

GLANDERS

(Droes, Farcy, Malleus)

- [Definition](#)
- [Etiology](#)
- [Host Range](#)
- [Geographic Distribution](#)
- [Transmission](#)
- [Incubation Period](#)
- [Clinical Signs](#)
- [Gross Lesions](#)
- [Morbidity and Mortality](#)
- [Diagnosis](#)
- [Field Diagnosis](#)
- [Specimens for the Laboratory](#)
- [Laboratory Diagnosis](#)
- [Differential Diagnosis](#)
- [Treatment](#)
- [Vaccination](#)
- [Control and Eradication](#)
- [Public Health](#)
- [References](#)
- [FAD Table of Contents](#)

Definition [top](#)

Glanders is a highly contagious disease of solipeds caused by *Pseudomonas mallei* and characterized by nodular lesions of the lungs and other organs as well as ulcerative lesions of the skin and mucous membranes of the nasal cavity and respiratory passages. The disease typically has a progressive course and poses a significant human health risk.

Etiology [top](#)

Glanders is caused by the bacteria *Pseudomonas mallei*. Former names of this pathogen include *Loefflerella mallei*, *Pfeifferella mallei*, *Malleomyces mallei*, *Actinobacillus mallei*, *Corynebacterium mallei*, *Mycobacterium mallei*, and *Bacillus mallei*. In experimental infection of guinea pigs *Ps. mallei* produces a tenacious capsule that may serve to protect it from phagocytosis (6). The organism is

closely related to *Ps. pseudomallei*, the cause of melioidosis, and is serologically indistinguishable in some cases (3,8). Genetic homology between *Ps. mallei* and *Ps. pseudomallei* approaches 70 percent. Because of this many consider them to be biotypes or isotypes.

The organism is destroyed by direct sunlight and is sensitive to desiccation. It is readily killed by common disinfectants. It may survive for up to 6 weeks in infected stables (3).

Host Range [top](#)

Glanders is primarily a disease of solipeds — particularly horses, donkeys, and mules. Traditionally, donkeys have been regarded as most likely to experience the acute form of the disease and horses a more chronic form, with mules intermediate in susceptibility (3,8). Recent reports suggest that chronic and even latent infections are equally likely in mules (10). Carnivores are susceptible to disease if they consume glandered meat; felids appear to more susceptible than canids, and outbreaks of glanders in captive wild felids have been reported (1,3,8). Several laboratory animals are susceptible to infection including, hamsters and guinea pigs. The susceptibility of the latter species formed the basis of the Strauss reaction in the diagnosis of the disease. Humans also are susceptible to infection with glanders, which is an important occupational disease of veterinarians, farriers, and other animal workers (8). Swine and cattle are resistant to infection with *Ps. mallei*, but goats can be infected.

Geographic Distribution [top](#)

Glanders is currently limited to parts of Asia, Africa, the Middle East, and Asia (specifically Turkey, Syria, Iraq, Iran, Pakistan, India, Burma, Indonesia, the Philippines, China, and Mongolia) and possibly the Balkan states, former Soviet republics, Mexico, and South America (7,8,9,10). Cross-reactions with serological tests for *Ps. pseudomallei* may confound estimates of worldwide distribution. Although glanders was once widespread throughout the world, it has been eradicated from many countries by diligent test and slaughter programs.

Transmission [top](#)

The disease is introduced into horse populations by diseased or latently infected animals. Ingestion of the pathogen, present in secretions from infected animals, constitutes the major route of infection in glanders. Experimental evidence suggests that inhalation of the organism is less likely to result in typical cases of the disease. Although invasion by way of skin lesions is possible, it is regarded as

being of minor importance in the natural spread of the disease. Close proximity alone does not usually result in transmission of glanders; transmission is facilitated if the animals share feeding troughs or watering facilities or if they nuzzle each other (3,8,10).

Incubation Period [top](#)

After artificial infection, a fever, 106° F (41° C), develops in about 3 days and clinical signs within a week. After natural infection, weeks or months may elapse before manifestations of the disease are apparent. Such latent infections are a feature of the epidemiology of glanders.

Clinical Signs [top](#)

Classical descriptions of glanders distinguish between cutaneous, nasal, and pulmonary forms of the disease, but in most outbreaks these forms are not clearly distinct and may occur simultaneously in an animal. Chronic infections with slow progression of an insidious disease are more common than the acute form of glanders. The acute form (more common in donkeys and mules than in horses) typically progresses to death within about a week.

The nasal form of glanders is characterized by unilateral or bilateral nasal discharge. The yellowish-green exudate is highly infectious. The nasal mucosa has nodules and ulcers. These ulcers may coalesce to form large ulcerated areas, or they may heal as stellate scars of the mucosa. In some cases the septum may even be perforated. Nasal lesions are accompanied by enlargement and induration, or sometimes rupture and suppuration, of regional lymph nodes.

In the cutaneous form of glanders, multiple nodules may develop in the skin of the legs or other parts of the body (Fig. 56). These nodules may rupture, leaving ulcers that discharge a yellow exudate to the skin surface and heal slowly. Cutaneous lymphatic vessels in the region become involved. They become distended and firm by being filled with a tenacious, purulent exudate (3,8). (These may be referred to as "Farcy pipes.") In the pulmonary form of glanders, lesions in the lungs develop in concert with nasal and cutaneous lesions or may be the sole manifestation of the disease (typical of latent cases). The lung lesions begin as firm nodules or as a diffuse pneumonic process (Fig. 57). The nodules are gray or white and firm, surrounded by a hemorrhagic zone, and may become caseous or calcified. Clinical signs in animals with lung lesions may only range from inapparent infection to mild dyspnea, or severe coughing and obvious lower respiratory tract involvement (3,8).

Lesions may also occur in the liver or spleen and, in male animals, glanderous orchitis is a common lesion (5,8).

Gross Lesions [top](#)

Nodular lesions of glanders are most consistently found beneath the pleura of the lung. In some acute cases, however, a more diffuse form of lobular pneumonia may be present. The nodular lesions, typically about 1 cm in diameter, consist of a gray or white core of necrotic material that may become calcified and are surrounded by a zone of hyperemia and edema. Similar lesions may be found in other viscera. Glanderous orchitis may be seen in intact males.

Nasal lesions consist of submucosal nodules surrounded by a small zone of hyperemia. These nodules may rupture, leaving exudative ulcers. As new lesions develop it is not unusual to find small nodules, ulcers, and scars side by side. Lymphadenitis of associated lymph nodes is a consistent finding. In some cases laryngeal lesions similar to the nasal lesions may be found.

Cutaneous lesions consist of cord-like thickening of subcutaneous lymphatics along which are distributed chains of nodules, some of which are ulcerated.

Morbidity and Mortality [top](#)

When horses, donkeys, and mules are concentrated, the morbidity can be high.

Diagnosis [top](#)

Field Diagnosis [top](#)

Typical nodules, ulcers, scars, and a debilitated condition can be sufficient to diagnose glanders. Unfortunately, many cases of glanders are latent and clinically inapparent. Therefore, systematic testing is essential to identify all infected animals in an outbreak (3,5,8,9). The mallein test has been the mainstay of field diagnosis. Mallein is a lysate of *Ps. mallei* containing both endotoxins and exotoxins elaborated by the organism. Infected animals are allergic to mallein and exhibit local and systemic hypersensitivity after mallein inoculation similar to that exhibited in tuberculin testing. Inoculation with mallein may trigger a humoral serologic reaction to the complement fixation test. This seroconversion is thought to be transient but may be permanent if the animal undergoes repeated mallein testing. This is extremely important to consider if animals are destined for export to countries that depend on the complement fixation test.

The preferred method of application of mallein is intrapalpebral. The mallein (0.1 ml) is injected into the dermis of the lower eyelid. In positive cases marked edema of the eyelid, purulent conjunctivitis, photophobia, pain, and depression may be observed within 12 to 72 hours. The test is usually read 48 hours after injection.

The ophthalmic mallein test consists of the instillation of mallein into the conjunctival sac. A positive reaction is characterized by development of severe purulent conjunctivitis within 6 to 12 hours. A larger volume of dilute mallein (2.5 MI) may be injected subcutaneously, causing fever, local swelling, and pain in positive animals.

Specimens for the Laboratory [top](#)

A whole or section of a lesion and a serum sample should be collected aseptically. The samples should be kept cool and shipped on wet ice as soon as possible. Sections of lesions in 10 percent buffered formalin and air-dried smears of exudate on glass slides should be submitted for microscopic examination.

Laboratory Diagnosis [top](#)

The causative organism may be cultured from fresh lesions or lymph nodes. It may also be demonstrated microscopically in films made from this material.

The Strauss reaction is observed when infectious material from glanders patients is injected intraperitoneally into male guinea pigs. In positive cases, the guinea pig develops localized peritonitis involving the scrotal sac. Glanderous orchitis follows with painful enlargement of the testes. The testis becomes enlarged and painful and ultimately necrotic and is discharged through the scrotal skin.

A variety of serologic tests for glanders have been developed. They are superior to mallein testing in sensitivity and specificity. The complement fixation test is widely used and is reported to have an overall accuracy of 95 percent. A counter-immunoelectrophoresis test has been described (4). Recently a dot enzyme-linked immunosorbent assay has been developed and found to be superior to all previously described tests in its sensitivity. This test is inexpensive, rapid, and easy to perform and is not influenced by anticomplement activity (11). Cross-reactions with *Ps. pseudomallei*, the cause of melioidosis, are features of all of the serological tests for glanders. Therefore, these tests will result in false positive reactions in animals from areas where melioidosis is endemic.

Differential Diagnosis [top](#)

Signs of glanders must be distinguished from strangles, epizootic lymphangitis, ulcerative lymphangitis, melioidosis, and other forms of pneumonia. Purulent sinusitis, guttural pouch empyema, and other causes of nasal catarrh should also be considered. Skin lesions may be similar to those of dermatophilosis or dermatomycoses such as sporotrichosis. Knowledge of the progressive debilitating nature of glanders and application of serological or mallein tests will serve to distinguish glanders from other similar diseases.

Strangles is caused by *Streptococcus equi*. It is characterized by fever, anorexia, and depression with swollen submandibular lymph nodes and mucopurulent nasal discharge. The nasal discharge is usually bilateral, whereas it is most often unilateral in cases of glanders. Skin nodules and typical lung lesions are absent. Animals with strangles will not react to mallein testing or serological tests for glanders. *S. equi* is readily demonstrable. Strangles does not develop into a chronic, debilitating condition, and most infected horses recover within a few weeks.

Epizootic lymphangitis (caused by *Histoplasma farciminosum*) is characterized by cutaneous nodules originating from superficial lymph vessels. In epizootic lymphangitis, conjunctivitis is a common lesion. Demonstration of the infectious agent and application of the mallein test and serological testing will help distinguish between these diseases.

Ulcerative lymphangitis (caused by *Corynebacterium pseudotuberculosis*) is characterized by dermatitis and abscess formation predominantly in the pectoral and ventral abdominal regions. Standard diagnostic tests are again valuable in distinguishing this disease from glanders.

Melioidosis (caused by *Ps. pseudomallei*) is characterized by multiple abscesses in a variety of tissues and organs. Unlike glanders, it is not specifically a disease of equids and occurs most often in sheep, goats, and swine. It is characterized by dyspnea and lameness, but a wide array of clinical signs may be elicited. Diagnosis is confirmed by isolation of the causative organism. Serological cross-reactions occur with *Ps. mallei*.

Treatment [top](#)

Ps. mallei is sensitive to many antimicrobials (2,5), but the risk of spreading infection to other equids or to people dictates that infected animals be destroyed. This policy has successfully eradicated glanders from most parts of the world. Sulfonamides have traditionally been used for the treatment of human infection (8).

Vaccination [top](#)

Protective vaccines have not been developed.

Control and Eradication [top](#)

In endemic areas, routine testing and destruction of positive animals have proven successful in the eradication of the disease. Particular care is required where animals are congregated — most often for military purposes. In endemic areas, communal feeding and watering sites should be avoided.

Ps. mallei is quite sensitive to heat, desiccation, and common disinfectants. In warm, moist environments, however, it may remain viable for several months. In outbreaks it is important to bury or burn all contaminated bedding and foodstuffs to prevent infection of susceptible animals. Stalls and harness equipment should be thoroughly disinfected. Removal of susceptible species from contaminated premises for a period of months is advisable.

Public Health [top](#)

People are susceptible to glanders. The human form of the disease is painful and frequently fatal. Laboratory workers and animal attendants are most at risk. Symptoms of glanders in people include nodular eruption on the face, legs, and arms; involvement of the nasal mucosa; and later pyemia and metastatic pneumonia. Human glanders may be confused with a variety of other diseases, including typhoid fever, tuberculosis, syphilis, erysipelas, lymphangitis, pyemia, yaws, and melioidosis. The diagnosis can be confirmed by serology and by isolation of the causative organism.

GUIDE TO THE LITERATURE [top](#)

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