

Plague discovered in 1894 by Alexander Yersin, gram(-), Bacillus

Historically: 3 pandemics. The second was the most severe known as (the Black Death)

\*Family: Enterobacteria  
 \*Gram(-), small rods, pleomorphic  
 \*Aerobic, facultative anaerobic, facultative intracellular  
 \*Most have animals as their hosts but they can cause disease in man  
 \*Grow better in blood agar, does not hemolyze blood (appear as white colonies)  
 \*Bipolar staining when stained with leishman's stain  
 \*Ferment glucose and mannitol with the production of acid only (non lactose fermenter so it doesn't produce black precipitation on triple sugar iron test)  
 \*It is pathogenic to mice, white rat and pigs (Rodents)  
 \*and virulence factors (F1, Murine exotoxin, LPS, endotoxin, coagulase, pesticin, plasminogen activator).  
 \*\*\*Several plasmids and virulence factors:-  
 • Anti-phagocytic (V and F1 antigens)  
 • Type 3 secretion system To:  
 1-inject proteins that are pore forming  
 2-inhibit phagocytosis and signaling inside innate immune cells  
 • Adhesins to invade the epithelial cells  
 • plasminogen activator to dissolve clot for systemic invasion



بمزيد المبريد الي البصم  
 لكات البكتريا

Causes :-  
 1- Tissue necrosis  
 2- multi-organ failure  
 3- overwhelming sepsis and death

عسان نعال ال دوات اللي ضاهما  
 وتقدر تنتشر بكل الدم

thrombocytopenia causes petech and purpura



Transmission vector:  
 1-From animals: A-By Fleas bite ( fleas found on rodents may carry disease 78%)  
 B-or direct contact with the infected animal  
 2-From human to human:  
 \*droplet contact - coughing or sneezing on another person (causes primary pneumonic plague)  
 \*airborne transmission - the microorganism can remain in the air for long periods (causes primary pneumonic plague).  
 \*direct physical contact - touching an infected person, including sexual contact.  
 \*fecal-oral transmission - usually from contaminated food or water sources.

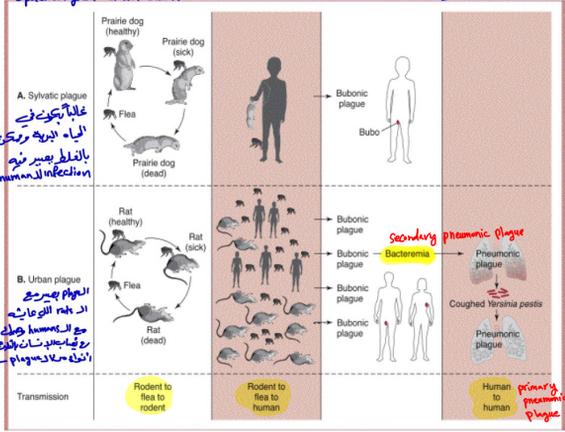
Contrary to popular belief, rats did not directly start the spread of the plague, mainly bubonic. It is mainly a disease in the fleas that infested the rats, making the rats themselves the first victims of the plague.  
 Infection in a human occurs when a person is bitten by a flea or come in contact with infected rat  
 • Several proteins contribute to the maintenance of the bacteria in the flea digestive tract, among them the hemin storage (Hms) system .  
 • Using the Hms sys. the bacteria stick together to form a plug that blocks its stomach and causes it to starve. The flea then bites a host and continues to feed, and consequently the flea vomits blood tainted with the bacteria back into the bite wound.

Micro2

Yersinia pestis causes (plague)

The epidemiology of plague

Epidemiological classification



Disease types

1-Bubonic plague: based on the lymphatic system.  
 lymph node



• 80-90% of cases  
 • IP: 2-6 days  
 • Clinical signs:  
 1-Fever, malaise, chills, headache  
 2-Bubo: swollen, painful lymph node (hemorrhagic inflammatory response)  
 3-vomiting, abdominal pain, nausea  
 • Mortality (untreated): 50-60%

2-Septicemic plague: Centers in the bloodstream.



No IP  
 • blood spread  
 • Clinical signs:  
 1-Similar to bubonic  
 2-circulatory collapse (hypotension...etc)  
 3-septic shock  
 4-organ failure  
 5-hemorrhage  
 6-DIC (disseminated intravascular coagulation)  
 7-Necrosis of extremities (due to Micro thrombi blocking capillaries (black-death))  
 • Mortality (untreated): 100%

3-Pneumonic Plague: Centers in the lungs, may be primary (aerosol) or secondary from blood



• IP: 1-6 days  
 • If this transmitted by aerosols from infected human, this is considered primary, while secondary occurs after symptoms of bubonic or septicemic infection.  
 • Clinical signs:  
 1-Purulent pneumonia  
 2-Fever, chills, headache, septicemia  
 3-Respiratory distress  
 4-hemoptysis  
 • Highly fatal  
 • Person-to-person possible

Other types: Plague meningitis, cutaneous plague, pharyngitis

Diagnosis

1-Isolation of organism, Culture of (blood, sputum, CSF) as indicated  
 \* Gram's stains can confirm the presence of gram-negative rods, and in some cases the leishman's stain (causes bipolar staining)  
 2-Serology (Fourfold rise in antibody titer)  
 \*An anti-F1 serology test can differentiate between different species of Yersinia  
 \*positive=Single titer of >1:128  
 3-leukocytosis  
 4-PCR

Treatment

\*With early treatment - Survival ~100%  
 1-Supportive  
 2-Antibiotics  
 \*Penicillins and cephalosporins are NOT effective why?  
 Because this bacteria is B-lactamases and the penicillin and cephalosporins considered as B-lactams

The main mechanisms of resistance of pathogenic bacteria to β-lactams are :  
 1-changes in the target sites of the drug (penicillin-binding proteins)  
 2-the activity of hydrolysing enzymes produced by the bacteria (B-lactamases)  
 3-the reduced accessibility of the target sites to the drug (permeability barrier). The latter barrier in Gram-negative bacteria consists of the outer membrane (OM)

Prophylaxis

1-Flea, rodent control (kill fleas before rodents)  
 2-Isolation for the suspected patients  
 3-vaccination: Killed vaccine and living attenuated given to travelers and workers with the bacteria and in disaster areas, to prevent bubonic plague.  
 • Primary vaccination: 0, 1-3, & 5-6 months  
 • Boosters: 12, 18, & 24 months, then every 1-2 y