Battlefield Medicine: The American Response to Gas Gangrene on the Western Front

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Introduction

On the eve of the Second World War Dr. Harold Macumber summarized the challenge faced by surgeons confronting wounds complicated by the invasion of gas producing bacilli. He wrote: "No other disease that falls to the lot of the surgeon places so heavy a burden on his judgment nor is so dependent upon his management as this dread condition."[1]

These words were apt, for in World War I, gas gangrene gained a lethal reputation because it complicated 1% of open wounds and 6% of open fractures.[2] Fortunately, this was not repeated in the Second World War because of the combination of World War I surgical techniques with the antibiotic therapy: penicillin.

Although gas gangrene is a military disease it still presents itself as an acute medical emergency. It has a new name: clostridial myonecrosis and a new term for its cause: clostridium perfringens. However, these terms are refinements of the microbiological analysis initiated during the war that give us a clear picture of the disease.

This picture was not clear in the fall of 1914. In fact, doctors couldn't develop a wound management strategy without first discovering the cause of gas gangrene[3] Therefore, the story of what led to the discovery is appropriate for this ASM meeting.

Between 1914 and 1918 microbiologists and physicians described the microorganisms that caused the infection. This led to prophylactic measures administered on the battlefield and during hospitalization.

To tell this story this presentation examines and develops its history through six topics.

Incidence

How many American soldiers had gas gangrene infections in World War I?

Over thirteen months of warfare the American army suffered 224,080 wounded.[4] Many of these wounds were complicated by gas gangrene.

The wounds were divided into two categories: first, injuries to soft parts of the body, and second, to fractures of the bone. Category one had 128,265 injuries, of which 1,389 developed gas gangrene, or over 1%. The deaths for this group were 674 or 48%

morality. Category two included 25,272 bone fractures resulting in 2,751 deaths. Gas gangrene was higher in fractures, at 1,329 cases and 593 deaths, or 44% mortality.[5] These figures are evidence of why gas gangrene earned its lethal reputation.

A study made by Evacuation Hospital No. 8 is illustrative. This hospital, only10 hours from the front, admitted 4,741 wounded from September 10 to November 13, 1918, of which 4,683 required surgeries.

Two measures that indicate the severity of gas gangrene are the number of amputations, their reason and the number of deaths. Out of 4,683 procedures there were 206 amputations of which 93 were for severity of the injury, 17 for infection and 96 for gas gangrene. The surgeons charted the locations and noted for gas gangrene the leading ones were arms, 68%, thigh, 75% and legs, 58%. During this period, they reported 363 deaths for which gas gangrene was second highest at 61, with the highest at 88 from shock and hemorrhage.[6]

They also charted 511 admissions based on the time between injury and an operation and whether they had gas gangrene. Their 6-hour interval measured from 0 to 120 hours.[7] The data revealed gas gangrene was highest between 18 to 48 hours. From 0 to 12 hours it was modest then doubled and began to drop off after 48 hours. This data tells us: What the incubation period was for the bacilli; the amount of time the army had to remove the man from the battlefield; administer first aid and stabilize him for evacuation to where he could receive surgical care before the infection progressed to where he was seriously compromised.

Etiology

What was the cause of gas gangrene in World War I?

A soldier contracted gas gangrene by receiving a traumatic injury that caused intensive damage to a muscle group, including the circulatory system, and by having clothing, soil and other material deposit anaerobic bacteria into the body. Without trauma anaerobic bacteria are harmless, for unlike aerobic bacteria which survive in an oxygenated environment anaerobic bacterium cannot.[8]

Early in the war, confusion as to the cause of the infection came from language barriers, different methods of investigation, limited literature and confusion between common pathogenic germs with the cause of the infection.[9] However, by late 1914, anaerobic bacilli were identified as the cause. Their growth was favored by dead muscle tissue, which allowed the bacilli to secrete toxins which in turn destroyed muscle tissue.

Trauma was essential for the genesis of gas gangrene because it caused the conditions of ischemia and subsequent necrosis that created an environment for the bacteria's growth. Trauma need not be severe if the wound was deep and within a large muscle group.

By December 1914 microbiologists identified the specific anaerobic organisms that produced the disease, especially those with saccharolytic ferments, which produced the gas that gave the infection its name. They also identified aerobic varieties that were present that played a secondary role by preparing the tissue for the anaerobic bacilli, and organisms that produced putrid abscesses that were found with the infection.[10] This identification helped microbiologists understand how these bacilli affected the extent and severity of the infection.

By 1917 Weinberg and Seguin shed further light on the complexities of the cause of the infection. In a study of 91 cases, they identified 11 different types of bacteria present. Ten patients had a single variety of bacteria, 14 had several, and 67 of them possessed both anaerobic and aerobic bacteria.[11] Their investigation was mirrored by British research. So, from 1916 on, microbiologists were able to explain the cause of the infection, state the most frequent types of bacilli and their degree of virulence. The general conclusion was the most frequent bacteria were B. welchii, followed by B. oedematiens and, although less frequent, Vibrion septique was more virulent.[12] All this suggested it was impossible to identify a single pathological picture for the infection which would impact producing a serum.

Finally, further explanations for the cause must include other conditions such as the patient's lowered resistance due to fatigue, lack of nourishment and shock due to blood loss. Also, local conditions such as humidity, temperature, the nature of the soil, the intensity of warfare and, finally, the length of time between the point of wounding and treatment were all conditions that would influence the possible development of a gas gangrene infection.[13]

Pathology

What were the effects from a gas gangrene infection in World War I?

The entry of anaerobic bacteria into the body was seen as an insidious invasion.[14] If these bacilli were lodged in the body's muscle, they then found an ideal environment for their growth. Their presence was unknown to the patient or physician until the incubation period had passed and the infection presented itself.

However, the presence of the bacilli was not sufficient to declare an infection. Instead, as stated in 1918 by the Allied Committee on Anaerobic Bacteria, the infection began when the bacteria surrounded itself with a zone of toxins that was so concentrated that it overcame the defenses of the affected tissues.[15]

In 1917 Emrys-Roberts and Crowell outlined the pathological changes for the infection by describing the wound's surface; the area of dead muscle; at the spreading edge; and in the contractible part beyond.[16]

At the wound's surface, initial pain intensified over time. Serous exudates from the muscle and subterraneous tissue caused swelling due to edema. This edema whitened the

skin. Edema also gave surgeons a measure to estimate the progress of the infection because it extended along the lymphatic spaces in the neurovascular sheaths and in the subcutaneous tissue.

In the early stage the skin was elastic but turned leather-like, and if gas was present, the skin became crepitus. The change in the skin's condition was matched by changes in color. What was whitened became orange, brown and at a malignant stage blue or violet. If the death of the muscle was caused by circulatory interference, it was purple, and if by gas bacilli, it was a brick red or mahogany.[17]

The discharge from the wound also evolved. At first it was serosanquinolent but as the serous element diminished it became pinkish and then brown. This discharge usually developed a foul odor due to the putrid bacteria in the wound. Later there appeared coalescing vesicles that encircled the extremity or followed the veins.

If the bacteria possessed sacchaolytic ferments, gas was produced. In the early stage it was not in an amount large enough to be noted. But as the infection progressed, its presence was detected by pressure which produced a crackling sound. At this stage as the gas increased, it appeared as bubbles in the discharge. The gas was not toxic, but its pressure on the muscle tissue extended the infection by compressing the blood supply which then contributed to the death of the muscle tissues.[18]

The toxic part of the process came from a toxin produced by the bacilli. The toxin was absorbed into the muscle fibers which in the end progressively killed them. As the infection took hold, it advanced. Compression of the blood supply or other predisposing conditions worsened the invasion and could affect an entire limb or lead to generalized toxemia and death.

The progressive effects of the infection were understood midway through the war. The color and texture of the patient's skin, the color and odor of the discharge and finally the presence of gas were clinical manifestations that advised battlefield surgeons of the presence of the infection and to what degree radical treatment was required.

However, this physical picture did not reflect the complexity of this wound. By 1915 and on Fleming, Ivens, Weinberg and Seguin and others reported other pathogenic organisms coexisted with the infection. It was found that by the process of symbiosis these organisms stimulated the growth of the anaerobic bacteria.[19]

Clinical Picture

What was the clinical picture for a gas gangrene infection in World War I?

The incubation period for the infection was between 12 and 48 hours.[20] The reason this varied was due to the organism's malignancy and the extent of the necrosis of tissue on the wound's track.[21] It was also affected by other conditions such as blood loss, damage to the area's circulatory system and other systemic causes.

Even if a patient escaped an immediate threat of infection, dormant anaerobic bacteria could be activated weeks later following a surgical procedure.[22]

By the end of the war the infection was grouped by symptoms into three types based on virulence, extent and character. These were: mild, malignant and intermediate, but with a caveat that a mild infection might become malignant.[23] Mild cases showed initial signs of infection but the wound healed without complication and remained localized. Surgeons thought these cases ended well because the muscle damage was superficial or the wound's exposure to air prevented the bacteria's growth.[24]

A mild infection presented the same clinical picture as a malignant one. It was up to the examining surgeon to judge what he saw before the more dramatic signs of infection -- odor, swelling and color -- first appeared. Two early symptoms suggestive of infection were the patient's rapid and weak pulse combined with a low body temperature of 101-103 degrees. Another sign was rapid breathing, [25] probably due to a decrease in red blood cells, and a marked pallor across the body's surface.

A malignant case, also called a fulminating type, started with the same initial picture, but was characterized by how quickly the infection spread. A malignant case had the same symptoms plus pain at the injury's site, swelling and an evolving discoloration of the skin. If the bacilli produced gas, it too was present. The patient was likely to have contributing complications such as shock, blood loss and exhaustion. The patient might present as cold, pale and restless with a very weak pulse, as well as low blood pressure with the likelihood of death within hours from generalized toxemia.

Prophylaxis

What were the measures taken to prevent gas gangrene infections in World War I?

By the time the Americans arrived in 1918 there were three techniques for treating gas gangrene. The most important was surgical excision followed by irrigating the wound with an antibacterial solution and to a lesser extent injecting a polyvalent serum.

None of these techniques were in use at the start of the war. Consensus for treatment took time and experience from two groups. One, the medical community, faced treating wounds that were compromised by an unknown infection. The second, the microbiologists, faced how to explain what caused the infection.

The explanation for the cause came in 1914 with French and British bacteriological analysis of the soil and from patients.[26] This led to knowledge about the varieties of anaerobic bacteria and provided a framework to investigate whether a serum could be produced.[27] This information, although useful, did not help the surgeon except to suggest they were dealing with bacilli lodged in the body at points that allowed it to grow.

Before being confronted with the complication of invading bacteria, military medicine treated wounds conservatively. They were opened, foreign bodies removed and irrigated with a sterile salt solution or hydrogen peroxide. They were left open to drain and then closed. By 1915, two RAMC doctors Colonel Gray and Major Milligan, pioneered a more radical approach called debridement, or excision.[28]

Both published their results in 1915. They advocated an early excision offered the best hope if it focused on not only removing all debris and devitalized tissue, but went beyond the wound's edge until encountering living tissue. In effect this approach cut away the whole wound. However, it was not unanimously accepted, and it took until 1917 for the British to adopt it, while the French showed less hesitancy.[29]

Thus, the Americans arrived on the battlefield prepared to respond to wounds contaminated with anaerobic bacteria. Abundant literature described techniques and inter-allied conferences shared the latest information. Americans also visited British and French medical units to observe procedures.

By 1918 the army could advise surgeons to anticipate anaerobic infections in injuries involving the maceration or crushing of tissues with an entry point for foreign bodies. And if this condition existed, a prompt debridement should be performed, to include considering an amputation as a realistic option. Prior to the Battle of Chateau-Thierry, June 1918, the army published nine guidelines on how to treat wounds that were susceptible to a gas bacilli infection. They urged an early operation to include what should be excised, the direction of incisions, leaving the wound open and not compressed.

The objectives for excision were to remove all foreign bodies such as shell fragments, clothing and dead tissue, especially necrotic muscle, and remove tissue until the divided muscle bled and contracted. The surgeon would then remove all hematomas and check for bleeding. The wound needed to be free from pockets, so that the discharge could pass without being retained, and unconstructed, so that the blood supply was maintained. Constrictions decreased the blood supply and forced toxins from the bacilli through the lymphatic and vascular channels and into general circulation.[30]

With the question of how doctors should treat gas gangrene answered the questions of when and where remained. Should surgical procedures be done in a hostile environment, where they could be compromised by atmospheric conditions and enemy gun-fire? The army responded to this question by designing a system of care that was delivered through stages from the point of injury to life saving surgery. A wounded man passed through a company aid post to the battalion aid station, to the dressing station, to the triage, to the field hospital and finally to an evacuation hospital. This migration from basic first aid and stabilization led to progressively larger units with more sophisticated medical care, but still on the battlefield.

Although it was advocated to operate at the earliest possible moment, this was not realistic given the size, equipment and staff of the frontline units. Since excision required

a sterile environment and anesthesia, the procedure was best performed at an evacuation hospital. This is not to imply that those units that treated the wounded prior to their arrival at the hospital did not affect patient prognosis. Their treatment for shock, fractures, hemorrhage, and exhaustion all contributed to survival of the evacuation process and eventual treatment at an evacuation hospital.

So, by 1918, the American response to preventing gas gangrene was similar to their allied colleagues who combined surgical excision with an antiseptic treatment and bacteriological analysis. The sequence began with debridement, followed by irrigation of the wound with a chemical antiseptic, which included a bacteriological analysis of the wound. These last two steps were done until it was found the wound was aseptic, which meant it could be closed.[31]

The development of an antiseptic for wound infections should not come as a surprise. The Lister paradigm of antiseptic surgery created hope that a new antiseptic could replace ineffective predecessors. In 1915 a new one, with a delivery system, was announced by Alexis Carrel and Henry Dakin. The antibacterial invented by Dakin was a sodium hypochlorite solution delivered to the wound by a system of tubes designed by Carrel.[32] After evaluation it was adopted by the British in 1917, which was almost a year after the French.

In 1917 William Keen's *The Treatment of War Wounds* published Dakin's formula along with five steps for its use with the Carrel tubes. Keen described the 'Operative Technique to Prepare the Wound' followed by the 'Introduction of the Tubes' and concluded with after care and the bacteriological examination.[33] Keen offered doctors and nurses the dos and don'ts, including a description of the bacteriological analysis to be done every second day. The objective was to count the microbes present in the wound, and when absent for three successive days, there lack of indicated the wound was sterile.[34]

Keen's description suggests the time and attention necessary to make the procedure work, including an aseptic environment where the surgical stage could be performed. This tells us that the procedure could only be initiated at an evacuation hospital and then completed at a rear area hospital. This assessment was shared by Colonel Gray who thought the Carrel tubes too complicated for use by front-line units.[35]

The third initiative was the invention of a serum that could be administered as a prophylactic and after the infection appeared. This goal was a natural outcome from the identification of the cause and characteristics of gas gangrene. Coupled with this was the effectiveness of the serums developed and used for tetanus and diphtheria. However, this optimism clashed with the reality of patients dying before the organism could be isolated, and by the fact that these infections were caused by a variety of bacillus.[36]

Nevertheless, by 1916 the first a single species serum was produced to be followed by a polyvalent serum by Weinberg and Seguin that was a mixture of sera from several of the organisms found in gas gangrene. Other polyvalent serums were added in 1917. However, the test would be whether they worked on humans. The most impressive study

was made by Dr. Frances Ivens on 433 cases where she used three different serums on three groups with a range injuries and gas bacilli infections.[37] In group one 222 received the serum before their first operation and there were no deaths. In group two there were 154 patients and 19 fatalities and in group three there were 57 patients with 2 deaths. This trial suggested to Ivens and others the potential value from using a serum.

In 1917 at the Third Inter-Allied Congress for the Study of War Wounds, the reports given on the use of the serums received a favorable response. Even if they were not as effective as the tetanus antitoxin, enough positive evidence justified there use as a prophylactic treatment for badly contaminated wounds.

The Americans invited French bacteriologists, who worked on serums, in 1918 to the US to help manufacture a serum on a large scale. By the fall of 1918, 5,000 doses reached the AEF hospitals of which 2,500 were used to treat patients.[38] However, the armistice in November brought an end to the urgent need for a serum, and the results from the army's trial left no record in depth as to its effectiveness.

Prognosis

What was the likelihood of an American soldier surviving a gas gangrene infection?

If a man's wound was complicated by gas gangrene, his survival depended upon how soon he received the surgical care that could prevent or control the infection. As stated earlier, mortality for infections was high as it was a reason to amputate a limb. And if located in the shoulder or buttocks this site was the worst, with a 30% mortality rate.[39]

Each case's prognosis depended upon what organisms invaded the body, along with other predisposing conditions that affected one's ability to survive. If we add the variables of the time from injury to treatment and the knowledge and skill of treating physicians, we begin to complete the picture for a patient's survival.

By the time American soldiers were seen by their medical personnel in 1918, they were in a much more advantageous position than their British and French counterparts from 1914 through 1916. They benefited from microbiological and medical knowledge that explained the cause of gas bacilli infections and the best techniques to treat them. Without these three years of knowledge, the American troops would have faced the same grim statistics that the French and British experienced early in the war.

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