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Conference Moderator: Dr. Derek Mosier, Diplomate ACVP
Diagnostic Medicine/Pathology
Kansas State University CVM
Manhattan, KS

CASE I – 04020321 (AFIP 2948688)

Signalment: Bovine, Angus, male, 5 months old.

History: Five-month-old Angus bullcalf found acutely dead. Lung tissues (only) submitted for microbiology and histopathology.

Gross Pathology: Not reported.

Laboratory Results: Aerobic culture of the lung retrieved trace numbers of contaminant bacteria: Alpha-*Streptococcus sp*., *Bacillus sp*., *Lactobacillus sp*. Moderate numbers of *Mycoplasma sp*. were obtained from *Mycoplasma* culture. Lung tissue was positive by PCR for bovine respiratory syncytial virus (BRSV).

Contributor's Morphologic Diagnosis: Severe acute bronchointerstitial pneumonia with syncytia.

Contributor's Comment: This case represents acute bronchointerstitial pneumonia secondary to BRSV infection in a young calf. The lung is diffusely hypercellular with widening of the interlobular septa by congestion, edema and inflammatory cells. The inflammation is intensified around bronchi and bronchioles with flooding of adjacent alveoli by edema. There is necrosis of bronchiolar epithelium and the bronchioles are partially occluded by large foamy macrophages, neutrophils, edema, fibrin and scattered multinucleated (syncytial) cells. Some bronchioles are surrounded by follicular type aggregates of lymphocytes implicating the role of *Mycoplasma* in the lesion.

The histological lesions and conspicuous syncytial cells suggest a viral pneumonia with the main differential diagnoses of BRSV or PI-3. The PCR test on DNA collected from lung tissue was positive for BRSV.

In the original sections obtained for diagnosis, the histological lesions were unique compared to normal field cases submitted to our laboratory because significant lesions from a secondary bacterial infection are not yet present. The features of the viral pneumonia were untainted. In the recuts of the tissue for WSC submission, there is regional variability in the purity of the viral lesion (vs. obscurity by the secondary bacterial component) and variability in the conspicuous numbers of syncytia.

Two excellent reviews on BRSV are provided.[1,2] BRSV was first isolated from an outbreak of respiratory disease in calves from Switzerland in 1970 and first reported in the United States in 1974. BRSV is a member of the pneumovirus genus, *Pneumovirinae* subfamily, *Paramyxoviridae* family within the virus order of *Mononegavirales*. The respiratory syncytial viruses are single stranded, negative-sense RNA viruses. The Pneumovirinae subfamily of paramyxoviruses is unique in that its members lack neuraminidase. The viruses attach to cells via membrane glycoprotein G, and following infection, viral antigen is detected in bronchiolar and alveolar epithelium as well as alveolar macrophages. Although usually a prelude to bacterial infections as part of the bovine respiratory disease complex, BRSV can produce outbreaks of respiratory disease and occasional deaths on its own. Severe BRSV respiratory disease is usually restricted to calves less than 6 months old.

AFIP Diagnoses: 1. Lung: Pneumonia, bronchointerstitial, acute, multifocal, moderate, with necrotizing bronchitis and bronchiolitis, syncytia, and intracytoplasmic eosinophilic inclusion bodies, Angus, bovine.
2. Lung: Peribronchitis and peribronchiolitis, lymphoplasmacytic, multifocal, mild.

Conference Comment: Bovine Respiratory Syncytial Virus (BRSV) is a pneumovirus in the family Paramyxoviridae and is a causative agent of "enzootic pneumonia" or "calf pneumonia". BRSV occurs in a variety of breeds, although some reports state that certain breeds are more susceptible. During natural outbreaks, clinical disease is most severe in 1 to 5 month old calves, is seldom seen in calves less than 2 weeks of age, and is virtually absent in calves over 9 months of age. The high prevalence of antibodies against BRSV suggests that infection is endemic in most areas.[2]

Although the mode of transmission has not been determined, it is thought to occur through direct contact or aerosolization over short distances. Many factors

influence the severity of disease, including: the animal's immune status, environmental conditions, animal management, and the presence of other infectious agents. The pathogenesis of BRSV is not clear; however, some research indicates that immune-mediated mechanisms play a dominant role. BRSV enhances bacterial colonization and adherence and alters the specific and non-specific defense mechanisms of the respiratory tract.[2]

Gross lesions are characterized as typical interstitial pneumonia involving the cranio-ventral region of the lungs. In affected areas, the lungs are consolidated and bronchi and bronchioles often are filled with mucopurulent exudate; hemorrhage and emphysema may also be present. The interlobular septa are expanded by pronounced edema. The cranio-dorsal and dorsal regions of the lungs often appear normal, but may also be markedly distended due to edema and severe alveolar, interstitial, and subpleural emphysema. Bronchial and mediastinal lymph nodes are often markedly enlarged, edematous, and occasionally emphysematous. Histologically, there is a bronchitis and peribronchitis accompanied by a large number of syncytial cells in the nasal and tracheal mucosa and alveolar and bronchiolar epithelium. Viral antigen is first detected in the bronchiolar epithelium, later in type I and type II pneumocytes, and may also be detected in alveolar macrophages. The virus is capable of cell-to-cell transmission, resulting in the generation of characteristic syncytial giant-cells. Degeneration, necrosis, and hyperplasia of bronchial epithelium and of lymphoid tissue around the bronchi are consistently present. The bronchial and bronchiolar exudates consist primarily of epithelial cells, neutrophils, and occasionally eosinophils, and are often accompanied by edema and hyaline membrane formation.[2]

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References:

1. Baker JC, Ellis JA, Clark EG: Bovine respiratory syncytial virus. Vet Clin North Am Food An Pract **13**:425-454, 1997
2. Larsen LE: Bovine respiratory syncytial virus (BRSV): a review. Acta Vet Scand **41**:1-24, 2000

CASE II – 03-21575 (AFIP 2936448)

Signalment: Unknown age, castrated male, mixed breed, *Bos taurus*, bovine.

History: Five out of 100 castrated male feedlot calves in the same pen died suddenly with no clinical signs prior to death.

Gross Pathology: The heart had numerous epicardial, myocardial and endocardial hemorrhages mixed with a few inconspicuous pale streaks.

Laboratory Results: *Haemophilus somnus* was isolated in pure culture from the heart, lung, and a pericardial swab. Immunohistochemical staining of an ear notch biopsy and the heart was negative for bovine viral diarrhea (BVD) virus.

Contributor's Morphologic Diagnosis: Heart: Multifocal and perivascular suppurative myocarditis and epicarditis with vasculitis, thrombosis, hemorrhage, myocardial degeneration and necrosis, and intralesional gram-negative coccobacilli.

Contributor's Comment: The submitted heart sections contain multiple perivascular and random infiltrates of numerous intact and degenerate neutrophils within the myocardium and epicardium. The tunica media of many small venules contains intact and degenerate neutrophils with necrosis of the vascular wall. Many of the affected venules contain fibrinous and fibrinocellular thrombi. The epicardium and myocardium contain multiple hemorrhages, which are often associated with the affected blood vessels. A few of the affected venules and inflammatory foci in the myocardium contain variable numbers of small gram-negative coccobacilli. There are a few foci where the cardiomyocytes are degenerate and necrotic, especially those entrapped within the inflammatory foci.

Haemophilus somnus is a small, gram-negative bacillus that can cause various and numerous clinical disease presentations in cattle.[1] The disease presentations in cattle include, but are not limited to, pneumonia, meningoencephalitis, myocarditis, myocardial abscesses, myositis, polyarthritis, abortion, endometritis, orchitis, epididymitis, placental vasculitis, intestinal thrombosis, laryngeal ulceration, and a single case report of a urachal abscess.[1-9] Although some of the disease presentations can be primary localized infections, such as pneumonia, many of the disease manifestations of *H. somnus* infections in cattle are due to septicemia, such as meningoencephalitis, polyarthritis, and myocarditis.[1]

Often, the septicemic form results in meningoencephalitis, but polyarthritis and myocarditis can be seen singly or in combination with other affected organs.[1] Although the lung can be involved with *H. somnus* septicemia, pneumonia is an uncommon feature of the septicemic disease. When the lung is involved, it more

commonly results in a primary fibrinous pneumonia or suppurative bronchopneumonia. The pneumonia caused by *H. somnus* can be microscopically indistinguishable from that caused by *Mannheimia* (*Pasteurella*) *haemolytica*.[1,10] Most affected cattle develop a fibrinopurulent polyarthritis, particularly in the atlanto-occipital joint.[1] Myocarditis supposedly following an asymptomatic episode of septicemia is a major manifestation of *H. somnus* infection in some parts of North America.[1] In one study performed in Canadian feedlots, *H. somnus* infection was found in 70 out of 92 cases of myocarditis in calves in the feedlot.[11]

The most common macroscopic lesions seen with *H. somnus* septicemia in cattle are multiple foci of hemorrhage and necrosis in multiple organs.[1] Microscopically, the consistent feature of *H. somnus* septicemia is an intense vasculitis, usually of small venules and veins. The inflammation can extend into the surrounding parenchyma of the affected organ. The vasculitis often results in hemorrhage and can result in infarction of the organ. The affected venules often contain fibrin thrombi, which commonly contain colonies of bacteria. These colonies of bacteria are believed to proliferate at the site of thrombosis and are believed not to be bacterial emboli.[1] Although vasculitis is a common feature of *H. somnus* septicemia in cattle, the exact pathogenesis of the vasculitis is not known, but it is believed to be due to *H. somnus*–induced apoptosis of endothelial cells.[12]

AFIP Diagnosis: Heart: Myocarditis and epicarditis, suppurative, perivascular and random, moderate, with vasculitis, thrombi, myocardial degeneration and necrosis, and colonies of coccobacilli, mixed breed, bovine.

Conference Comment: Conference attendees discussed the histopathological changes seen in cardiomyocyte degeneration and necrosis. Cardiac muscle is structurally similar to skeletal muscle and is subject to the same anatomic changes associated with degeneration. Cardiac myocyte degeneration is characterized by a swollen vacuolated sarcoplasm, while necrosis is generally characterized by shrunken, hypereosinophilic or fragmented sarcoplasm, loss of cross-striations, and karyorrhexis, karyolysis, or pyknosis. However, the gross and microscopic appearance of myocardial necrosis is dependent on the interval between the initial insult and death.

In this case, the inflammation, degeneration, and necrosis occurred around, and frequently obscured and disrupted vessels. Other causes of embolic myocarditis include *Salmonella* sp., *E. coli*, and rarely *Erysipelothrix rhusiopathiae*.[13] Clinically, *Clostridium chauvoei* is another common cause of acute death in cattle with no clinical signs prior to death. However, gross and histologic lesions of *C. chauvoei* differ from those of this case. *Clostridium chauvoei* affects skeletal muscle and

cardiac muscle, with gross lesions characterized by focally extensive myonecrosis with edema and emphysema; histologically, the lesions are not vasocentric, but are focally extensive.

Histophilus somni is the proposed new name for *Haemophilus somnus,*[14] and infection causes a variety of disease syndromes, as previously mentioned by the contributor. However, *H. somni* is an opportunistic pathogen that is a relatively non-invasive commensal of the bovine respiratory and reproductive mucosal surfaces. Nonetheless, when factors that favor disease and compromise immunity, such as the stress of transportation, concurrent viral infection, overcrowding, pregnancy, lactation, and harsh weather significantly affect the animal, disease ensues.[15]

Vasculitis is the hallmark of systemic *H. somni* infections; however, the pathogenesis of vascular damage is poorly understood.[15] Identifying the mechanism by which *H. somni* induces endothelial cell damage is difficult because the virulence factors are not well characterized. In one report, the *H. somni* virulence factor LOS (lipo-oligosaccharide) induced endothelial cell apoptosis in a time- and dose-dependent manner in-vitro.[12] Another recent report noted virulence factors such as LOS phase variation, induction of endothelial apoptosis, intraphagocytic survival, and immunoglobulin Fc-binding proteins were important to survival and colonization of *H. somni*.[15]

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References:

1. Jubb KV, Huxtable CR: The nervous system. *In*: Pathology of Domestic Animals, eds. Jubb KV, Kennedy PC, Palmer N, 4th ed., vol. 1, pp. 397-400. Academic Press, San Diego, CA, 1993
2. Storts RW, Montgomery DL: The nervous system. *In*: Thomson's Special Veterinary Pathology, eds. McGavin MD, Carlton WW, Zachary JF, 3rd ed., pp. 440-441. Mosby, St. Louis, MO, 2001
3. Jones TC, Hunt RD, King NW: Diseases caused by bacteria. *In*: Veterinary Pathology, eds. Jones TC, Hunt RD, King NW, 6th ed., pp 439-440. William and Wilkins, Baltimore, MD, 1997
4. Hulland TJ: Muscle and tendon. *In*: Pathology of Domestic Animals, eds. Jubb KV, Kennedy PC, Palmer N, 4th ed., vol. 1, p. 245. Academic Press, San Diego, CA, 1993
5. Ladds PW: The male genital system. *In*: Pathology of Domestic Animals, eds. Jubb KV, Kennedy PC, Palmer N, 4th ed., vol. 3, p. 547. Academic Press, San Diego, CA, 1993

6. Kennedy PC, Miller RB: The female genital system. *In*: Pathology of Domestic Animals, eds. Jubb KV, Kennedy PC, Palmer N, 4th ed., vol. 3, pp. 413-415. Academic Press, San Diego, CA, 1993
7. Barker IK, Van Dreumel AA, Palmer N: The alimentary system. *In*: Pathology of Domestic Animals, eds. Jubb KV, Kennedy PC, Palmer N, 4th ed., vol. 2, p. 102. Academic Press, San Diego, CA, 1993
8. Dungworth DL: The respiratory system. *In*: Pathology of Domestic Animals, eds. Jubb KV, Kennedy PC, Palmer N, 4th ed., vol. 2, pp. 568-569. Academic Press, San Diego, CA, 1993
9. Starost MF: *Haemophilus somnus* isolated from a urachal abscess in a calf. Vet Pathol **38**(5):547-548, 2001
10. Lopez A: Respiratory system, thoracic cavity, and pleura. *In*: Thomson's Special Veterinary Pathology, eds. McGavin MD, Carlton WW, Zachary JF, 3rd ed., p. 170. Mosby, St. Louis, MO, 2001
11. Haines DM, Moline KM, Sargent RA, Campbell JR, Myers DJ, Doig PA: Immunohistochemical study of *Haemophilus somnus*, *Mycoplasma bovis*, *Mannheimia haemolytica*, and bovine viral diarrhea virus in death loss due to myocarditis in feedlot cattle. Can Vet J **45**(3):231-234, 2004
12. Sylte MJ, Corbeil LB, Inzana TJ, Czuprynski CJ: *Haemophilus somnus* induces apoptosis in bovine endothelial cells in vitro. Infect Immun **69**(3):1650-1660, 2001
13. Radostits OM, Gay CC, Blood DC, Hinchcliff KW: Veterinary Medicine A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats, and Horses, 9th ed., pp. 64, 741. W.B. Saunders Company Ltd, Philadelphia, PA, 2000
14. Angen O, Ahrens P, Kuhnert P, Christensen H, Mutters R: Proposal of *Histophilus somni* gen. nov., sp. nov. for the three species *incertae sedis* '*Haemophilus somnus*', '*Haemophilus agni*' and '*Haemophilus ovis*'. Int J Syst Evol Microbiol 53:1449-1456, 2003
15. Siddaramppa S, Inzana TJ: *Haemophilus somnus* virulence factors and resistance to host immunity. Anim Health Res Rev 5(1):79-93, 2004

CASE III – 03-9400 (AFIP 2933956)

Signalment: 3 month old female, Jersey calf, bovine (*Bos taurus*).

History: Three calves from a group of approximately 70 had bloody diarrhea and were recumbent. Animals were euthanized by the practitioner and tissues were submitted to the diagnostic laboratory.

Gross Pathology: The only gross lesions noted on the necropsy report by the practitioner were ulcers throughout the abomasums of the calves.

Laboratory Results: Rare oocysts of *Eimeria zuernii* and *Eimeria ellipsoidalis* were noted on routine fecal examination. A fluorescent antibody test of colon and an immunohistochemical stain of colon were positive for bovine coronavirus.

Contributor's Morphologic Diagnosis: Colon: Colitis, lymphoplasmacytic and eosinophilic, diffuse, moderate with numerous coccidial schizonts and gametocytes, Jersey, bovine.

Contributor's Comment: Diffusely throughout the colon, there is mild to moderate lymphoplasmacytic eosinophilic colitis with many second generation coccidial schizonts and gametocytes present within epithelial cells of colonic crypts. In addition, multifocally there is marked dilation of colonic crypts with occasional epithelial attenuation and crypt hyperplasia. Occasionally colonic crypts are collapsed. Within sections of small intestine examined (sections not submitted with this case), there is multifocal crypt dilation and hyperplasia with occasional crypts containing inflammatory debris.

Histologic lesions within the colon in this case are characteristic for coccidial enteritis in calves. The second generation schizonts of the more pathogenic coccidia enter the crypt epithelial cells of the colon and cecum approximately 14 – 18 days after infection.[2] Virtually all cells lining cecal and colonic glands can be infected.[2] As cells rupture and oocysts are released, the remaining intact glandular epithelium can become markedly attenuated and the glands may even collapse.[2] Crypt hyperplasia occurs in an attempt to regenerate mucosal epithelium in areas where it has been ulcerated and/or denuded.[2]

Eimeria zuernii and *Eimeria bovis* are most often implicated in cases of clinical coccidiosis in cattle up to 2 years of age[3]; however, other coccidia including *E. ellipsoidalis* and *E. auburnensis* are also known to cause less severe diarrhea.[2]

Additionally, laboratory testing indicated infection with bovine coronavirus in this case. It is likely the characteristic histologic lesions of this viral infection are masked by the coccidial colitis present; however, some of the lesions present may non-specifically support the presence of viral enteritis. For example, colonic lesions indicative of coronavirus infection include a mixed inflammatory reaction within the lamina propria, dilated colonic glands with attenuated epithelium and glandular hyperplasia.[1] Additionally, crypt epithelium in the small intestine may also be hyperplastic as a result of coronavirus infection.[1]

AFIP Diagnosis: Colon: Colitis, lymphoplasmacytic and eosinophilic, diffuse, moderate, with crypt loss, regenerative hyperplasia and ectasia, and myriad intracellular coccidia, etiology consistent with *Eimeria* spp., Jersey, bovine.

Conference Comment: Over a thousand species of Eimeria are known that primarily infect intestinal epithelial cells of domestic and wild mammals and birds. The life cycle of each species is host specific and direct. Unsporulated oocysts are shed in the feces and sporulate in the environment to become infectious. Following ingestion, sporozoites excyst, invade intestinal epithelial cells, form trophozoites and undergo asexual multiplication (schizogony, merogony) within a schizont or meront. Merozoites are released and eventually form sexual stages (micro- and macrogametes), which unite to form oocysts.[4] Some common coccidia species of domestic and wild mammals and birds include the following:[3,4,5]

Animal	Coccidia	Organ affected
Cattle	*E. bovis*	1st gen schizont – Jejunum 2nd gen schizont – Cecum and colon
Sheep	*E. ahsata* *E. bakuensis* *E. ovinoidalis*	Small intestine Small intestine Ileum/Large intestine
Goats	*E. christenseni* *E. arloingi* *E. ninakohlyakimovae*	Small intestine Small intestine Large intestine
Equine	*E. leuckarti*	Small intestine
Swine	*I. suis*	Small intestine
Canine	*I. canis*	Ileum, colon occasionally
Feline	*I. felis*	Small intestine, colon occasionally
Mice	*E. falciformis*	Colon
Rabbit	*E. stiedae* *E. intestinalis* *E. flavescens*	Bile ducts Ileum & cecum Ileum & cecum
Birds		
Chickens	*E. acervulina* *E. necatrix* *E. maxima* *E. tenella*	Duodenum Mid-intestine Mid-intestine Ceca
Turkey	*E. adenoeides* *E. meleagrimitis* *E. gallopavonis*	Ceca Mid-intestine Colon, rectum
Geese & ducks	*E. truncata* *E. anseris*	Kidney Mid-intestine

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References:
1. Barker IK, Van Dreumel AA, Palmer N: The alimentary system. *In*: Pathology of Domestic Animals, eds. Jubb KV, Kennedy PC, Palmer N, 4th ed., vol.2, pp. 187–189. Academic Press, San Diego, CA, 1993
2. Barker IK, Van Dreumel AA, Palmer N: The alimentary system. *In*: Pathology of Domestic Animals, eds. Jull KV, Kennedy PC, Palmer N, 4th ed., vol. 2, pp. 297–300. Academic Press, San Diego, CA, 1993
3. Georgi JR, Georgi ME: Protozoans. *In:* Parasitology for veterinarians, 5th ed., pp. 84–91. WB Saunders Company, Philadelphia, PA, 1990
4. Gardiner CH, Fayer R, Dubey JP: An Atlas of Protozoan Parasites in Animal Tissues, 2nd ed., pp. 20-30. The Armed Forces Institute of Pathology, Washington, D.C., 1998
5. McDougald LR: Protozoal infections. In: Diseases of Poultry, ed. Saif YM, 11th ed., pp. 976-981, 986, 989. Iowa State Press, Ames, IA, 2003

CASE IV – 04-K0227 (AFIP 2937322)

Signalment: 2 month old female polled Hereford calf.

History: This calf was found dead in the field where she was housed with her dam and 90 other cows and calves. The calf had recently (< 1 week ago) been moved to pasture from a calving pen, where management had been of good quality (adequate colostrum, good navel care, perinatal vitamin E / selenium injection). No previous illnesses had been identified in this calf.

Gross Pathology: Body condition was fair. The liver was pale and mottled, with capsular petechiation. Mesenteric lymph nodes were slightly enlarged. Lungs were red, wet, and heavy, with obvious interlobular edema in some areas. The rumen contained black, semi-fluid, odiferous debris.

Laboratory Results: Bacteriology: *Listeria monocytogenes* was isolated in large numbers from liver and spleen.

Contributor's Morphologic Diagnoses: 1. Acute multifocal necrotizing adrenalitis and hepatitis with intralesional bacilli.
2. Multifocal renal intravascular bacterial emboli.
3. Multifocal microvascular pulmonary thrombosis.

Contributor's Comment: Histologic lesions were consistent with septicemic listeriosis but were more severe than typically seen.[1] In the liver, multiple foci of acute coagulation necrosis involved over 75% of hepatic parenchyma, and large

numbers of plump gram-positive bacilli were present within hepatocytes and extracellularly at the margin of necrotic and viable parenchyma. Lesions were similar in the adrenal gland, with multiple discrete foci of coagulation necrosis, involving approximately 50% of cortical parenchyma and large numbers of bacilli present among necrotic cellular debris within most foci. In the kidney, dense clusters of similar bacilli filled the lumens of several cortical and medullary interstitial blood vessels and glomerular capillaries. Pulmonary alveoli were flooded with proteinaceous edema fluid occasionally mixed with fibrin or low numbers of macrophages, and a moderate number of alveolar capillaries contained luminal fibrin thrombi.

Small intestinal submucosal lymphoid aggregates were sparsely populated and rare individual mucosal crypts had widely dilated lumens filled with neutrophils and necrotic cellular debris. Immunohistochemistry (IHC) for bovine viral diarrhea virus (BVDV) identified abundant viral antigen in mononuclear cells in the intestinal lamina propria, submucosa, and Peyer's patches; in the tunica media of submucosa blood vessels; and in scattered mucosal epithelial cells. Abundant positive staining for BVDV was also evident in Kupffer cells and few intact hepatocytes in liver; mononuclear cells in lymph node; and tunica media of myocardial blood vessels. IHC was negative for bovine herpesvirus-1 antigen in liver and adrenal gland. Immunosuppression in cattle due to BVDV infection can promote susceptibility to other infectious agents, and BVDV may have predisposed this calf to severe septicemic listeriosis.[2]

AFIP Diagnosis: Liver: Hepatitis, necrotizing, acute, random, severe, with myriad bacilli, Hereford, bovine.

Conference Comment: *Listeria monocytogenes* is a small, rod-shaped, gram-positive intracellular bacterium that causes disease in most species of animals and humans. The organism is ubiquitous in nature and can be found in soil, vegetation, dairy products, animal feces, and sometimes the oropharynx and tissues of healthy animals.[1]

Listeriosis occurs as three distinct syndromes, which ordinarily do not occur together: systemic infection (septemia) in humans, cattle, sheep, swine, dogs, cats, and rodents; encephalitis in humans, cattle, sheep, goats, and swine; and abortion in humans, cattle, and sheep. Less commonly, *L. monocytogenes* is a cause of endocarditis and purulent lesions in other tissues.[1]

Systemic infection is the more common form of listeriosis in monogastric animals and in human infants. The most characteristic lesion in this form is focal necrosis

of the liver. However, lesions may also occur in the spleen, lymph nodes, lungs, adrenal glands, gastrointestinal tract, and brain. Microscopically, there are areas of necrosis infiltrated by mononuclear cells and some neutrophils. The organisms may be seen in sections stained with H&E (Hematoxylin and Eosin) or can be easily demonstrated with B&B (Brown-Brenn) and B&H (Brown-Hopps).[1]

Encephalitis is the most characteristic form of the disease in ruminants. Clinical signs include abnormal posturing of the head and neck, walking aimlessly in a circle ("circling disease"), nystagmus, blindness, and paralysis. The organism is thought to reach the central nervous system by ascending peripheral nerves, particularly the trigeminal nerve, and localizing in the brain stem, particularly the medulla oblongata, and in the spinal cord. Gross lesions are usually absent; however, leptomeningeal opacity and foci of necrosis in the terminal brain stem have been noted.[3] Microscopically, there are perivascular mononuclear cell infiltrates, with or without neutrophils. Diffuse cellular infiltration and microabscessation involving both the gray and white matter may occur, but there is usually relatively little tissue necrosis.[1] However, necrosis and accumulation of gitter cells can be prominent in some cases. Other changes include neuronal necrosis and leptomeningitis.[3]

Listeric abortion in animals is important in cattle and sheep. Abortion usually occurs in the last quarter of gestation without signs of infection in the dam. The fetus dies in utero and may be severely autolyzed when expelled.[1] Placental lesions include severe diffuse necrotizing and suppurative inflammation of both the cotyledons and the intercotyledonary areas. The fetal lesion is an enlarged liver with numerous 1 mm yellow foci. Microscopically, severe inflammation involves the mesenchyme of the villi and the upper intercotyledonary chorion. Chorionic epithelial cells, especially in areas between the villi, are filled with gram-positive bacilli. The cells in the areas of acute multifocal necrotizing hepatitis are also filled with organisms.[4]

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References:

1. Jones TC, Hunt D, King NW: Diseases caused by bacteria: Listeriosis. *In*: Veterinary Pathology, Jones TC, Hunt RD, and King NW, pp 461-463. Williams and Wilkins, Baltimore, MD, USA, 1997
2. Brackenbury LS, Carr BV, Charleston B: Aspects of the innate and adaptive immune responses to acute infections with BVDV. Vet Microbiol **96**:337-344, 2003

3. Storts RW, Montgomery DL: The nervous system. *In*: Thompson's Special Veterinary Pathology, eds. McGavin MD, Carlton WW, Zachary JF, 3rd ed., pp. 441-442. Mosby, St. Louis, MO, 2001
4. Acland HM: Reproductive system: Female. *In*: Thompson's Special Veterinary Pathology, eds. McGavin MD, Carlton WW, Zachary JF, 3rd ed., pp. 617-618. Mosby, St. Louis, MO, 2001

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