Results of motor and sensory examinations were normal. The hand was warm. The radial pulse was palpable, and the capillary refill was brisk. The remainder of the overall physical examination was unremarkable. A blood specimen was obtained for cul-

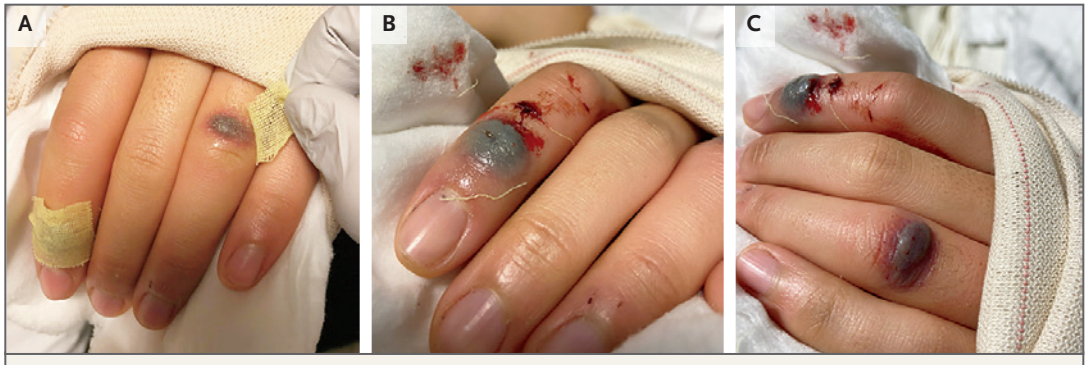

Figure 1. Clinical Photographs.

Photographs of the patient's left hand show violaceous bullae on the proximal interphalangeal joint of the fourth digit (Panel A) and the distal interphalangeal joint of the second digit (Panel B), each measuring 7 mm by 3 mm (Panel C).

ture, a complete blood count, and a metabolic panel. Initial laboratory test results are shown in Table 1.

Dr. Arvin Kheterpal: Computed tomography (CT) of the left hand and arm, performed after the administration of intravenous contrast material, revealed focal soft-tissue swelling overlying the distal interphalangeal joint of the second finger and the proximal interphalangeal joint of the fourth finger on the dorsal aspect of the left hand (Fig. 2). There was no evidence of osseous erosion, periosteal reaction, fracture, joint effusion, fluid collection, or subcutaneous air. The left forearm had a normal appearance on imaging.

Dr. Winkler: While the patient was in the emergency department, additional history was obtained. The patient was not taking any medications and had no known allergies to medications. He had recently graduated from college and was seeking employment. He lived with his parents in a suburban area of New England. There was no history of recent travel or sick contacts. He rarely drank alcohol and did not smoke cigarettes or use illicit drugs. There was no notable family history.

The patient had recently begun practicing taxidermy as a new hobby. Four days before presentation, he received a frozen deer hide from a friend, which he began to prepare for tanning. When he first received the deer hide, he scraped the skin and fur to remove numerous ticks. On the day before presentation, he kneaded the hide with his hands and massaged into it a mixture of deer brain and tap water that had been prepared the day before and left to stand overnight. While the patient was working with the animal hide, he did not wear protective clothing or gloves. His mother had assisted in part of the process but remained well.

Intravenous fluid, ceftriaxone, and vancomycin were administered, and the patient was admitted to the hospital. Consultants from the orthopedic hand service, dermatology, and infectious diseases evaluated the patient. A diagnostic test was performed.

DIFFERENTIAL DIAGNOSIS

Dr. Lisa G. Winston: In developing the differential diagnosis in this case, it is helpful to consider several key points about this patient's medical and social history, his relevant exposures, and his clinical presentation. The patient was a healthy young adult who had not been taking any medications, so coexisting medical conditions would not be expected to play a role. He lived in a suburban area of New England and had no risk factors related to travel, sick contacts, employment, or health-related behaviors. The compelling aspect of his exposure history is that 4 days before admission, he had scraped a frozen deer hide to remove ticks without the use of gloves. He had also kneaded the hide and massaged into it a mixture of deer brain and tap water on the day before admission.

The patient had been well until the day of presentation, when a locally progressive process involving his left hand and arm developed. The process was characterized by pain, swelling, and bullae of the hand, along with streaking ery-

Table 1. Laboratory Data.*

Variable	Reference Range, Adults†	On Presentation
Hemoglobin (g/dl)	12.0–16.0	14.8
Hematocrit (%)	41.0–53.0	45.5
White-cell count (per μl)	4500–11,000	10,520
Differential count (per μl)		
Neutrophils	1800–7700	8650
Immature granulocytes	0–100	30
Lymphocytes	1000–4800	990
Eosinophils	0–900	30
Basophils	0–300	30
Monocytes	200–1200	790
Platelet count (per μl)	150,000–450,000	254,000
Sodium (mmol/liter)	135–145	139
Potassium (mmol/liter)	3.4–5.0	3.7
Chloride (mmol/liter)	98–108	102
Carbon dioxide (mmol/liter)	23–32	23
Creatinine (mg/dl)	0.60–1.50	0.97
Urea nitrogen (mg/dl)	8–25	19
Aspartate aminotransferase (U/liter)	9–32	8
Alanine aminotransferase (U/liter)	7–33	12
Alkaline phosphatase (U/liter)	30–100	40
Total bilirubin (mg/dl)	0.0–1.0	0.5
International normalized ratio	0.9–1.1	1.2
Prothrombin time (sec)	11.5–14.5	15.0
Partial-thromboplastin time (sec)	22.0–36.0	31.3
C-reactive protein (mg/liter)	<8.0	4.1
Erythrocyte sedimentation rate (mm/hr)	0–13	2
Lactic acid (mmol/liter)	0.5–2.0	1.2

* To convert the values for creatinine to micromoles per liter, multiply by 88.4. To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the values for bilirubin to micromoles per liter, multiply by 17.1. To convert the values for lactic acid to milligrams per deciliter, divide by 0.1110.

† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are for adults who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

thema that had spread up the arm. The physical examination was otherwise notable only for fever and mild tachycardia, and the patient appeared well. The laboratory evaluation was largely normal, except for a slightly elevated neutrophil count. A CT scan showed soft-tissue swelling involving the dorsal aspect of the left hand but no evidence of a deeper process. On the basis of the information provided in the case description and the clinical photographs, I would describe the patient's illness as an acute cellulitis with hemorrhagic bullae and streaking erythema that developed after exposure to a deer hide and possible minor trauma associated with vigorous scraping. Given the patient's appearance, the time course of the illness, and the absence of signs and symptoms suggestive of a preexisting or concurrent systemic condition, the cause is likely to be a local infection. Although the exposure to deer brain is intriguing, there are no organisms specifically associated with this tissue that cause cellulitis, and the incubation period would have been quite short.

SKIN AND SOFT-TISSUE INFECTIONS

Before we discuss infectious pathogens associated with deer and deer hides, we should consider typical human pathogens that cause skin and soft-tissue infections. Beta-hemolytic streptococcus, especially group A streptococcus (*Streptococcus pyogenes*), is the most common cause of cellulitis, and blistering and lymphangitis may occur. *Staphylococcus aureus* can cause cellulitis, but a purulent infection of the skin and soft tissue usually develops.

It is appropriate to provide antibiotic coverage targeting typical causes of cellulitis while an evaluation for more unusual organisms is in progress. Although there is a long list of zoonoses that are potentially associated with exposure to deer, the most relevant infections to consider are those that manifest with skin lesions, those that have a relatively short incubation period, and those that may be transmitted during contact with a frozen deer hide.

PLAGUE

Could this patient have an infection with *Yersinia pestis*, known as the plague? This infection has multiple possible routes of transmission, including handling of infected animal tissue. Transmission by this route can result in bubonic or septicemic plague. Although *Y. pestis* can be found in deer, it is more commonly associated with other animals. In addition, since 1970, all U.S. cases of the plague have occurred in western states, except for one laboratory-acquired case.[1] Bubonic plague has an incubation period of 2 to 6 days, which is compatible with the time course in this case, and it can result in papules, ulcers,

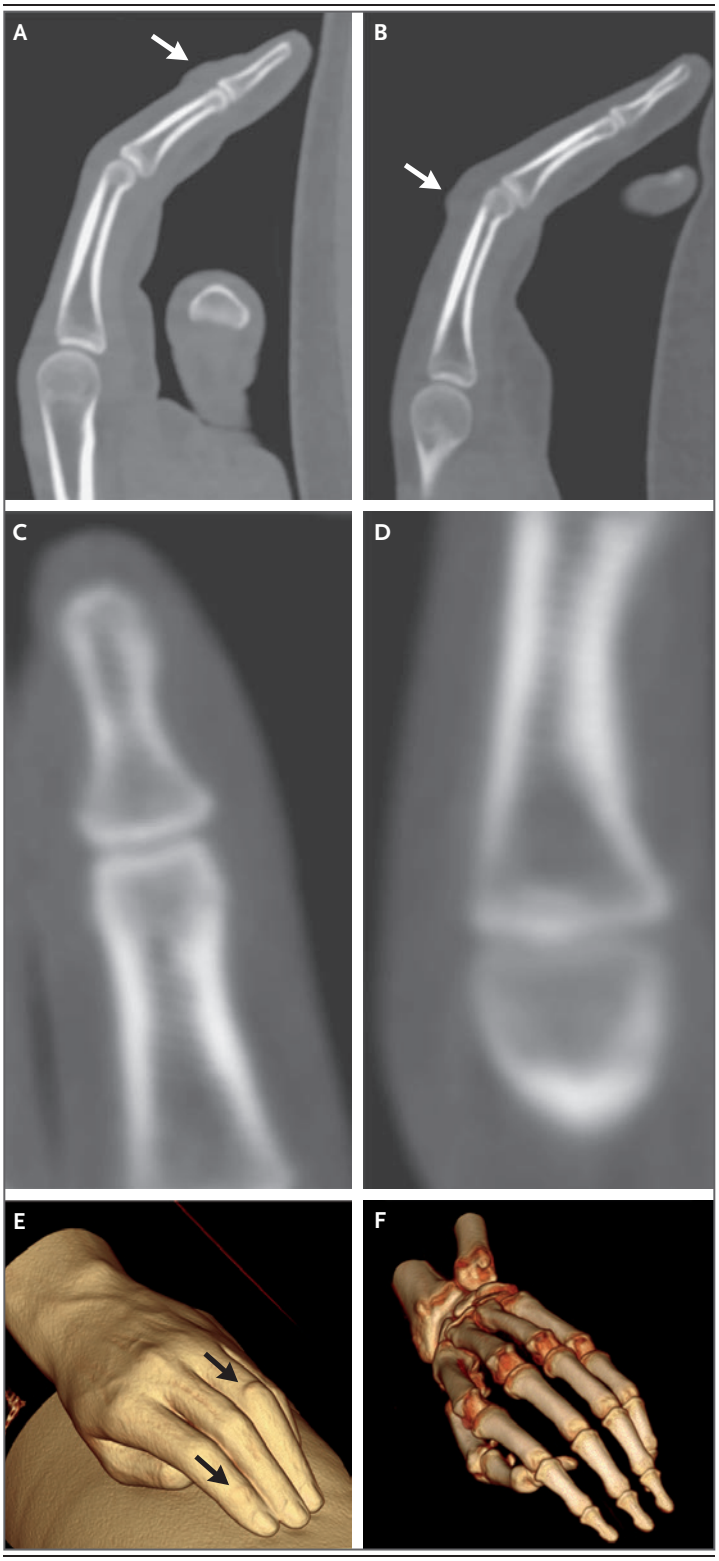

Figure 2. Imaging Studies.
CT of the left hand was performed after the administration of intravenous contrast material. Sagittal images centered on the second digit and the fourth digit (Panels A and B, respectively) show focal soft-tissue swelling (arrows) overlying the second distal interphalangeal joint and the fourth proximal interphalangeal joint on the dorsal aspect of the hand. Coronal images centered on the second distal and fourth proximal interphalangeal joints (Panels C and D, respectively) show no evidence of osseous erosion, periosteal reaction, fracture, joint effusion, or fluid collection. A three-dimensional reconstruction of the left hand (Panel E) also shows focal soft-tissue swelling (arrows) overlying the second distal and fourth proximal interphalangeal joints. A three-dimensional image of the bones (Panel F) shows no abnormal features.

and eschars at flea-bite inoculation sites. However, we have no reason to suspect that this patient had exposure to fleas, and the skin findings are not compatible with buboes or with the lesions that develop at bite inoculation sites. Therefore, the plague is unlikely to account for this patient's presentation.

BRUCELLOSIS

Brucellosis can be caused by multiple species of brucella. *Brucella abortus* is well described in deer, posing a risk of infection in hunters.[2] Brucellosis can develop after skin contact with infected animal tissue. The usual incubation period is 2 to 4 weeks, but it is possible for the disease to manifest in 5 days.[3] Although skin findings can develop, they are relatively uncommon and typically diffuse. Rashes associated with brucellosis may be papulonodular or maculopapular or may resemble erythema nodosum. In this case, neither the time course nor the skin findings fit well with the diagnosis of brucellosis.

TULAREMIA

Tularemia is caused by the bacterium *Francisella tularensis*. Like the plague, tularemia has multiple possible routes of transmission, including contact with infected animal tissue. *F. tularensis* has been described in deer, but infection in humans is usually associated with contact with other animals. Indeed, tularemia is sometimes referred to as rabbit fever.[4] In patients who have the ulceroglandular form of tularemia, a skin ulcer is present, along with a swollen lymph node; these manifestations do not match the skin findings in this case. Also, patients with tularemia usually have a systemic illness, whereas this patient appeared well, with few systemic signs and symptoms other than fever.

ORF

Orf, also known as contagious ecthyma, should be considered in this patient. The orf virus is in the parapoxvirus genus. Humans are infected after contact with animals directly or with contaminated equipment. Although most cases result from contact with sheep or goats, the virus has been found in deer, and transmission to a human from a deer carcass has been described.[5] Humans have less commonly been infected with other parapoxviruses after animal exposure, and transmission of novel parapoxviruses to deer hunters has been described.[6] After an incubation period of 3 to 7 days, orf initially manifests as a small papule and then progresses through stages involving the development of a hemorrhagic bulla or pustule. Low-grade fever and lymphangitis may be present. However, in this case, given the amount of time that had passed since the first exposure to the deer hide, one would expect to see only a papule at the time of presentation if the patient's illness were due to orf.

ANTHRAX

A diagnosis of cutaneous anthrax, which is caused by *Bacillus anthracis*, fits with several features of this case, particularly the deer-hide exposure.[7] Of the several types of anthrax, cutaneous anthrax is the most common and least lethal. Infection usually results from handling of infected animal products. The spores can be found in soil, and hoofed animals, including deer, are most likely to host the organism. Anthrax is rare in the United States, and a vaccine is available for use in livestock. Cutaneous anthrax manifests 1 to 7 days after exposure, initially with a painless papule, which progresses to a vesicle and subsequently erodes to a painless ulcer with an eschar.[8] Extensive edema is often present because of the production of edema toxin, and lymphangitis and systemic symptoms may occur. A hallmark of cutaneous anthrax is that it is painless; this patient had marked and progressive pain. Thus, we should continue to look for a diagnosis that is consistent with all the features of this case.

ERYSIPELOTHRIX RHUSIOPATHIAE INFECTION

Erysipelothrix rhusiopathiae can infect humans after animal exposure. When the infection occurs in humans, the most common presentation is a localized cutaneous disease known as erysipeloid. When the infection occurs in pigs, it can cause a disease known as swine erysipelas, which is characterized by fever, arthritis, and skin abnormalities. The terminology is confusing because human erysipelas is a superficial cellulitis that is usually caused by group A streptococcus.[9]

In humans, most erysipeloid lesions occur on the fingers after occupational exposure to animals. The infection is most common in those who handle fresh or frozen fish or crabs; slaughterhouse workers, butchers, and farmers are also at risk.[9] *E. rhusiopathiae* has been isolated from multiple animals and is widespread in the environment. In addition to infecting fish, shellfish, and swine, it can be found in cattle, horses, sheep, turkeys, chickens, cats, dogs, and other animals.[10] In the literature, there is at least one report of erysipeloid occurring after the patient had slaughtered a deer.[11]

Erysipeloid manifests as cellulitis 2 to 7 days after exposure to *E. rhusiopathiae*. Violaceous and well-defined lesions are typical, and vesicles may develop. Early pain and localized swelling without pitting edema are thought to be characteristic clinical manifestations. Lymphangitis and regional lymphadenopathy may occur. Systemic symptoms are relatively uncommon with localized erysipeloid, but fever may occur.[12] In some cases, *E. rhusiopathiae* may cause an infectious syndrome other than erysipeloid, such as a diffuse cutaneous form, bacteremia with possible seeding of distant sites, or endocarditis. On the basis of this patient's exposure history and his clinical presentation of fever, marked pain, and characteristic bullous lesions, I suspect that the most likely diagnosis in this case is erysipeloid due to infection with *E. rhusiopathiae*.

E. rhusiopathiae is a nonsporulating, gram-positive, rod-shaped bacterium. In the clinical microbiology laboratory, it can be visualized on Gram's staining and recovered in bacterial culture, although a deep-tissue specimen may be needed. Blood cultures are rarely positive in patients with erysipeloid.

CLINICAL IMPRESSION

Dr. Winkler: I was on the consulting team for this patient. We were initially concerned about the possibility of a cutaneous *B. anthracis* infection,

given the deer-hide exposure. We thought that this diagnosis was unlikely because the patient did not have an eschar and had a substantial degree of pain. Nevertheless, out of an abundance of caution, we notified the microbiology laboratory that *B. anthracis* infection was a possible diagnosis to ensure that appropriate biosafety practices would be used in the handling of specimens.

Because the patient had bullae and lymphangitic streaking, our working diagnosis was infection with a typical bacterial pathogen such as *S. pyogenes* or *S. aureus*. However, the exposure history was compelling, and we were also concerned about zoonotic bacterial pathogens including *E. rhusiopathiae* and *F. tularensis*. Given the exposure to tap water in the deer-brain mixture, we also considered inoculation with *Pseudomonas aeruginosa* or *Vibrio vulnificus*. At the time of the initial evaluation, we did not think that we had enough information to target a specific pathogen, and therefore, we initiated broad-spectrum antibiotic therapy with cefepime, doxycycline, and ciprofloxacin while results of the additional workup were pending.

CLINICAL DIAGNOSIS

Cutaneous bacterial infection.

DR. LISA G. WINSTON'S DIAGNOSIS

Erysipeloid due to *Erysipelothrix rhusiopathiae* infection.

PATHOLOGICAL DISCUSSION

Dr. Julian A. Villalba: Two superficial swabs and a punch-biopsy specimen from the dorsal surface of the left index and ring fingers were obtained 1 day after the onset of symptoms and were submitted for routine microbiologic cultures; the biopsy specimen was also submitted for pathological evaluation. Because the initial clinical differential diagnosis included cutaneous anthrax, all specimens were handled inside a biosafety cabinet in accordance with recommendations from the Centers for Disease Control and Prevention.[13,14] A swab was sent to the Massachusetts State Public Health Laboratory. Real-time polymerase-chain-reaction analysis performed for the detection of *B. anthracis* and *B. cereus* biovar *anthracis* nucleic acids was negative. Direct Gram's staining revealed rare neutrophils but no organisms. Cultures were performed in a liquid medium (thioglycolate broth), as well as on routine solid-based mediums, including sheep-blood agar, chocolate agar, and MacConkey agar.

Microscopic examination of the punch-biopsy specimen revealed bullae formation and lymphatic dilatation (Fig. 3A) associated with epidermal necrosis with intraepidermal microvesicles and underlying subepidermal edema (Fig. 3B, 3C, and 3D). There was a mixed cellular infiltrate that had periadnexal and perivascular predominance but extended from the reticular dermis to the subcutaneous tissue (Fig. 3E and 3F). The infiltrate had focal areas of necrosis and was composed of abundant histiocytes and lymphocytes, with occasional neutrophils (Fig. 3G and 3H). These histologic features were suggestive of, but not specific for, an infectious dermatosis. Special histochemical stains for microorganisms, including a Brown–Hopps stain, Steiner silver stain, periodic acid–Schiff stain, and Gomori methenamine silver stain, were negative.

Bacterial growth was detected in the thioglycolate-broth culture on day 2. Bacterial colonies grew throughout the medium but were seen primarily in the top and middle parts of the tube (Fig. 4A); this feature indicates the presence of a facultative anaerobe. Gram's staining of the broth revealed long, gram-positive, nonsporulating, nonbranching, rod-shaped bacteria with filamentous features and rounded ends (Fig. 4B). Biochemical testing revealed that the isolate was catalase-negative and produced hydrogen sulfide when incubated on triple sugar iron agar (Fig. 4C). The isolate was subcultured on sheep-blood agar, and within 1 day of incubation, colonies with pinpoint, transparent, and alpha-hemolytic microbiologic characteristics were observed (Fig. 4D). Disk diffusion, a culture-based assay to semiquantitatively determine the antimicrobial susceptibility of bacteria, was performed. The isolate was inoculated into solid agar in the presence of a vancomycin-containing paper disk (30-μg disk), and after overnight incubation, the size of the zone of inhibition revealed resistance to vancomycin (Fig. 4E). A biochemical test panel that combined carbohydrate fermentation tests and direct enzyme de-

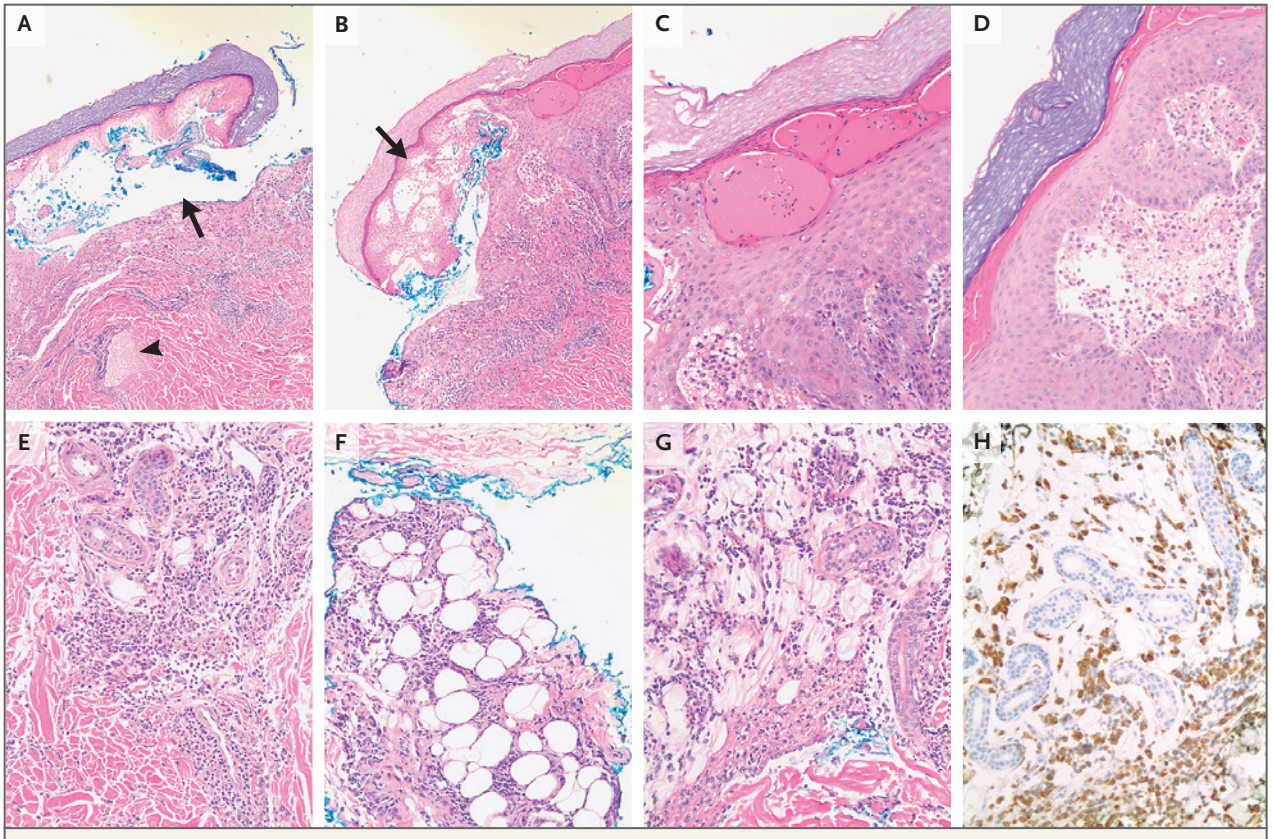

Figure 3. Microscopic Analysis of Skin Specimens.

Hematoxylin and eosin staining of a punch-biopsy specimen of skin from the dorsal surface of the left fourth digit shows bullae formation (Panel A, arrow), lymphatic dilatation (Panel A, arrowhead), and epidermal necrosis (Panel B, arrow). At higher magnification, the epidermis shows intraepidermal microvesicles (Panel C) and underlying subepidermal edema with early bullae formation (Panel D). There is a dermal periadnexal and perivascular cellular infiltrate (Panel E) that extends into the adipose tissue of the hypodermis (Panel F). The infiltrate has focal areas of necrosis with associated dermal edema, and it is predominantly lymphohistiocytic, with occasional neutrophils (Panel G). Immunohistochemical staining shows abundant cells expressing CD68, a marker of histiocytic origin (Panel H, in brown).

tection assays (Fig. 4F) and MALDI-TOF (matrix-assisted laser desorption ionization–time of flight) mass spectrometry confirmed that the pathogen was *E. rhusiopathiae*. Cultures of the blood specimen that had been obtained on the first day of admission were negative.

Zoonotic infection with *E. rhusiopathiae* is commonly restricted to mild cutaneous forms.[15] Bacterial identification can present a challenge to the clinical microbiology laboratory, because at least two different morphotypes in colonies and on Gram's staining have been reported.[16] However, accurate identification of the pathogen is critical to ensure effective antimicrobial therapy, because erysipelothrix species are intrinsically resistant to vancomycin and many isolates can also be resistant to aminoglycosides and sulfonamides.[17]

PATHOLOGICAL DIAGNOSIS

Cutaneous *Erysipelothrix rhusiopathiae* infection.

FOLLOW-UP

Dr. Winkler: After the initiation of antimicrobial therapy, the patient's condition improved during the hospitalization, and he was discharged on hospital day 6. He had increased mobility of the second and fourth fingers, recession of the lymphangitic streaking, and no further fevers. After the culture results were received, the antimicrobial regimen was changed. The preferred agent for the treatment of *E. rhusiopathiae* infection is penicillin or a cephalosporin; the organism is intrinsically resistant to vancomycin.[18] We chose to initiate treatment with amoxicillin–clavulanic

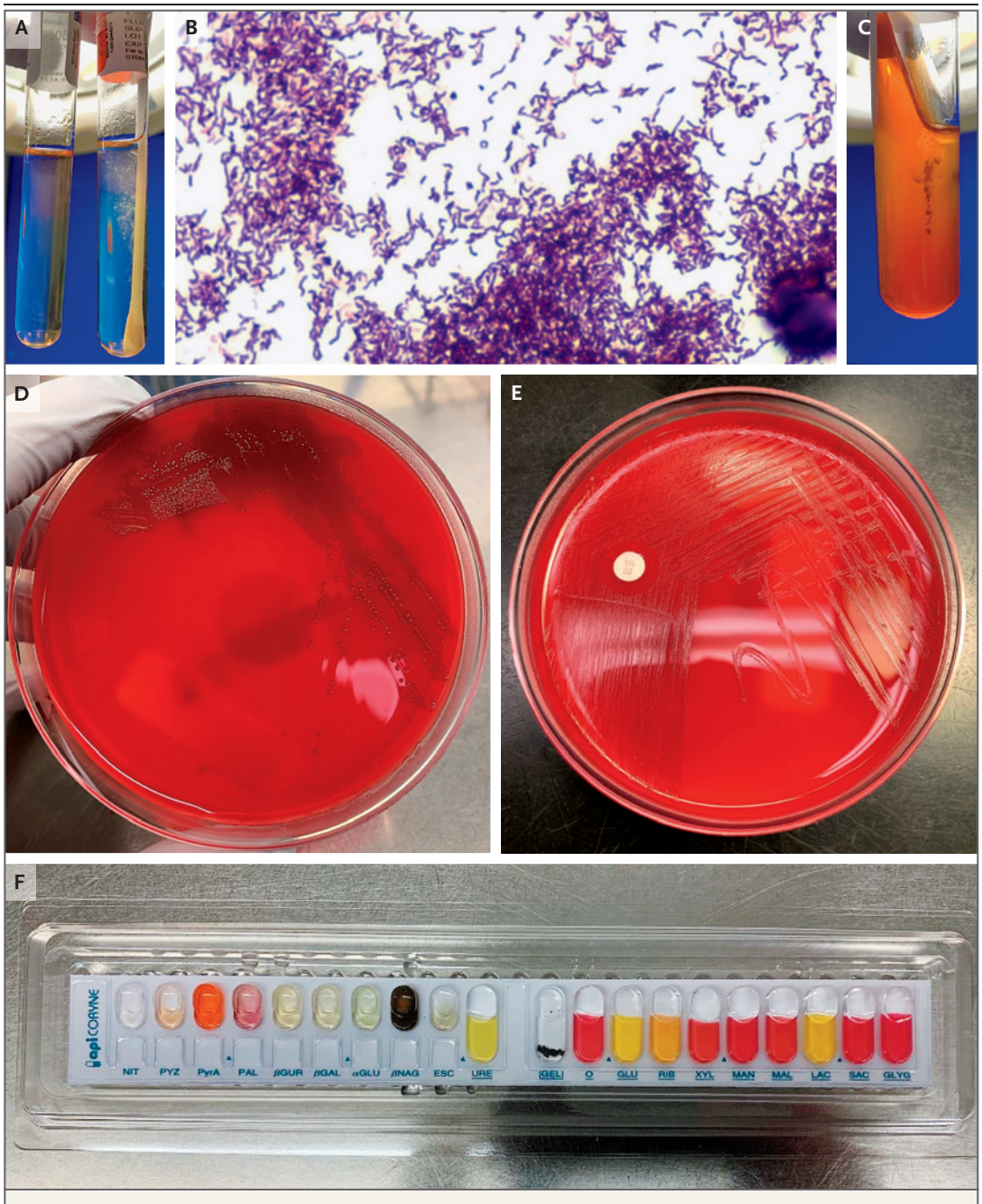

Figure 4. Microbiologic Culture of Skin Specimens.

A superficial swab of a skin lesion was inoculated into thioglycolate broth and incubated in an atmosphere of 3 to 5% carbon dioxide at 35 to 37°C. Panel A shows bacterial growth on day 2 throughout the medium but primarily in the top and middle parts of the tube (right tube), a classic feature of facultative anaerobic bacteria. In Panel B, Gram's staining of the colonies shows gram-positive, rod-shaped bacteria with nonsporulating, nonbranching, filamentous features. Panel C shows that the organism produced hydrogen sulfide after inoculation into a triple sugar iron agar slant tube. The production of hydrogen sulfide is indicated by the presence of ferrous sulfide (shown as a black precipitate), which is produced when ferrous ammonium sulfate (present in the triple sugar iron agar) reacts with hydrogen sulfide gas. Panel D shows that the organism developed small alpha-hemolytic colonies after subculture and overnight incubation on routine sheep-blood agar. Panel E shows bacterial colonies abutting a 30-μg vancomycin disk, a finding that indicates antimicrobial resistance. In Panel F, a biochemical test panel confirms that the pathogen is *Erysipelothrix rhusiopathiae*.

acid for ease of dosing. Amoxicillin–clavulanic acid is taken two times per day and provides broader coverage than amoxicillin alone, which is taken three times per day and has limited anaerobic coverage. Another option would have been penicillin, which is taken four times per day. Antibiotic therapy was administered for a total of 14 days, given the severity of the infection at presentation. On his final day of therapy, the patient was seen in the orthopedic surgery clinic for the removal of sutures from the punch biopsy. At that time, his lesions had nearly healed. The full range of motion had returned to his hand and fingers, and the infection appeared to have resolved.

FINAL DIAGNOSIS

Erysipelothrix rhusiopathiae infection.

This case was presented at the Massachusetts General Hospital and Harvard Medical School postgraduate course "Infectious Diseases in Adults 2021," directed by Nesli O. Basgoz, M.D., Rajesh T. Gandhi, M.D., Sandra Bliss Nelson, M.D., and Rochelle P. Walensky, M.D., M.P.H.

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

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