

Caseous Lymphadenitis vs. Oedematous Skin Disease: Diagnostic Differentiation of *Corynebacterium pseudotuberculosis*

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ABSTRACT

Corynebacterium pseudotuberculosis is a Gram-positive intracellular pathogen that causes various disease forms in livestock the most common of which are caseous lymphadenitis (CLA) and oedematous skin disease (OSD). Despite the etiological agent causing both conditions, the conditions differ very much in terms of clinical presentation, lesion characteristics, disease progression and diagnostic need. CLA is normally a chronic suppurative but with lymph nodes and OSD is an acute disease caused by toxins with diffuse subcutaneous edema. Proper distinction between the two conditions is essential to proper management of the disease, epidemiological monitoring and effective administration of the correct control measures. This review indicates the microbiological, clinical characteristics as well as methods of diagnosis between CLA and OSD.

Keywords: Caseous Lymphadenitis, Oedematous, *C. pseudotuberculosis*, CLA, OSD

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Introduction

C. pseudotuberculosis is an economically important pathogen that infects large number of domestic animals especially sheep, goats, cattle and buffaloes. Bacterium has a strain-specific pathogenicity, which causes various clinical syndromes that can make it difficult to make a diagnosis [2]. Two opposite outcomes of the disease are the caseous lymphadenitis and oedematous skin disease among others. Although both CLA and OSD are caused by a common etiological agent, the morphology of lesions, the severity of the disease and the response of the host in response are significantly different in both diseases [7]. These conditions may be wrongly interpreted and treated as a result of misinterpretation may result in improper treatment and subpar control measures. Thus, the veterinarians and microbiologists need to know their diagnostic differentiation.

Etiological Agent and Strain Variability

C. pseudotuberculosis is a non-motile, Gram-positive, pleomorphic, bacillus, which is a member of the family *Corynebacteriaceae*. It is a facultative intracellular pathogen that can survive in macrophages, and that leads to chronic infections [12].

Two major biovars are recognized:

- **Biovar ovis**, commonly associated with CLA in sheep and goats [13].
- **Biovar equi**, more frequently linked to OSD and ulcerative lymphangitis in cattle and buffaloes [1].

Differences in virulence factor expression, particularly phospholipase D (PLD), play a central role in determining disease manifestation [6].

Caseous Lymphadenitis (CLA)

Clinical Presentation

CLA is a persistent illness that mostly affects sheep and goats with the development of abscess in both the superficial and deep lymph nodes. Animals can be presented with clinical normalcy, until rupture of abscesses or internal organ involvement [5]. The illness develops gradually, and in most occasions the results are weight loss, low productivity as well as condemnation of the carcasses. Internal CLA can go unnoticed that it cannot be detected with the use of advanced diagnostic tools.

Lesion Characteristics

A well-encapsulated abscess with thick caseous pus is the hallmark lesion of CLA. The pattern of concentric layers on sectioning a subcutaneous abscess bears an appearance of an onion-ring outlook due to repeated bacterial replication and immune reaction. The lesions are local and chronic and have minimal surrounding inflammation owing to fibrous encapsulation [10].

Oedematous Skin Disease (OSD)

Clinical Presentation

OSD is a serious disease that is acute, accelerated and reported mostly in buffaloes and cattle. It is defined by painful and acute swelling onset especially on neck, brisket, limbs and ventral parts of the body. Systemic signs that are common among affected animals include fever, lethargy and

anorexia. In extreme instances, toxemia and death can be experienced in a short period of time [11].

Lesion Characteristics

OTSD lesions are non-encapsulated and diffuse in contrast to CLA. Phospholipase D acts to produce an excessive level of edematous subcutaneous tissue by raising the vascular permeability. Blood loss, serous liquid formation, and necrosis can be diagnosed, which proves the acute and aggressive character of the disease [3].

Pathogenesis and Role of Phospholipase D

The major virulence factor of *C. pseudotuberculosis* is phospholipase D that is central to both CLA and OSD. PLD helps bacteria to disseminate to lymph nodes and survive intracellularly in CLA [4]. Overexpression of PLD causes endothelial damage, enhanced vascular permeability and general edema in OSD. The distinction between the presence and absence of PLD is not in the intensity but in the expression and host-pathogen interaction of the pathogen [14].

Diagnostic Differentiation

Clinical and Gross Examination

- **CLA:** Chronic, localized lymph node abscesses; thick caseous material; minimal systemic signs
- **OSD:** Acute diffuse swelling; severe edema; systemic illness
Clinical history and disease progression provide important initial clues.

6.2 Laboratory Diagnosis

Both conditions require confirmation of *C. pseudotuberculosis* through:

- Culture and biochemical identification.
- PCR targeting the *pld* gene.
- Serological tests (e.g., ELISA), particularly useful for herd-level screening in CLA [9].

Further histopathology aids differentiation by revealing granulomatous inflammation in CLA versus acute edema and vascular damage in OSD.

Epidemiology and Transmission

The spread of CLA is mainly done by contaminating skin wounds during shearing, manipulating, or by fomites. It is extremely common in the small ruminants of the world. OSD outbreaks are sporadic and frequently linked with environmental stress, poor hygiene, or a result of activity of the vectors [8]. Early diagnosis is crucial since the infection spreads rapidly and is very morbid.

Treatment and Control

Caseous Lymphadenitis

Antibiotic therapy is not effective because of low penetration into abscesses. The measures of control depend on vaccination, infectious animal culling, and the strict application of biosecurity measures [15].

Oedematous Skin Disease

Early antibiotic therapy may be helpful in acute ones. The supportive therapy and timely management of environmental risk factors will be used to reduce mortality.

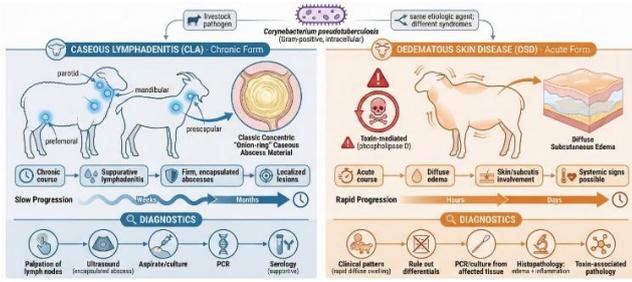


Figure 1: Graphical representation of difference between CLA and OSD caused by *C. pseudotuberculosis*.

Conclusion

Caseous lymphadenitis and oedematous skin disease though have a common etiological agent they are different clinical entities, which have diverse pathological processes. Effective disease control necessitates accurate diagnostic differentiation that is done using clinical presentation, lesion characteristics, and laboratory confirmation. The identification of such differences helps to develop better surveillance procedures and health management of livestock

References

[1] Arafa, M., Hamouda, S., Rateb, H., Abdel-Hafeez, M., Aamer, A. 2019. Oedematous Skin Disease (OSD) transmission among buffaloes. 19, 15-19.

[2] Bastos, B., Portela, R.D., Dorella, F., Ribeiro, D., Seyffert, N., Castro, T., Miyoshi, A., Oliveira, S., Meyer, R., Azevedo, V. 2012. *Corynebacterium pseudotuberculosis*: immunological responses in animal models and zoonotic potential. 4, 10.4172.

[3] Chakraborti, S., 2023. Phospholipases in physiology and pathology. Elsevier.

[4] Dominguez, M., Jimenez, R., Guerreo, J. 2021. Caseous lymphadenitis: Virulence factors, pathogenesis and vaccines. 12, 1221-1249.

[5] Dopud, M., Reil, I., Zdelar-Tuk, M., Špičić, S., Duvnjak, S. 2025. Caseous Lymphadenitis in sheep and goats—“Cheese Glands”. 56, 303-316.

[6] McKean, S., Davies, J., Moore, R. 2007. Expression of phospholipase D, the major virulence factor of *Corynebacterium pseudotuberculosis*, is regulated by multiple environmental factors and plays a role in macrophage death. 153, 2203-2211.

[7] Moussa, I., Ali, M., Hessain, A., Kabli, S., Hemeg, H., Selim, S. 2016. Vaccination against *Corynebacterium pseudotuberculosis* infections controlling caseous lymphadenitis (CLA) and oedematous skin disease. 23, 718-723.

[8] Mustafa, N., Hian, C., Al-Rashdan, Y., Al-Jaff, K., Ghazali, M., Ibrahim, N.S.J.V.I.S., 2025. Comprehensive review on prevalence of caseous lymphadenitis (CLA) in dairy goats: A systematic review and meta-analysis 2025,074. 23, 1-18.

[9] Nagel-Alne, G., Valle, P., Krontveit, R., Sølvørød, L., 2015. Caprine arthritis encephalitis and caseous lymphadenitis in goats: use of bulk tank milk ELISAs for herd-level surveillance. 176, 173-173.

[10] Scott, P.R., 2016. Cattle and Sheep Medicine: Self-Assessment Color Review. CRC Press.

[11] Selim, S., Series B. 2001. Oedematous skin disease of buffalo in Egypt. 48, 241-258.

[12] Torky, H., Saad, H., Khaliel, S., Kassih, A., Sabatier, J., Batiha, G., Hetta, H., Elghazaly, E., De Waard, M. 2023. Isolation and molecular characterization of *Corynebacterium pseudotuberculosis*: Association with proinflammatory cytokines in caseous lymphadenitis pyogranulomas. 13, 296.

[13] Tsiplakou, E., Mountzouris, K., Zervas, G. 2006. Concentration of conjugated linoleic acid in grazing sheep and goat milk fat. 103, 74-84.

[14] Usatyuk, P., Kotha, S., Parinandi, N., Natarajan, V. 2013. Phospholipase D signaling mediates reactive oxygen species-induced lung endothelial barrier dysfunction. 3, 108-115.

[15] Wagner, C., Saueremann, R., Joukhar, C. 2006. Principles of antibiotic penetration into abscess fluid. 78, 1-10.