

EPIZOOTIC LYMPHANGITIS

(Pseudoglanders, Histoplasmosis farciminosi, Equine Blastomycosis, Equine Histoplasmosis, Equine Cryptococcosis, African Farcy)

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Definition [top](#)

Epizootic lymphangitis is a chronic infectious granulomatous disease of the skin, lymph vessels, and lymph nodes of the neck and legs of horses caused by *Histoplasma farciminosum*.

Etiology [top](#)

Epizootic lymphangitis is caused by a dimorphic fungus, *Histoplasma farciminosum*, formerly known as *Cryptococcus farciminosus*, *Zymonema farciminosus*, *Saccharomyces farciminosus*, or *H. capsulatum* var. *farciminosum*. In tissue, the organism is present in a yeast form; it forms mycelia in the environment, has a saprophytic phase in the soil, and is relatively resistant to ambient conditions, which allows it to persist many months in warm, moist

conditions (3).

Host Range [top](#)

The natural host range seems to be limited to horses, donkeys, and occasionally mules. Rare cases of human infection have been reported, but identification of the causative organism has not been substantiated.

Geographic Distribution [top](#)

Currently the disease is endemic in west, north, and north-east Africa, the Middle East, India, and the Far East. The disease earned its designation of epizootic during the international conflicts of the first half of the twentieth century in which large numbers of horses were congregated and moved. Many outbreaks occurred in military animals.

Transmission [top](#)

H. farciminosum is introduced via open wounds. Transmission generally involves infection of wounds by flies contaminated by feeding on the open wounds of infected animals (1,7). (The organism has been isolated from the gastrointestinal tract of flies [1]).

Incubation Period [top](#)

The incubation period is variable and is usually several weeks.

Clinical Signs [top](#)

There is no breed, sex, or age predilection in epizootic lymphangitis. This disease most typically involves the skin and associated lymph vessels and nodes. In addition, the conjunctiva and nictitating membrane may be involved. Occasionally there is involvement of the respiratory tract (1,3,7,8). The body temperature and general demeanor of the animal are not changed. The initial lesion is a painless cutaneous nodule about 2 cm in diameter. This nodule is intradermal and is freely moveable over the subcutis. Lesions are most commonly found on the skin of the face, forelimbs, thorax, and neck or the (less often) medial aspect of the rear limbs. The subcutaneous tissue surrounding the nodule becomes diffusely edematous. The nodule gradually enlarges and ultimately bursts. Some cases do not progress beyond small, inconspicuous lesions that heal spontaneously. More typically, resultant ulcers increase in size and undergo cycles of granulation and

partial healing followed by renewed eruption. The surrounding tissues become hard, variably painful, and swollen. The infection spreads along lymph vessels and causes cord-like lesions, leading to diffuse and irregular involvement of an area of skin. After, a lesion initially increases in size, additional cycles of eruption and granulation lead to progressively smaller areas of ulceration until eventually only a (usually stellate) scar remains. The development and regression of a lesion takes about 3 months (1,3,7,8). Where lesions overlie joints, involvement may extend to synovial structures and produce severe arthritis.

Conjunctivitis or keratoconjunctivitis may occur — usually in conjunction with skin lesions (1). A serous or purulent nasal discharge containing abundant organisms may be observed. Although respiratory lesions are described as common in older literature (3), this form of the disease appears to be rare in more recent outbreaks (1)

Gross Lesions [top](#)

The affected skin and subcutaneous tissue is thickened, fibrous, and firm. Several purulent foci may be apparent on cut section. Lymphatic vessels are distended with pus. Regional lymph nodes are swollen, soft, and reddened and may contain purulent foci. Arthritis, peri-arthritis, and periostitis have been described. The nasal mucosa may have multiple, small gray-white nodules or ulcers with raised borders and granulating bases. Nodules and abscess may occur in internal organs, including the lungs, spleen, liver, and testes (3).

Morbidity and Mortality [top](#)

The incidence of disease is high only when large numbers of animals are collected together (as in military situations, for racing, or on village commonages). Mortality is low.

Diagnosis [top](#)

Field Diagnosis [top](#)

Although the clinical presentation of the disease may lead to a presumptive diagnosis of epizootic lymphangitis, the similarity of this disease to glanders makes laboratory confirmation essential.

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](#)

Demonstration of the yeast in tissue sections or smears of lesions is considered the most reliable means of diagnosis. Attempts to culture the organism fail in up to half of cases (1,2,8). The organism in the tissues is in its yeast form. It may be stained with Giemsa, Diff-Quik, or Gomori methenamine silver (2,8). In addition, an indirect fluorescent antibody technique for demonstration of the organism has been developed (4).

Affected animals do mount a humoral immune response to the infection, and an enzyme-linked immunosorbent assay (ELISA) for the diagnosis of epizootic lymphangitis has been developed (5,6). Attempts have also been made to utilize intradermal skin testing (with histoplasmin or histofarcin) with encouraging results (5,10).

Differential Diagnosis [top](#)

Epizootic lymphangitis must be differentiated from glanders (mallein test and serology, absence of yeasts in the pus), strangles (which usually occurs in outbreak form, affects mainly young animals, is always acute and febrile, and is not associated with cutaneous nodules, buds and ulcers), and ulcerative lymphangitis (which is more acute and caused by *Corynebacterium pseudotuberculosis*).

Treatment [top](#)

Successful treatment with intravenous administration or sodium iodide, oral administration of potassium iodide, and surgical excision of lesions where possible has been reported, but recurrences of clinical signs months later is possible (3,7). In vitro sensitivity of the organism to amphotericin B, nystatin, and clotrimazole has been reported (7,8). In most areas, epizootic lymphangitis is a reportable disease, and treatment is not allowed. Affected animals must be destroyed (1).

Vaccination [top](#)

Horses that recover from clinical infection are immune to reinfection. Although

promising results have been obtained with experimental vaccines, a vaccine is not commercially available.

Control and Eradication [top](#)

Strict hygienic precautions are essential to prevent spread of epizootic lymphangitis. Great care should be taken to prevent spread by grooming or harness equipment. Contaminated bedding should be burned. The organism may persist in the environment for many months.

Epizootic lymphangitis is a chronic disease. Many mildly affected horses recover. Those that do are reputedly immune for life — a belief that has led to a premium being placed in endemic areas on horses with characteristic scars (3). In most areas of the world, however, this is a reportable disease; treatment of clinical cases is not permitted, and destruction of affected horses is usually mandatory. In most areas, epizootic lymphangitis has been eradicated by a strict policy of slaughter of infected animals.

Public Health [top](#)

Although rare cases of human infection have been reported, they have not been substantiated by unequivocal identification of the causative organism.

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