

From the Epidemiology Archives

The case of the arthritis epidemic

The following information was brought to the attention of Allen C. Steere, a rheumatologist at Yale University. A cluster of cases of arthritis had occurred in three townships in Connecticut between June and September. The rate of disease was 1 in 10 children whereas the rate in the rest of the country was 1 in 100,000 children. Twenty-five percent of the patients remembered having a skin rash during their arthritis episode. The disease was treatable with penicillin. Steere concluded that this was a new infectious disease and did not have an environmental, genetic, or immunologic cause.

Questions

1. What was the factor that caused Steere to reach his conclusion?
2. What is the disease?

The case of encephalitis

On May 30, a 22-year-old man complained of right hand weakness.

On June 1, he complained of right arm numbness.

On June 2, he exhibited several episodes of staring and unresponsiveness lasting 10-15 seconds. He consulted a physician in Mexico, who prescribed an unknown medication. That evening he presented himself to a hospital emergency room in Texas complaining of right hand pain. He had been punctured by a catfish fin earlier in the week, so, based on this information, he was treated with ceftriaxone and tetanus toxoid.

On June 3, when he returned to the emergency room complaining of spasms, he was hyperventilating and had a white blood cell (WBC) count of 11,100 per mm³. Although he was discharged after reporting some improvement, he began to have intermittent episodes of rigidity, breath holding, hallucinations, and difficulty in swallowing. Eventually he refused liquids. That evening he was admitted to the intensive-care unit of another hospital in Texas with a preliminary diagnosis of either encephalitis or tetanus. Manifestations included frequent spasms of the face, mouth, and neck; stuttering speech; hyperventilation; and temperature of 37.8°C. The WBC count was 17,100 per mm³ with a granulocytosis. He was sedated and observed.

On the morning of June 4, the patient was confused, disoriented, and areflexic (without reflexes). Although his neck was supple, muscle tonus was increased in his upper extremities. Analysis of cerebrospinal fluid indicated slightly

elevated protein; slightly elevated glucose and 1 WBC per 0.1 mL. An electroencephalogram showed abnormal activity. Because he had uncontrolled oral secretions, he was intubated. His temperature rose to 41.7°C and he was sweating profusely.

The man died on June 5.

The patient had worked as a phlebotomist for a blood bank and had donated blood on May 22. His platelets had been transfused before he became ill, but the remainder of his blood products were destroyed.

Questions

1. What was the purpose of the ceftriaxone? The tetanus toxoid?
2. What is granulocytosis?
3. What is the most likely cause of the man's illness and death?
4. What other information do you need to be sure?
5. How could he have been treated?
6. How should the platelet-recipient be treated?

Nosocomial Infection

Seventeen patients in ten hospitals had cutaneous infections caused by *Rhizopus* species. In all 17 patients, Elastoplast bandages were placed over sterile gauze pads to cover wounds. Fourteen of the patients had surgical wounds, two had venous line insertion sites, and one, a bite wound. Lesions present when the bandages were removed ranged from vesiculopustular eruptions to ulcerations and skin necrosis requiring debridement.

Questions

1. How did the wound most likely get contaminated?
2. How can this type of infection best be prevented?
3. Is the risk of this type of infection high?

Determining the Effectiveness of a Food Preservative

In order to determine whether a newly synthesized chemical might be a useful food preservative, the chemical was tested for its ability to inhibit bacterial growth.

Control: 500 ml of cottage cheese was inoculated with 2 ml of a 24-hr culture of *Pseudomonas aeruginosa* and incubated at 25°C. Five hours after inoculation, a standard plate count showed there were 200 bacterial cells/ml in the cottage cheese. After 29 hours at 25°C, there were 1,000,000 cells/ml in the cottage cheese.

Experiment: 500 ml of cottage cheese was inoculated with 2 ml of a 24-hr culture of *P. aeruginosa*. After 6 hours of incubation at 25°C, a standard plate count was performed. There were 700 bacterial cells/ml in the cottage cheese. After 38 hours, there were 61,000,000 bacterial cells/ml in the cottage cheese.

Number	Log
1	0.00
2	0.30
5	0.70
6	0.78
24	1.38
32	1.51
200	2.30
700	2.85
1.00×10^6	6.00
6.10×10^6	6.79
6.10×10^7	7.79

Questions

1. Why were plate counts used instead of direct microscopic counts or turbidity measurements?
2. How did the control cottage cheese and the experiment cottage cheese differ? Was this a fair test?
3. Determine the effectiveness of the new food preservative?
4. Does this type of test determine bacteriostatic or bactericidal activity?

The case of the delayed diagnosis

A 25-year-old New Mexico rancher was admitted to an El Paso hospital on February 12th because of a 2-day history of headache, chills, and fever (40°C). The day before admission he began vomiting; the day of admission, an orange-sized swelling in the left axilla was noted. A lymph-node aspirate and a smear of peripheral blood were reported to contain gram-positive cocci, often in pairs. Under the assumption that a

gram-positive organism had caused the patient's illness, he was given cefoxitin. The man was acutely ill. Within a few hours of admission, he had a cardiopulmonary arrest. During resuscitation efforts, he vomited and aspirated his vomitus; a chest X-ray showed bilateral infiltrate. Additionally, the patient bled from several body sites. The patient died within 6 hours of admission. In the 2 weeks before becoming ill, the patient had trapped, killed, and skinned 3 kit foxes, 4 coyotes, and 1 bobcat. The patient had cut his left hand shortly before skinning the bobcat on February 7th.

After his death, biochemical testing of a gram-negative rod isolated from blood cultures identified the etiology as *Enterobacter agglomerans*.

Questions

1. Identify the following periods: incubation, prodromal, illness, decline.
2. Identify the etiologic agent of this disease. Briefly explain how you arrived at your conclusion.
3. What microbiologic tests would you perform to verify the etiology?
4. How might have the patient been treated between February 7th and 12th?
5. What special precautions needed to be taken by the hospital and mortuary personnel?

Infectious Ulcers

In 1981, the following information came to the attention of Barry Marshall, a gastroenterologist at the Royal Perth Hospital in Australia. Household members of ulcer patients do not develop antibodies against *Helicobacter*. However, clinical staff involved in obtaining biopsy samples from ulcer patients develop antibodies against *Helicobacter*. If acid-suppressive therapy is combined with antibiotics, ulcers usually do not recur. Marshall concluded that ulcers are an infectious disease.

Questions

1. What caused Marshall to reach his conclusion?
2. What additional proof would be needed?