Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Chapter 7

Camelidae

Dalen Agnew
Michigan State University, Lansing, MI, United States

INTRODUCTION

The Camelidae family (generally falling in the suborder Tylopoda and the order Artiodactyla) consists of the Old and New World camelids. The 3 Old World camelids (OW) are the dromedary, the domestic Bactrian camel, and the wild Bactrian camel. The lineage of New World camelids (NW) has been more controversial, though most now agree it consists of the genus *Lama* and a separate genus *Vicugna.* The llama belongs to *Lama glama* and the guanaco belongs to *L. guanicoe*. The alpaca and vicuña belong to *Vicugna pacos* and *V. vicugna,* respectively. Interbreeding between NW species and between OW species produce fertile crosses, but to date fertile young have not been produced by crosses between NW and OW camelids (Fowler, 2010c). Only the free-ranging population of wild Bactrian camel is endangered. Bactrian camels held in zoos are generally of domestic origin. Vicuña are listed as vulnerable. This chapter focuses on diseases of camelids, most of which have been described in domesticated and zoo animals. Where disease has also been described in free-range animals, it will be specifically mentioned.

UNIQUE FEATURES

Camelids diverged from ruminants at least 40 million years ago, and while both are foregut fermenters, there many unique features that differentiate them.

Camelids have much smaller red blood cells than domestic ruminants. Cells have an elliptical shape that is thought to prevent intravascular sludging and to have developed as an evolutionary mechanism for drought tolerance (Fig. 7.1). Red blood cells in the NW camelids are 3.2 × 6.5 µm; those of OW camelids are of a similar size. Leukocyte numbers in the blood are generally higher in both OW and NW camelids than other ruminants, with neutrophils being the most numerous (Fowler, 2010d).

Anatomically, the anterior digestive tract is the most important and exceptional feature in camelids. Camelids are foregut fermenters, but the anatomy of the foregut is markedly different from that of other ruminants. A common nomenclature has not been adopted, but most divide the stomach into three compartments: C-1, C-2, and C-3 (Vallenas et al., 1971). Unlike other ruminants, all of the compartments have glandular regions and none have papillae. C-1 fills the left abdomen and is partially divided into cranial and caudal sacs by a tranverse pillar. Glandular saccules are present along the ventral aspect of both the cranial and caudal sacs (Fig. 7.2A). The mucosa of C-2 is largely glandular except for an area along the lesser curvature that forms the esophageal groove. The remaining mucosal surface of C-2 is criss-crossed by bands or crests covered by stratified squamous epithelium forming deep pockets that are lined by glandular epithelium. C-3 is lined entirely by glandular epithelium that appears in three patterns. In the first fifth of its length, there is a retiform (net-like) pattern of shallow depressions along the lesser curvature and folds of mucosa along the greater curvature. In the middle three fifths of C-3, there are permanent longitudinal folds. In the distal fifth of C-3, the mucosa is lined by true gastric glands, is more deeply red and smoother, and the wall is thicker. The pH drops abruptly from 6.5 to 2.0 in the terminal region of C-3.

A common finding in the saccules of C-1 and C-2 are green to black irregular concretions or gastroliths (Fig. 7.2B) These can often be found within multiple saccules and vary in size from 1-2 cm to 10 cm or more in diameter. While these often have projections and sharp edges, they are not associated with perforations or other pathology and are generally considered incidental findings.

The gross morphology of the liver and spleen is significantly different from that seen in other ruminant species. The

Pathology of Wildlife and Zoo Animals. http://dx.doi.org/10.1016/B978-0-12-805306-5.00007-9

normal camelid liver has a remarkably fimbriated caudal margin. The spleen can have multiple deep folds and creases that should not be mistaken for previous healed traumatic lesions.

The male reproductive tract includes a fibrocartilaginous penis with a sigmoid flexure, much like domestic ruminants, though there is some erectile potential at the penile tip. Accessory organs are limited to the prostate gland and paired bulbourethral glands. Both are discrete round structures located dorsal to the trigone and dorsal and lateral to the pelvic urethra at the ischial arch, respectively. Seminal vesicles are not present. Camelids have a dorsal urethral diverticulum located cranial to the ischial arch, making catheterization of the urinary bladder from the penile tip impossible (Fowler and Bravo, 2010).

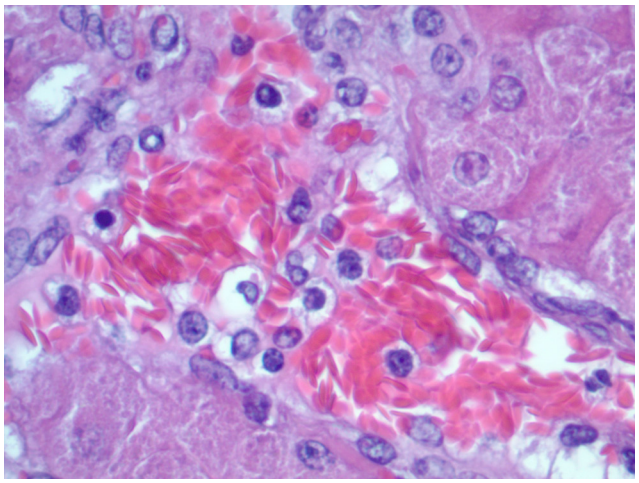

FIGURE 7.1 Normal red blood cells from an alpaca. Camelid erythrocytes have a unique elliptical shape and are much smaller than those of domestic species. This is suggested to be an evolutionary mechanism that prevents blood sludging during periods of dehydration.

Male dromedary and Bactrian camels have a poll gland that secretes a brown to black odiferous fluid that mats the hair along the dorsum of the head (Fig. 7.3). Male dromedary camels have a diverticulum on the ventral soft palate called the dulaa (or dulla or dulah) that the male can fill with air and extrude from the oral cavity during rut or when the animal is excited, disturbed, or anesthetized (Fowler and Bravo, 2010) (Fig. 7.4).

Female camelids have a bicornuate uterus. The camelid placenta is diffuse, epitheliochorial and microcotyledonary. Dome-shaped, highly vascular placentomes develop over the chorionic surface. Each dome corresponds to and inserts into depressions in the uterine mucosa, forming a loosely adherent bond. These domed placentomes become papillated and folded, increasing the surface area over the course of pregnancy. Another unique feature of the camelid fetal membranes is the epidermal membrane, which is an extra membrane of fetal epidermal origin that covers the body, head, and limbs in a near full term fetus. It is attached to the fetus at mucocutaneous junctions and at the coronary band, footpad, and umbilicus (Benirschke, 2008; Fowler and Bravo, 2010). This membrane is composed of a layer of squamous epithelium. The purpose of this membrane is unknown.

NON-INFECTIOUS DISEASES

Nutritional

Nutrition-related disease is relatively common in NW camelids maintained in captivity. While arguably not true "ruminants," both OW and NW camelids can utilize coarse forage by anaerobic fermentation in C-1 and C-2 and both OW and NW camelids are able to utilize coarse forage through anaerobic fermentation in C1 and C2. New World

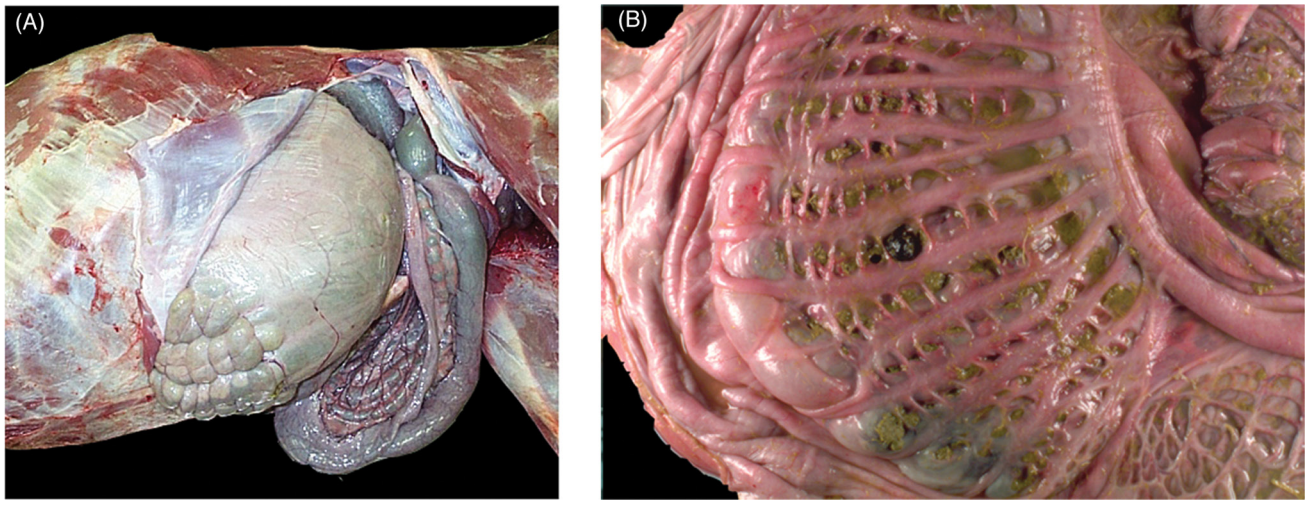

FIGURE 7.2 Normal compartment 1 (C-1) saccules in a llama. (A) The serosal surface of compartment 1 (seen in the ventral aspect of this image) in camelids has multiple glandular saccules. (*Photo Courtesy of A. Tibary, Washington State University*) (B) The mucosal surface of compartment 1 of the normal camelid stomach shows multiple glandular saccules that often contain feed or mineralized concretions. Note the black gastrolith distending one of the glands in the center. These are typically incidental findings.

camelids have evolved in the presence of highly variable food resources, with a "feast and famine" routine – periods of high dietary input followed by very low input. Because of this physiologic adaptation, **over feeding** in captive and domesticated NW camelids is common and can lead to significant consequences. Maintaining and monitoring proper body condition in camelids can be challenging. Due to their heavy wool, NW camelids may be too thin and managers will not recognize this unless they are palpated. In OW camels, there is not a similar "feast and famine" syndrome, but these animals are well adapted to storing nutrients and maintaining condition with poor quality feeds. The hump stores fat and will increase and decrease in size and firmness depending on the nutritional condition (Fowler, 2010b).

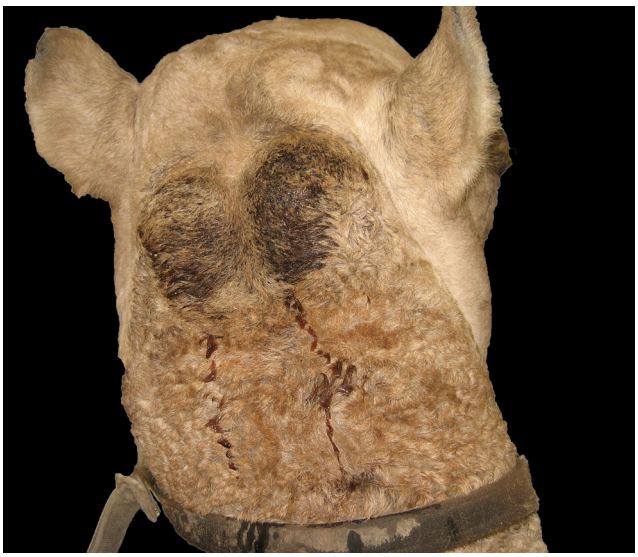

FIGURE 7.3 **Normal poll gland in a dromedary camel**. The poll gland is present in male dromedary and Bactrian camels. It produces an odiferous brown fluid. *(Photo Courtesy of A. Tibary, Washington State University)*

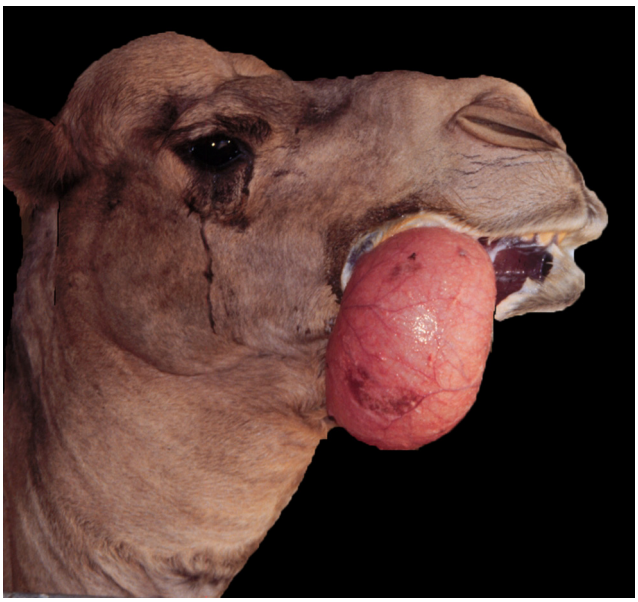

FIGURE 7.4 **Normal dulaa in a dromedary camel**. The dulaa is a diverticulum on the ventral soft palate that male dromedary camels fill with air and extrude during rut or when otherwise excited. *(Photo Courtesy of A. Tibary, Washington State University)*

Obesity, the accumulation of excessive amounts of fat in the subcutis, abdomen, and retroperitoneal cavity is a common problem in captive OW and NW camelids. Without carefully managing animal weight and incorporating lower quality feeds and "lean" periods of nutrition, animals are prone to developing obesity and secondary complications including infertility and susceptibility to hyperthermia.

At necropsy, **starvation** is usually recognized by the absence of abdominal, retroperitoneal, pericardial, subcutaneous, or marrow fat. If mobilization of fat is rapid, it may present as serous atrophy in any of these fat depots. The bone marrow is typically the last to be depleted (Fig. 7.5). Testing of bone marrow for percentage of fat has been validated in other species but not in camelids, though a very low percentage of marrow fat (<10%) is a likely indicator of poor nutritional condition and starvation in camelids (Lamoureux et al., 2011).

Polioencephalomalacia (cerebral cortical necrosis) in ruminants is frequently associated with thiamine (vitamin B1) deficiency resulting from dietary changes that lead to acidosis, such as the feeding of carbohydrate rich grain diets that encourage the proliferation of thaminase-producing bacteria. Thiaminase containing plants and some anthelmintics (e.g., amprolium) may also cause polioencephalomalaica. This condition is commonly diagnosed in camelids, both NW and OW, and is similar to that described in ruminants (Milad and Ridha, 2009). Grossly, diffuse swelling and multifocal necrosis of the cerebral cortex is seen in the superficial gray matter with occasional extension into the underlying white matter. Yellow areas of discolored necrotic tissue will autofluoresce under ultraviolet light (Fig. 7.6). Histologically,

FIGURE 7.5 **Serous atrophy of fat in a llama**. Bone marrow fat is considered the last depot to be depleted during starvation. Serous atrophy of fat is yellow to red, translucent, and gelatinous. This contrasts to normal firm white to tan marrow fat. Atrophy in this marrow cavity was the result of dramatic caloric insufficiency.

bilateral laminar necrosis of the neurons in the deep laminae, edema of the superficial laminae, and gliosis are characteristic. While necrosis is most typically seen in the cerebrum, it can also be found in the cerebellum. Gliosis will vary with the age of the lesion, being more prominent as the lesion matures. Very chronic lesions may contain cystic cavities (Kiupel et al., 2003; Whitehead and Bedenice, 2009).

NW camelids are particularly prone to **hyperglycemia**. Poor glucose tolerance, partial insulin resistance, and low concentrations of circulating insulin are characteristic in these species and are likely a consequence of evolutionary factors that allow them to survive on a uniquely nutrition poor diet (Cebra, 2009). Besides primary metabolic diseases, the effects of these energy disturbances likely exacerbate or predispose to other diseases including sepsis. In particular, environmental, metabolic, or social stress and the release of corticosteroids is likely to lead to persistent hyperglycemia. Dehydration is the most common and clinically significant sequela to persistent hyperglycemia, glucosuria, and glucose diuresis. This, in turn, can be associated with hypernatremia, metabolic acidosis, and azotemia. While these signs may mimic diabetes mellitus, diabetes mellitus is rare in NW camelids (Cebra, 2009).

Sick NW camelids, particularly crias, may develop **hypoglycemia**, though this is less commonly noted than in other species. A number of diseases, including hepatic disease, sepsis, and *Mycoplasma haemolama* predispose to its development (Cebra, 2009). It is also associated with hypothermia and failure of passive transfer in compromised crias that have not nursed (Whitehead, 2009).

Hepatic lipidosis and **hyperlipemia** are also relatively common conditions seen in NW camelids. Grossly, the liver is enlarged with rounded edges and it is pale tan to yellow with a greasy texture (Fig. 7.7). In rare cases, hepatic changes may be so severe as to cause the liver to float in formalin or water. Histologically, changes are diffuse with large clear, distinct cytoplasmic vacuoles and peripheralized nuclei in hepatocytes. Pregnant and lactating camelids are prone to lipidosis in a manner similar to ketotic ruminants with inadequate carbohydrates in the diet (Cebra, 2009). In other NW camelids, stressful events and/or hepatic toxins are more likely responsible. Secondary hepatoencephalopathy associated with lipidosis has been described in llamas. Alzheimer type II astrocytosis in the cerebrum and to a lesser extent in the hippocampus, thalamus, and cerebellum are present in affected animals (Pillitteri and Craig, 2012). Decreased astrocytic immunohistochemical labelling intensity for glial fibrillary acid protein is seen in the brains of neurological llamas with hepatic dysfunction. In addition, distinct from ruminants, fat can accumulate in renal tubular epithelium under similar circumstances as hepatic lipidosis and lead to kidney dysfunction in camelids.

Vitamin E and **selenium** are important anti-oxidants that typically act synergistically via glutathione peroxidase across the taxonomic spectrum. As in domestic ruminants, deficiencies in camelids can be due to selenium deficient (some geographic areas have low soil selenium) or Vitamin E deficient (mature or inadequately stored) forage. Vitamin E is a fat soluble nutrient that cannot be stored in the liver, so daily ingestion is required. With very severe deficiency, cardiac and skeletal muscle degeneration and necrosis develop; mineralization may be present in areas of necrosis. Heavily used muscles such as the intercostals, diaphragm, and tongue are commonly affected. Cardiac necrosis can lead to sudden death; important differentials for sudden cardiac death due to cardiomyonecrosis are oleander or ionophore toxicity, both of which are relatively common in captive camelids. It is likely that other plants throughout the natural ranges of camelids and ornamental plants used in zoos have similar cardiotoxic effects. In addition to feed and tissue level testing for selenium and Vitamin E, toxicologic testing may be required to rule out toxins when myocardial necrosis is seen.

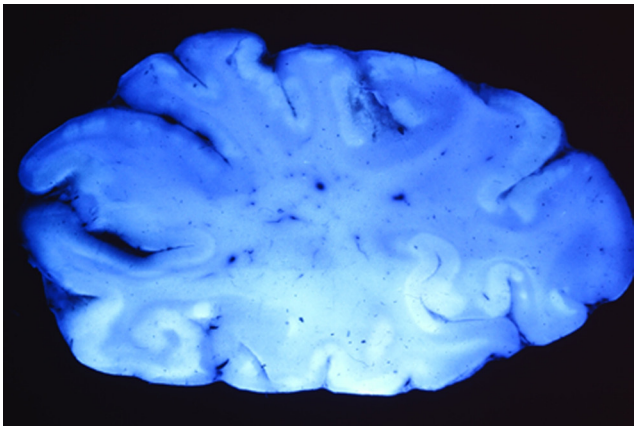

FIGURE 7.6 **Polioencephalomalacia in a llama**. Necrotic gray matter autoflouresces with ultraviolet light. In this image, polioencephalomalacia can be seen as bright white fluorescent areas in the cortical gray matter.

FIGURE 7.7 **Hepatic lipidosis in a llama**. Hepatic lipidosis is characterized by yellow to light tan, locally extensive to diffuse discoloration of the liver, a greasy friable texture, and rounded lobe margins. It is common in NW camelids, particularly in pregnant and lactating camelids when dietary carbohydrates are inadequate. *(Photo Courtesy of A. Tibary, Washington State University)*

Fat necrosis in camelids is associated with vitamin E and selenium deficiency, rapid weight loss, and some toxins (such as endophyte infected fescue), particularly in overweight animals with excessive intra-abdominal fat. In dromedary camels, hump fat necrosis is also anecdotally associated with vitamin E and selenium deficiency and causes histologic lesions similar to those seen in other species, including saponification and mineralization.

Goiter has been described in OW camelids in Africa and in the United States. Iodine deficiency has been incriminated in a fetus, neonate and in adults (Abu Damir et al., 1990; Decker et al., 1979). In adult animals, goiter has been characterized by enlarged thyroids with variable follicular dilation. Follicles are filled with copious colloid and lined by plump cuboidal and sometimes columnar epithelium. Clinically, disease has been associated with low serum thyroid hormones (T3, T4) as well as normocytic normochromic anemia and infertility.

Zinc responsive dermatosis, similar to that seen in domestic livestock, has also been reported in NW camelids (Fowler, 2010f; Rosychuk, 1994). Black NW adult camelids seem predisposed and lesions are typically noted on the face, ventral abdomen, lateral thorax and inguinal region. Pruritus is variable. Lesions consist of alopecia, dermal thickening, scaling and hyperpigmentation (Fig. 7.8A). Microscopically, variably thick parakeratotic hyperkeratosis and perivascular lymphoplasmacytic, eosinophilic, and histiocytic dermatitis are observed (Fig. 7.8B). Low plasma zinc levels (<0.5 mg/dl) may be associated with these lesions.

Congenital/Genetic

In North America, NW camelids have been selectively bred for many generations, often from a small founder population, leading to the presence of heritable defects. Teratogens and viral infections as well as nutritional deficiencies are likely additional contributing factors. Fowler has generated an extensive list of defects in NW camelids that have been reported in the literature or have been personally observed or communicated (Fowler, 2010a). Of these, the most common are angular limb deformities, polydactyly, hemivertebra, and cranial dysgenesis. Congenital deafness associated with pure white haircoat and blue iris pigmentation is seen in alpacas. Diagnosis of this condition is usually confirmed by brainstem auditory-evoked potential assessment (Gauly et al., 2005).

Due to the obligate nasal breathing of crias, **choanal atresia**, characterized by the presence of a membranous or bony partition between the nasal cavity and the pharynx, is an often lethal defect (Fowler, 2010a; Gerros and Stone, 1994). In some cases, atresia may be incomplete and lead to partial respiratory obstruction. Choanal atresia is often coupled with other facial and skull deformities and appears highly heritable. Cleft palate in NW camelids does not appear to be associated with choanal atresia, but is typically also a lethal defect.

Reproductive tract defects are also common in NW camelids. In a Peruvian study, 10% of the animals studied had one or more abnormalities (Fowler, 2010a; Sumar, 1989). Defects include uterine fistulae or agenesis/hypogenesis of the uterus or vagina (with secondary mucometra and dilation), intersexes, cryptorchidism, penile hypoplasia or malformation, retained penile frenulum, and testicular cysts.

In contrast, **fewer congenital defects have been reported in OW** camels, possibly due to less intensive breeding programs or under-reporting. Reported conditions include ophthalmic defects, ventricular septal defects, polydactyly, cleft palate, atresia ani, hernia, and growth retardation (Bani-Ismail et al., 1999; Moore et al., 1999; Sakamoto et al., 2004). In a survey of abattoir samples from 850 female dromedary camels in Nigeria, only 0.12% had cervical hypoplasia or any other congenital reproductive abnormality (Ribadu et al., 1991).

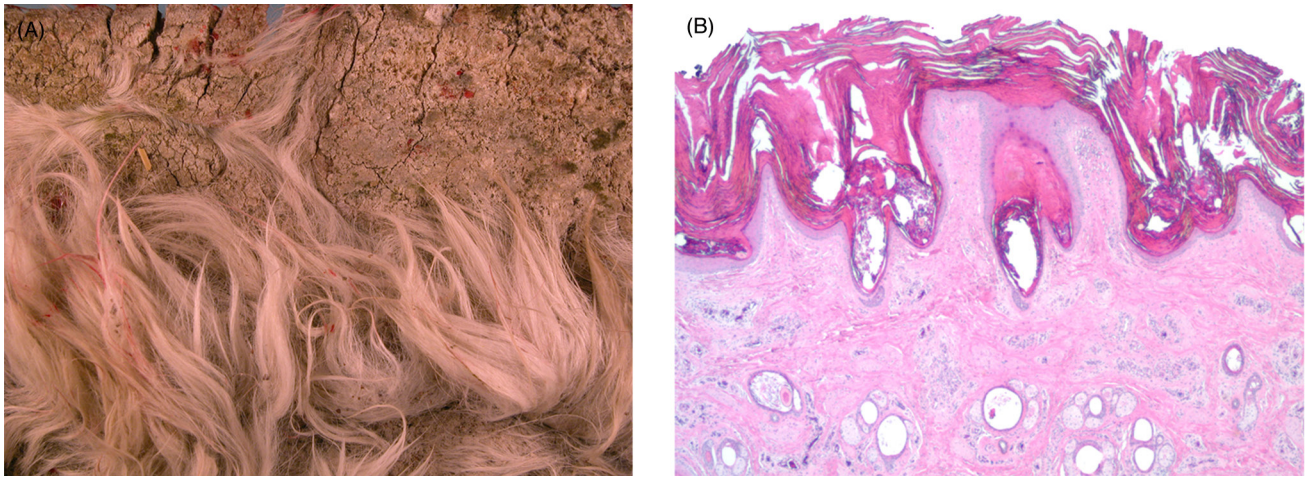

FIGURE 7.8 Zinc responsive dermatosis in a llama. (A) This condition is seen in in NW camelids on the face, ventral abdomen, lateral thorax, and inguinal region. It is characterized by alopecia, dermal thickening, scaling, crusting, and variable hyperpigmentation. (B) Markedly thickened parakeratotic hyperkeratosis, abundant keratin accumulation, and hair follicle distention are common features. Diffuse perivascular lymphoplasmacytic, eosinophilic, and histiocytic dermatitis are also often present.

Age-related/Degenerative

Osteoarthritis and degenerative joint disease is common in NW and OW camelids, though no diseases unique to these species have been reported. In older animals, overextension of the metacarpophalangeal or metatarsophalangeal joint (also called **dropped fetlocks**) has been well-described. Copper deficiency in conjunction with zinc excess rather than degeneration or trauma is a potential etiology (Reed et al., 2007). It is likely that in other animals, angular limb deformities and poor conformation, possibly coupled with obesity and lack of exercise, lead to degenerative joint diseases such as carpal and tarsal disease commonly reported later in life.

Ulceration and secondary infection of the sternal callosity (Fig. 7.9) occurs in all species of camelid in captivity and is probably related to lameness and prolonged recumbency in older animals. These lesions are characterized by chronic fibrosis, neovascularization, and granulation tissue, as well as pockets of suppurative inflammation. In OW camels, a bony protuberance underlies this callosity and it can become chronically inflamed, infected, and undergo bony remodeling and irregular proliferation secondary to extension of the overlying inflammatory process.

Abnormal **tooth wear** is another commonly observed age related change, particularly in NW camelids. Congenitally deformed dental structures can lead to elongated teeth, uneven wear, broken teeth, and laceration of the oral mucosa; this can occur even at a young age. Heredity is likely involved in many **deformities of the oral cavity** as it is in other mammalian species. Clinical monitoring and management is critical because emaciation and chronic bacteremia can result from impaired mastication.

FIGURE 7.9 Ulceration and secondary infection of the sternal callosity of a dromedary camel. The sternal callosity (or pedestal) is ulcerated and secondarily infected. It contains a large central cavitation with hemorrhage, and is covered by crusted, serocellular debris. In older NW and OW camelids this condition is due to prolonged recumbency or poor substrates. In OW camels, a bony protruberance underlies this callosity and extension of inflammation can lead to osteomyelitis.

Inflammatory Non-infectious

Autoimmune thyroiditis has been reported in dromedary camels in association with hypothyroidism and thyroid hormone autoantibodies (Al-Qarawi, 2005; Al-Qarawi et al., 2001). It has been associated with *Trypanosoma evansi* infection, a possible predisposing factor, as well as lowered fertility in the male. Further investigation into this condition is needed to better elucidate the mechanisms involved and the associated pathology.

Ovarian hydrobursitis is a unique condition described in dromedary camels. It is reported in up to 15% of females at slaughter, and in up to 33% of infertile camels (Ali et al., 2011; Tibary and Anouassi, 2001). The disease may be unilateral or bilateral and is most often seen on the left ovary though studies vary in the specific percentages of unilateral versus bilateral lesions. Grossly, the bursa is usually mildly to markedly distended by hemorrhagic fluid (Fig. 7.10). Histologically, there is degeneration and hyperplasia of the bursal epithelial lining; infiltration of the lining by variably intense aggregates of lymphocytes, plasma cells, and macrophages; cysts within the epithelial lining; multiple variably expansive regions of hemorrhage; and intralesional hemosiderin-laden macrophages. The high estrogen content of the bursal fluid in this condition suggests it is a mixture of hemorrhage and follicular fluid (the estrogen content of the distended bursa tends to be much higher than serum). While a specific cause has not been proven, over 86% of camels with hydrobursitis have serological evidence of *Chlamydia abortus* infection, suggesting this organism may be involved in the pathogenesis (Ali et al., 2012). Oxytetracycline, an anti-chlamydial drug, returns some of the fertility to affected animals, further supporting the theory that this is, at least in part, an infectious disease syndrome.

Miscellaneous

While OW camels are very resistant to hyperthermia, NW camelids, perhaps because of their evolutionary development at high altitudes and cool temperatures, are more susceptible. A history of high environmental temperatures, high humidity, physical activity, social stress, dehydration, and/or fever in a heavily fleeced down, moribund or dead NW camelid is highly suggestive of hyperthermia or heat stress. Tissues will rapidly autolyze under these conditions, so prompt post mortem inspection is critical to make a diagnosis. Lesions suggestive of hyperthermia include petechial hemorrhages, hyperemia, thrombosis, pulmonary edema and congestion, neuronal necrosis, hepatic necrosis, and renal tubular necrosis (Fowler, 2010g). Hypothermia can also be a concern in NW and OW camelids kept in northern climates where frostbite can occur; it primarily affects the ears. Some NW camelids have congenitally shortened ears

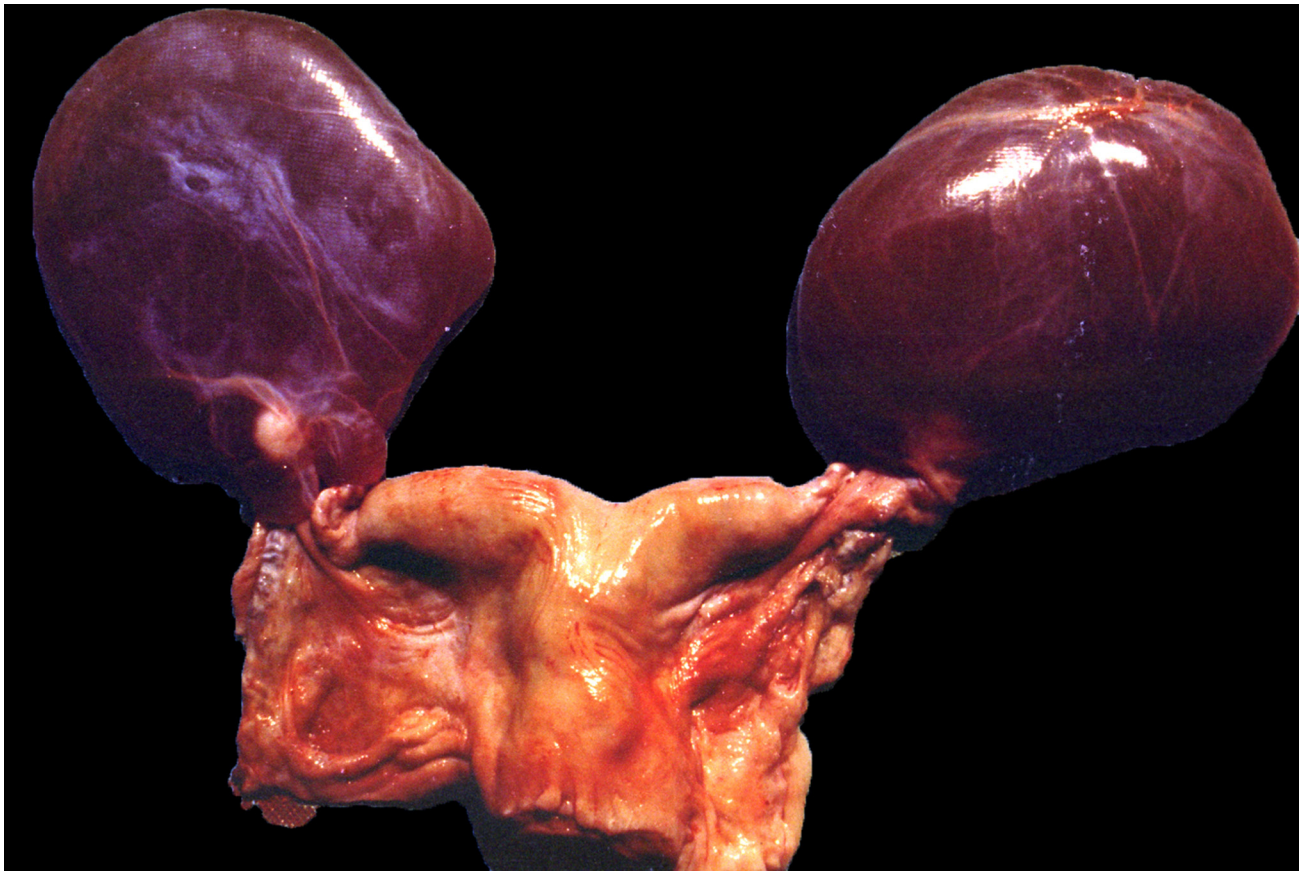

FIGURE 7.10 Ovarian hydrobursitis in a dromedary camel. This is a unique condition seen exclusively in dromedary camels. The ovarian bursa is mildly to markedly distended and filled with a mixture of follicular fluid and hemorrhage. The condition may be unilateral or bilateral. *(Photo Courtesy of A. Tibary, Washington State University)*

("gopher ear") that can be distinguished from healed frostbite by the presence of fibrosis and alopecia at the squared margin of the latter, distinct from the haired and tapered tip of the former (Fowler, 2010g).

Camelids can develop many of the same non-infectious skin diseases that are seen in other livestock.Two relatively unique conditions occur in NW camelids: **idiopathic superficial necrolytic dermatitis** and **zinc responsive dermatosis** (see above). Idiopathic superficial necrolytic dermatosis is characterized by non-pruritic, waxing and waning erythematous and crusting vesicles that are typically present in the axilla, inguinal area, ventral abdomen, distal extremities, perineum, and on the face. Microabscesses and neutrophilic infiltration of the superficial epidermis with ballooning degeneration are associated with orthokeratotic and parakeratotic hyperkeratosis. These lesions sometimes form the characteristic laminar distribution of "red, white, and blue" lesions (superficial parakeratosis ["red"] overlying keratinocyte vacuolation and spongiosus ["white"], and basilar epidermal cell hyperplasia ["blue"] similar to those seen in other species. An etiology has not been determined, but it may be multifactorial (Fowler, 2010f; Rosychuk, 1994).

Ulceration of the camelid stomachs (C-1 through C-3) is a common lesion that can be a primary disease process or develop in association with other disease states (Fowler, 2010g). In C-1 and C-2, ulcerations are typically noted along the margins of saccules primarily in the glandular regions (Fig. 7.11). In C-3, ulcers are typically linear along the longitudinal pleats in the proximal 4/5th, but are focal and deeper in the last 1/5th. Expansive ulcers may occur at the margin of compartment C-2 and C-3. These are most often seen in neonates with hypothermia, sepsis, or during periods of social or metabolic stress. Ulcers in the distal part of C-3 are the most likely to perforate, leading to peritonitis and rapid death. Underlying causes of ulcer development is poorly understood and likely multifactorial. Social and environmental stresses and associated catecholamine secretion has been suggested. Hypercortisolism is associated with acid hypersecretion in the gastric lumen of other species and is presumed to also be the case in camelids. Duodenal reflux and gastric vasoconstriction may also be factors in stressed animals. There is no acid secretion in C-1, however, so ulceration in this area cannot be explained by this mechanism. Other lesions that might be associated

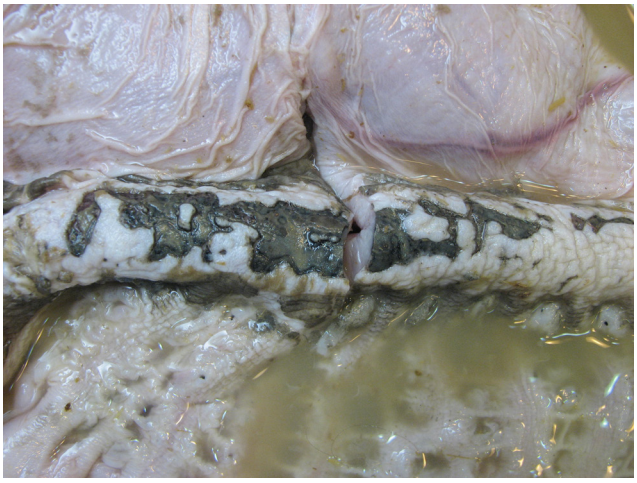

FIGURE 7.11 Gastric ulceration in a llama. Multifocal to coalescing, variably and irregularly shaped, sharply demarcated ulcers with cavitation and gray/black discoloration are present along pillars and at the margin between saccules in the glandular regions. Compartments C-1 and C-2 are most commonly affected.

with stress-induced gastric ulceration are lymphoid atrophy and adrenal cortical hyperplasia, the latter used to build a case for stress induced disease when ulceration is noted at postmortem examination.

Neoplastic

Neoplasia is not common in camelids. Relatively few reports exist in the literature, though one review of cases in a diagnostic laboratory puts the prevalence in llamas at 11% and in alpacas at 4.9% (Fowler, 2010g; Valentine and Martin, 2007). It is unlikely that this is due to decreased reporting in these species, as there is considerable veterinary and abbatoir attention to camelids.

Of the reported cancers, the most common is **lymphosarcoma** (Cebra et al., 1995; Irwin, 2001; Rosa and Rissi, 2013; Sartin et al., 2004; Shapiro et al., 2005; Simmons et al., 2005; Underwood and Bell, 1993). Immunohistochemistry using antibodies against CD3 (for T-cells), PAX-5, BLA36, CD79α (for B-cells), CD18, and synaptophysin are useful in differentiating lymphoid neoplasms in alpacas and llamas (Aboellail, 2013; Martin et al., 2009). Lymphomas that do not stain with either T- or B-cell markers may be non-B, non-T-cell lymphomas or primitive malignant round cell tumors (PMRCT) (Aboellail, 2013; Martin et al., 2009). Interestingly, 3 cases (one llama and two alpaca) were reported from Canada in animals 6-7 months of age; other reports also describe young camelids (especially alpacas) with disseminated lymphosarcoma (Aboellail, 2013; Irwin, 2001; Sartin et al., 2004). With the exception of bovine leukemia virus induced lymphosarcoma, confirmed by *in situ* hybridization and serology in a 13-month old alpaca (Lee et al., 2012), no underlying etiologies or predisposing factors have been confirmed in lymphosarcoma (or any other cancer) of camelids. No trend towards any particular type of lymphosarcoma has been confirmed in camelids.

Mucocutaneous fibropapillomas are common in NW camelids. One study demonstrated papillomavirus in multiple mucocutaneous fibropapillomas (Schulman et al., 2003). Efforts have been made to look for viruses in other tumors via electron microscopy without success.

Other cancers that have been reported in camelids are listed in Table 7.1.

INFECTIOUS DISEASES

A number of viral, bacterial, fungal, and parasitic diseases have been described in NW and OW camelids. Prion diseases have not been diagnosed in camelids, and camelids are considered low risk for bovine spongiform encephalopathy (BSE), though no studies have looked at transmission and only one has examined prion protein gene polymorphisms in camels (Xu et al., 2012).

DNA Viruses

Equine Herpesvirus (EHV-1), a DNA virus in the *Herpesvirus* family, has been frequently reported in NW and OW camels, and has been experimentally reproduced in llamas from an alpaca isolate (House et al., 1991). Infection in camelids likely develops as a "spill-over" from infected co-housed equids. In camelids, clinical signs are typical of equine neurological herpesviral infection. Edema, neuronal necrosis, and perivascular and meningeal lymphocytic inflammation are usually present (House et al., 1991). Eosinophilic intranuclear inclusion bodies are typically present in endothelium and neurons. Clinical neurologic signs include blindness, which appears particularly common. Histologic lesions include necrotizing retinitis with retinal detachment, hemorrhage, choroiditis, vitritis, and optic neuritis characterized by edema and gitter cell infiltration (Rebhun et al., 1988). Abortion has not been reported in camelids due to EHV-1. Vaccination has been proposed but there are no published studies on the effectiveness in prevention of infection or disease.

Camelpox is caused by Camelpox virus, a DNA virus in the *Poxviridae* family and *Orthopoxvirus* genus. It is an OIE listed reportable disease and is the most common viral disease in dromedary and Bactrian camels. It is zoonotic and capable of infecting immunocompetent humans, though it is more likely to cause severe disease in immunocompromised individuals. With electron microscopy this virus appears as brick-shaped, enveloped particles with rounded edges measuring 360 µm x 270 µm x 250 µm. Parapoxviruses can be differentiated by their more ovoid shape than camelpox. Camelpox has been experimentally reproduced in NW camelids, though it is not present in the Americas or Australia.

TABLE 7.1 Reported Camelidae Neoplasia

Neoplasm	Species of camelid
Epithelial tumors	
Ameloblastoma[r]	Llama
Basal cell carcinoma[a]	Dromedary camel
Cholangiocarcinoma[ii,kk]	Llama, Alpaca
Cutaneous squamous cell carcinoma[cc,kk]	Llama, Alpaca
Gastrointestinal adenocarcinoma[v,kk]	Llama, alpaca, Bactrian camel
Gastrointestinal squamous cell carcinoma[g,dd,kk]	Llama, Alpaca
Mammary carcinoma[d,e,kk]	Llama, Alpaca, Dromedary camel
Nephroblastoma[h]	Guanaco
Ocular meduloepithelioma (non-teratoid)[ee]	Llama
Pulmonary carcinoma[e,bb]	Llama, Dromedary camel
Renal cell carcinoma[ll]	Dromedary camel
Retinoblastoma[i]	Llama
Trichoepithelioma (multiple)[hh]	Alpaca
Uterine adenocarcinoma[u]	Llama
Mesenchymal neoplasms	
Ameloblastic odontoma[gg]	Llama
Astrocytoma[j]	Llama
Chondrosarcoma[s]	Dromedary camel
Embryonal rhabdomyosarcoma[l]	Alpaca
Fibromas/Fibropapillomas[ff,kk]	Llama, Alpaca
Fibrosarcoma[o,kk]	Llama, Alpaca
Hemangiosarcoma[m]	Llama
Lipoma[kk]	Llama
Meningioma[z]	Bactrian camel
Ossifying fibroma[x]	Llama
Osteosarcoma[jj]	Dromedary camel
Peripheral nerve sheath tumor[t]	Dromedary camel
Rhabdomyosarcoma[nn]	Dromedary camel
Uterine leiomyosarcoma[n]	Alpaca
Round cell and other neoplasms	
Granulosa cell tumor[c]	Llama
Histiocytic sarcoma[z]	Bactrian camel
Interstitial cell tumor (ovary)[kk]	Alpaca
Malignant melanoma[aa]	Alpaca
Mast cell tumor[w]	Llama
Melanocytoma[kk]	Llama
Mesothelioma[mm]	Dromedary camel
Neuroendocrine tumor[dd]	Alpaca
Ocular melanoma[p]	Alpaca
Pituitary adenoma[f,k]	Llama; Alpaca
Seminoma[b]	Dromedary camel
Teratoma[q,y]	Alpaca, Dromedary camel

(*Continued*)

TABLE 7.1 Reported Camelidae Neoplasia (*Cont.*)

[a]Al-Hizab, F.A., Ramadan, R.O. Al-Mubarak, A.I., Adbelsalam, E.B., 2007. Basal cell carcinoma in a one-humped camel Camelus dromedarius – a case report. Journal of Camel Practice and Research 14, 49-50.
[b]Ali, A., Ahmed, A.F., Mehana, E.E., El-Tookhy, O., Al-Hawas, A., 2013. Unilateral seminoma in a dromedary camel. Reproduction in Domestic Animals 48, 17-9.
[c]Anderson, D.E., Couto, C.G., Oglesbee, M., 2010. Granulosa theca cell tumor with erythrocytosis in a llama. Canadian Veterinary Journal 51, 1157-60.
[d]Bangari, D.S., Stevenson, G.W., 2007. Carcinoma in a mixed mammary tumor in a llama Llama glama. Journal of Veterinary Diagnostic Investigation 194, 450-453.
[e]Bryant, B., Portas, T., Montali, R.. 2007. Mammary and pulmonary carcinoma in a dromedary camel (Camelus dromedarius). Australian Veterinary Journal 85, 59-61.
[f]Chalkley, M.D., Kiupel, M., Draper, A.C., 2014. Pituitary null cell adenoma in a domestic llama (Lama glama). Journal of Comparative Pathology 151, 51-6.
[g]Cornick, J.L., 1988. Gastric squamous cell carcinoma and fascioliasis in a llama. Cornell Veterinarian 78, 235-241.
[h]Dietrich, J.G., Brewer, J.L., 1988. Nephroblastoma with pulmonary metastases in a guanaco. Veterinary Pathology 25, 329-330.
[i]Fugaro, M.N., Kiupel, M., Montiani-Ferreira, F., Hawkins, J.F., Janovitz, E.B., 2005. Retinoblastoma in the eye of a llama (Llama glama). Veterinary Ophthalmology 8, 287-90.
[j]Garlick, D.S., Doherty, T.J., Paradis, M.R., 1990. Gemistocytic astrocytoma in a one-month-old llama.
[k]Gilsenan, W.F., Habecker, P.L., Coyne,T.M., Johnson, A.L., 2012. Neurologic disease attributed to a pituitary adenoma in an alpaca. Journal of Veterinary Internal Medicine 26, 1073-7.
[l]Goncarovs-Gran, K.O., Frank, C.B., Baird, A.N,, Couetil, L.L., Ramos-Vara, J.A.. 2013. Pathology in practice. Embryonal rhabdomyosarcoma in an alpaca. Journal of the American Veterinary Medical Association 243, 1113-5.
[m]Hamir, A.N., Pierce, V., Richardson, D., 1997. Intraosseous hemangiosarcoma with metastasis in a three-month-old llama. Journal of Veterinary Diagnostic Investigation 9, 210-3.
[n]Hardefeldt, L.Y., Poulsen, K.P., McGuirk, S.M., Livesey, M.A., Koch, C., Perrier, M.P., Pinkerton, M.E., 2010. Urogenital leiomyosarcoma in an alpaca. Canadian Veterinary Journal 51, 1387-90.
[o]Higginbotham, M.L., McCaw, D.L., Anderson, D.E., Lattimer, J.C., Armbrust, L., Andrews, G.A., McBride, B.D., 2015. Treatment of a maxillary fibrosarcoma in an adult alpaca. Journal of the American Veterinary Medical Association 246, 674-80.
[p]Hill, F.I., Hughes, S.M., 2009. Osteogenic intraocular melanoma in an alpaca (Vicugna pacos). Journal of Veterinary Diagnostic Investigation 21, 171-3.
[q]Hill, F.I., Mirams, C.H., 2008. Intracranial teratoma in an alpaca (Vicugna pacos) in New Zealand. Veterinary Record 162, 188-9.
[r]Ivester, K.M., Baird, A.N., Baird, D.K., Oertley-Pihera, K., 2006. Diagnostic imaging in veterinary dental practice. Ameloblastoma. Journal of the American Veterinary Medical Association 228, 1343-4.
[s]Janardhan, K.S., Ganta, C.K., Andrews, G.A., Anderson, D.E., 2011. Chondrosarcoma in a dromedary camel (Camelus dromedarius). Journal of Veterinary Diagnostic Investigation 23, 619-22.
[t]Khodakaram-Tafti, A., Khordadmehr, M., 2011. Multicentric fibromyxoid peripheral nerve sheath tumor (multicentric schwannoma) in a dromedary camel (Camelus dromedarius): morphopathological, immunohistochemical, and electron microscopic studies. Veterinary Pathology 48, 1180-4.
[u]Klopfleisch, R., van der Grinten, E., Gruber, A.D., 2009. Metastatic uterine adenocarcinoma and hepatic lipomatosis in a llama (Lama glama). Journal of Veterinary Diagnostic Investigation 21, 280-2.
[v]Kühn, N., Gröne, A., Pagan, O., Bacciarini, L.N., 2003. Metastatic gastric adenocarcinoma and diffuse hyperplastic gastritis resembling human Menétrier's disease in a camel (Camelus ferus bactrianus). Journal of Veterinary Medicine, A, Physiology, Pathology, and Clinical Medicine 50, 359-62.
[w]Lin, T.Y., Hamberg, A., Pentecost, R., Wellman, M., Stromberg, P., 2010. Mast cell tumors in a llama (Lama glama). Journal of Veterinary Diagnostic Investigation 22, 808-11.
[x]McCauley, C.T., Campbell, G.A., Cummings, C.A., Drost, W.T., 2000. Ossifying fibroma in a llama. Journal of Veterinary Diagnostic Investigation 12, 473-6.
[y]Mesbah, S.F., Kafi, I.M., Nili, H.. 2002. Ovarian teratoma in a camel (Camelus dromedarius). Veterinary Record 151, 776.
[z]Molenaar, F.M., Breed, A.C., Flach, E.J., McCandlish, I.A., Pocknell, A.M., Strike, T., Routh, A., Taema, M., Summers, B.A., 2009. Brain tumours in two Bactrian camels: a histiocytic sarcoma and a meningioma. Veterinary Record 164, 684-8.
[aa]Mollat, W.H., Gailbreath, K.L., Orbell, G.M., 2009. Metastatic malignant melanoma in an alpaca (Vicugna pacos). Journal of Veterinary Diagnostic Investigation 21, 141-4.
[bb]Ramos-Vara, J.A., Loiacono, C.M., Williams, F. 3rd, Pardo, I., Lakritz, J., 2004. Pulmonary neoplasia in two llamas (Lama glama). Veterinary Pathology 41, 520-3.
[cc]Rogers, K., Barrington, G.W., Parish, S.M., 1997. Squamous cell carcinoma originating from a cutaneous scar in a llama. Canadian Veterinary Journal 38, 643-644.
[dd]Sartin, E.A., Crowe, D.R., Whitley, E.M., Treat, R.E., Purdy, S.R., Belknap, E.B., 2004. Malignant neoplasia in four alpacas. Journal of Veterinary Diagnostic Investigation 16, 226-229.
[ee]Schoeniger, S., Donner, L.R., Van Alstine, W.G., 2006. Malignant nonteratoid ocular medulloepithelioma in a llama (Llama glama). Journal of Veterinary Diagnostic Investigation 18, 499-503.
[ff]Schulman, F.Y., Krafft, A.E., Janczewski, T., Reupert, R., Jackson, K., Garner, M.M., 2003. Camelid mucoutaneous fibropapillomas: clinicopathologic findings and association with papillomavirus. Veterinary Pathology 40, 103-107.
[gg]Step, D.L., Ritchey, J.W., Drost, W.T., Bahr, R.J., 2003. Ameloblastic odontoma in the mandible of a llama. Canadian Veterinary Journal 44, 824-827.
[hh]Suedmeyer, W.K., Williams, F. 3rd., 2005. Multiple trichoepitheliomas in an alpaca (Lama pacos). Journal of Zoo and Wildlife Medicine 36, 706-8.
[ii]Taulescu, M.A., Bolfa, P.F., Buiga, R., Gal, A.F., Sevastre, B., Morar, I., Catoi, C., 2012. Metastatic cholangiocarcinoma in a llama (Lama glama). Journal of Veterinary Diagnostic Investigation 24, 986-9.
[jj]Tuttle, A.D., Frederico, L., Linder, K., Gunkel, C., Remick, A., Redding, R., 2007. Pathological fracture of the ulna due to osteosarcoma in an Arabian camel (Camelus dromedarius). Veterinary Record 161, 30-3.
[kk]Valentine, B.A., Martin, J.M., 2007. Prevalence of neoplasia in llamas and alpacas (Oregon State University, 2001-2006). Journal of Veterinary Diagnostic Investigation 19, 202-204.
[ll]Vítovec, J.,1982. Renal cell carcinoma in a camel (Camelus dromedarius). Veterinary Pathology 19, 331-3.
[mm]White, J.,2004. Pericardial mesothelioma in an Arabian camel (Camelus dromedarius). In: Proceedings of the American Association of Zoo Veterinarians Annual Meeting, San Diego, CA.
[nn]Zakia-Mohammed, A., Ramadan, R.O., Al-Mubarak, A. I., 2007. Rhabdomyosarcoma in a she-camel. Journal of Camel Practice and Research 14, 165-167.

Transmission is usually due to direct exposure through skin abrasions or inhalation of infected secretions from lesions, milk, saliva, blood, ocular or nasal secretions, or urine. Arthropods, including flies and ticks, are suspected vectors. Upon entry, the virus replicates locally producing a primary erythematous lesion. The infection then spreads to local lymph nodes and a leukocyte-associated viremia develops (Balamurugan et al., 2013). During this time virus can be identified in skin, turbinates, lungs, and lymphoid organs. A few days after viremia begins, widespread secondary skin lesions (typical pox) appear and persist for 2–3 days until viremia subsides. Like other poxviral diseases,

FIGURE 7.12 Camel pox in a dromedary camel. Camel pox lesions begin with erythematous macules that develop into papules and vesicles and progress to pustules that rupture and become covered by crusts. Lesions often affect the head, nostrils, ear margins, and eyelids. Camel pox affects the mucous membranes, including the lips, and oral and nasal cavities, making it difficult for animals to eat or nurse. *(Photo Courtesy of A. Tibary, Washington State University)*

camelpox virus encodes multiple genes that modulate the host immune response by interfering with interferon (IFN), pro-inflammatory cytokines [(Interleukin-IL-1b, IL-18 and tumor necrosis factors (TNFs)], chemokines, and complement (Duraffour et al., 2011).

Severity of disease is dependent of viral strain, the immune status of the infected host, and the age of the host (younger animals are much more susceptible). Typical pox lesions begin as erythematous macules that develop into papules and vesicles, and progress to pustules that rupture leading to crust formation. Lesions typically affect the head, nostrils, ear margins and eyelids (Fig. 7.12). Mucous membranes, including the lips, and oral and nasal cavities, can be affected and infection may also spread to other parts of the body including the genitalia and mammary gland. Healing can take up to 4–6 weeks and can leave scars or cause multifocal alopecia (Balamurugan et al., 2013). Lesions are most commonly found in the skin, but a disseminated form may also occur in young animals stressed by weaning or poor husbandry. In this form, similar crusting lesions may spread more widely, particularly affecting the head and the limbs but also the neck and abdomen. In the most severe cases, proliferative necrotizing lesions in the oral cavity or respiratory or gastrointestinal tracts may lead to death. These animals demonstrate hypersalivation, anorexia, mucopurulent nasal and ocular discharge, and diarrhea. Abortion can also occur. Death is often due to secondary bacterial infection and subsequent septicemia.

Histopathology is typical for poxviral lesions: cytoplasmic swelling and ballooning vacuolation of keratinocytes in the stratum spinosum of the epidermis with subsequent rupture and vesicle formation, lymphoplasmacytic perivascular cuffing and infiltration of neutrophils and eosinophils in the dermis, and prominent epithelial hyperplasia at the border of the epidermal lesions. It is notable that, unlike many other poxviral lesions, inclusions are not a diagnostic feature of poxvirus infection in camels. Lung lesions are characterized by bronchial epithelial cell proliferation with vacuolation, proliferative alveolitis and bronchiolitis, necrosis, and in older lesions, fibrosis. Immunohistochemistry, *in situ* hybridization, and PCR can all be used to confirm the diagnosis, and transmission electron microscopy (TEM) on scabs or tissue can be used to differentiate the brick-shape of the orthopoxvirus from clinically relevant differentials, importantly contagious ecthyma ("Orf;" a parapoxvirus), papillomatosis (papillomaviruses), and insect bites. After infection, immunity to camelpox is life-long.

Parapoxvirus infection (also called **contagious ecthyma**, **contagious pustular dermatitis**, or "**Orf"**), caused by a double stranded DNA virus in the family *Parapoxviridae*, is well-recognized in both OW and NW camelids. The epidemiology, pathogenesis, and lesions are very similar to those seen in small domestic ruminants. Typical lesions are characterized by marked epidermal proliferation with superficial necrosis that is most common on the oral commissures and lips of crias and calves, and on the teats and perineum of dams. Microscopically, there is swelling and vacuolation of keratinocytes, epidermal proliferation, suppurative inflammation, and a marked accumulation of crusts. Eosinophilic intracytoplasmic inclusions appear by 2-3 days post-infection and persist for 3-4 days. Lesions in camelids tend to persist longer than those seen in goats and sheep. Transmission is likely associated with shared housing with small ruminants. This is a zoonotic disease, and there are reports of humans also infecting llamas (anthropozoonotic) (Fowler, 2010e).

Papillomatosis, caused by a DNA virus in the family *Papillomaviridae*, is relatively common in OW camels. In dromedary camels it is associated with two identified papillomaviruses, *Camelus dromedarius* papillomavirus types 1 (CdPV1) and 2 (CdPV2) (Ure et al., 2011). Lesions present as large, round, cauliflower-like, pedunculated masses that in their mature stage are unlikely to be confused with other lesions but in early stages may be confused with parapox or camelpox lesions. Fibropapillomas in NW camelids (both alpaca and llamas) are associated with a unique papillomavirus that shares only 73% homology to bovine papillomavirus (Schulman et al., 2003). These lesions are similar to equine sarcoids, with a fibroblastic proliferation in the dermis, hyperplasia of the overlying epithelium, and prominent tendrils of rete ridges. Inclusions are not seen.

RNA Viruses

Bovine virus diarrhea virus (BVDV) is an RNA virus in the *Flaviviridae* family and *Pestivirus* genus. Two BVDV

genotypes, BVDV-1 and BVDV-2, each with multiple subtypes, are recognized. Many ruminant species, including OW and NW camelids, can be infected. Clinical signs include immunosuppressive and respiratory and reproductive tract diseases. *In utero* infection can lead to the birth of persistently infected offspring. In NW camelids, *in utero* infection with subsequent abortion or birth of persistently infected (PI) offspring are the most commonly described outcomes. Infection in alpaca has been associated with abortion and BVDV positive fetal tissues, or chronically ill, stunted, and immunosuppressed crias that may be seronegative, but positive for BVDV by PCR or virus isolation (Carman et al., 2005; Mattson et al., 2006; Nelson et al., 2015). Other outcomes include stillbirth, neonatal death, and congenital deformities; chronic respiratory or gastrointestinal disease have also been reported. Disease has not been described in OW camelids, but both serological response and molecular presence of BVDV-1 in both Bactrian and dromedary camels suggest exposure (Gao et al., 2013; Intisar et al., 2010). A new substrain of BVDV (BVDV-1q) was first identified in Bactrian camels and later identified in dairy cattle and swine in China (Gao et al., 2013).

Pathogenesis has been investigated in experimental infections. An experimental BVDV-1 infection study in alpacas less than 2 years of age showed lymphocytopenia and a decrease in peripheral lymphocytes (Steffen et al., 2014). Gastrointestinal associated lymphoid tissue (GALT) B-cells were depleted, while parafollicular and submucosal T-cells remained. Viral antigen was identified by immunohistochemistry in GALT of ileum, proximal colon and C-3; it was also variably present in other lymphoid tissues. Histologic depletion of lymphoid tissues correlated with the presence of antigen. Viremia was present in all infected alpacas by day 5 and as early as day 3 post-infection. Nasal shedding was present in all infected alpacas. None of these experimentally infected alpacas were clinically ill.

Antigen distribution has been reported in persistently infected PI alpacas and is widespread being prominent in neurons, endothelial cells, and vascular tunica media myocytes, less in epithelium of multiple organs, and still less in lymphoid tissues (Henningson et al., 2012). Macrophages were also strongly labeled in the intestinal submucosa and medullary sinuses of lymph nodes. Antigen distribution was similar in reproductive organs to that seen in cattle, suggesting possible venereal transmission and impact on fertility. Unlike most bovids, antigen was only rarely found in the bone marrow.

Peste des petites ruminants (PPR) is a paramyxoviral disease of small ruminants (similar in pathogenesis and lesions to the now eradicated rinderpest). Common clinical signs include fever, dehydration, oral ulcers, diarrhea, lymphadenopathy, dermatitis, corneal ulceration, conjunctivitis, and respiratory distress. Pulmonary congestion, edema, and pneumonia; hepatic necrosis; renal tubular necrosis; and multifocal necrotizing dermatitis and mucosal ulcerations are common necropsy findings. PPR has been recently been reported in dromedary camels in the Middle East and East Africa (Khalafalla et al., 2010; Zakian et al., 2016).

Foot and mouth disease (FMD) (also called **Hoof and Mouth disease**) is caused by *Aphthovirus sp.* in the family *Picornaviridae*. There are seven types and over 60 subtypes. This is an OIE reportable disease that has major impacts on livestock trade worldwide. Susceptibility varies markedly between species of camelids, making the role of camelids in the epidemiology and regulation of this disease controversial. Dromedary camels are considered essentially resistant to FMD infection, with no confirmed reports of natural infection and no seroconversion even in the face of on-going outbreaks in closely associated cattle (Wernery et al., 2006). Bactrian camels, in contrast, are susceptible and may play a small but significant role in the epidemiology of the disease in Central Asia. NW camelids can contract the disease both naturally and experimentally, but they are significantly less susceptible than cattle and sheep and stop shedding 14 days post-infection; they do not remain carriers after infection (Wernery and Kinne, 2012a). Lesions are similar to those described in ruminants: vesicle formation followed by erosions and ulcers on the tongue, dental pads, oral cavity, palate, lips, nostril, coronary band, interdigital space with undermining of the footpads, teats, and the pillars of C-1 and C-2.

Vesicular and erosive lesions similar to those of FMD present in **vesicular stomatitis (VS),** a disease caused by an RNA virus in the *Rhabdoviridae* family (*Vesiculovirus* genus) that has a broader host range than FMD. This is an internationally recognized, reportable disease, but is not an OIE listed reportable disease. New World camelids likely play a very small to no role in the epidemiology of the disease. Only one natural case of VS been reported in the literature in NW camelids and none in OW camelids. Experimental infections require inoculation directly into the dorsum of the tongue and produce localized vesicles, fever, and recumbency (Fowler, 2010e).

Borna disease virus (*Bornaviridae* family, *Bornavirus* genus) affects multiple taxa. Infection and disease are often associated with horses and donkeys in central Europe, but it has also been identified in captive NW camelids (Altmann et al., 1976). The virus is likely transmitted by a tick, but oral transmission has also been demonstrated in horses. Infection in llamas and alpacas can cause anorexia and weight loss, loss of libido, and stretching convulsions (Jacobsen et al., 2010). No gross lesions are apparent, but histologically, there is non-suppurative encephalomyelitis and perivascular cuffing.

Eastern equine encephalomyelitis (EEE) and **West Nile virus (WNV)** infection, both caused by arboviruses (*Alphavirus* and *Flavivirus,* respectively), have been reported in NW camelids. Infection with either causes very similar

clinical signs and pathology. Fever, seizures, ataxia, lethargy, recumbancy, and a high mortality rate are characteristic; differentials include listeriosis, rabies, polioencephalomalacia, and meningeal nematodiasis. Gross lesions are inapparent. Histologic lesions of polioencephalitis include typical lymphocytic perivascular cuffs, neutrophil infiltration, gliosis, satellitosis, multifocal neuronal and glial necrosis, and edema. For EEE, immunohistochemistry and *in situ* hybridization will demonstrate intracytoplasmic neuronal and glial antigen and RNA (Nolen-Walston et al., 2007). Immunohistochemistry highlights abundant WNV in the neurofibers as well as in neuronal and glial cytoplasm (Dunkel et al., 2004).

In neonatal and young NW camelids, like young domestic ruminants, **rotavirus** (family *Reoviridae)* **and coronavirus** (family *Coronavirdae)* infections are likely very common. One North American serosurvey suggested up to 98% prevalence of rotavirus on some farms and another report suggested 42% of diarrhea in young NW camelids was due to coronavirus infection (Kapil et al., 2009). Like cattle, predisposition to disease is likely linked to failure of passive transfer of maternal antibodies. Clinical signs and gross and histologic lesions are also similar to those domestic cattle. Diarrhea in young OW camelids associated with both rotavirus and coronavirus have been reported as well; however gross and or histologic lesions have not been described. These are likely similar to those seen in cattle.

Middle Eastern Respiratory Syndrome (MERS; also called camel flu) is caused by **MERS coronavirus (MERS-CoV)** and is an OIE reportable disease. Clinical signs in humans vary from mild to severe and life-threatening. Dromedary camels likely play a role in transmission to humans, though how this occurs is currently unclear (Younan et al., 2012). Serosurveys suggest the virus has been circulating in camels since the 1980s, well before the disease was recognized in humans in 2012. The virus appears to infect and spread among young camels more readily than older camels, but there are no documented lesions in camels. The virus is significant for the substantial risk it poses to humans exposed to camels shedding the virus. Experimental infections of alpacas with MERS-CoV showed successful infection, replication, and transmission of the virus (Adney et al., 2016).

A **novel coronavirus** was detected by TEM and PCR as the cause of an outbreak of respiratory disease in alpacas in California in 2007 (Crossley et al., 2010). Disease was primarily seen in pregnant animals, but also identified in other demographics. Gross lesions included pulmonary congestion and edema and pleural effusion. Histologically, diffuse, acute bronchointerstitial pneumonia centered on terminal airways was characterized by fibrin deposition and hyaline membranes accompanied by variable epithelial necrosis and regeneration and mixed inflammation. Subsequent outbreaks have not been reported, but additional sequence analysis indicates the virus is closely related to the common human coronavirus (HCoV) 229E (Crossley et al., 2012).

Bacteria

Johne's disease is caused by ***Mycobacterium avium* ssp. *paratuberculosis***. Infection has been reported in alpacas, llamas, dromedary and Bactrian camels. Clinically, animals present with severe weight loss (often difficult to detect visually due to heavy fleece) and diarrhea. Gross and histologic lesions are similar to those cattle, with mild to marked expansion of the intestinal lamina propria and mesenteric and ileocecocolic lymph nodes by macrophages containing few to numerous acid fast organisms (Tharwat et al., 2012). In a group of alpacas infected in Australia, extensive granulomatous lesions extended to the lungs, liver, and peritoneal lymphatics (Ridge et al., 1995).

Tuberculosis (***Mycobacterium tuberculosis, M. bovis,*** and other members of the Mycobacterium tuberculosis complex) is an OIE listed reportable disease that is widely reported in OW camelids. Infection is less often documented in NW camelids, though cases have been reported in llamas co-housed with *M. bovis* infected cervids (Fowler, 2010e; Wernery and Kinne, 2012b). Lesions in camelids are most often associated with the respiratory tract and affect the lungs and associated lymph nodes, though disseminated disease has been reported. Inflammation is granulomatous with characteristic tubercles composed of a central core of caseous necrosis and mineralization surrounded by macrophages, lymphocytes, plasma cells, and neutrophils and a dense fibrous capsule. Unlike infection in cattle, giant cells are rarely present in OW camelids, but may be seen in NW camelids infected with *M. microti*. Lesions contain very rare, acid fast positive bacilli; some lesions in NW camelids have had more numerous intralesional organisms (Narnaware et al., 2015; Wernery and Kinne, 2012b). Antemortem diagnostics, as in many non-domestic species, is problematic and at this time, there is no definitive and reliable test.

Brucellosis, caused by ***Brucella* spp.**, a Gram-negative, facultatively intracellular coccobacillus, is common in OW camels and an OIE listed reportable disease (See also Chapter 5). Zoonotic transmission typically occurs via the consumption of unpasteurized milk. In dromedary camels, brucellosis is primarily caused by ***B. melitensis*** and rarely by ***B. abortus*** (Wernery, 2014). Brucellosis is less common in NW camelids though outbreaks in alpacas and zoonotic transmission have occurred. Abortion is the most common and significant clinical presentation, though this is less common in camels than in small ruminants and cattle. Placental retention is also uncommon in camels, likely due to the different structure of their placenta. Necrosis, suppurative inflammation, and fibrosis of the chorion in more chronic cases is typical. Organisms are often abundant and can be found in trophoblasts lining the chorion.

Reproductive infections are a very common source of infertility and poor production in both NW and OW camelids (Tibary et al., 2006). In NW camelids, ***Escherichia coli*** and ***Streptococcus equi* ssp. *zooepidemicus*** are common causes of endometritis. Infection is associated with endometrial ulceration, neutrophilic infiltrates, intraglandular aggregates of debris and pus, and in chronic cases, fibrosis, glandular atrophy and infertility. An endometrial biopsy scoring system similar to that used in horses has been developed and can provide valuable treatment and prognostic information (Powers et al., 1990). While abortion can be due to a variety of genetic, management, and nutritional factors, infectious diseases of all kinds have been estimated to cause from 10% to more than 70% of abortions in some areas (Tibary et al., 2006). **Leptospirosis**, **toxoplasmosis** and **chlamydiosis** have been frequently diagnosed in llamas and alpacas while in dromedary camels, **brucellosis** and **trypanosomiasis** are more common in the Middle East and Africa. Mastitis is rare in South American camelids but can be very common in dromedary and Bactrian camels. It is most often subclinical and chronic, with non-suppurative inflammation and fibrosis being the most common histologic lesions.

Alpaca fever (*la fiebre de las alpaca*), caused by ***Streptococcus equi* ssp. *zooepidemicus*,** is an important disease of alpacas that is associated with low morbidity and very high mortality (up to 100%) (Jones et al., 2009). It is seen primarily in Peru and South America but has also been reported in North America. Acute forms are usually seen in young animals that develop high fever, depression, and recumbency. Polyserositis is a significant common finding and systemic effects include fibrinous peritonitis and pleuritis, as well as fibrinosuppurative meningitis. Chronic abscesses are more common in adults in which there may be an environmental source of infection. Transmission is likely by direct contact or oral routes, and more severe forms are generally associated with an environmental, nutritional, or social stress (Fowler, 2010e; Jones et al., 2009). Similar disease has recently been described in OW camelids in North America and in abbatoirs in Africa, suggesting that this may be a more widespread disease among camelids than previously thought (Abubakar et al., 2010; Stoughton and Gold, 2015).

Along with coronavirus, rotavirus, and cryptosporidiosis, **colibacillosis** is a significant cause of morbidity and mortality in NW camelid neonates (and presumably OW camelids, as well, though this is less well documented) (Whitehead, 2009). **Escherichia coli** can lead to neonatal diarrhea and sepsis, is often associated with a failure of passive transfer of maternal antibodies, and occurs most commonly in animals less than 7 days of age. When combined with viral or other factors, older animals may be affected. Clinical signs and lesions are typical of those in domestic ruminants with the same disease, though enteropathogenic and enterotoxigenic strains have not been identified in camelids. Late in the disease, hemorrhagic enteritis may be present (Whitehead, 2009). Typical enteric lesions include piecemeal necrosis, increased neutrophil, lymphocyte, and plasma cell infiltration of the lamina propria, crypt abscesses, and variably adherent Gram-negative coccobacilli adhered to the surface of enterocytes.

Enterotoxemia due to ***Clostridium perfringens* type A** is an important disease of neonatal alpacas, particularly in Peru where there may be high mortality rates; it also affects young and adult OW camelids (Wernery et al., 1991). Racing camels appear more commonly affected than less intensively trained animals. Death often occurs at 2-3 weeks of age as colostral antibodies wane and before naturally acquired antibodies increase in circulation (Fowler, 2010e). Sudden death, bloating and constipation, or neurologic signs are common; diarrhea is not a feature without concurrent infections with other bacteria. Gross lesions include edema, congestion, and hemorrhage of the lungs, lymph nodes and thymus, distension of the intestine by gas or fluid, and congestion of the saccules of C-1 and C-2. Congestion of kidney, intestines, and other organs is also common, and myocardial necrosis and degeneration have been reported (el Sanousi and Gameel, 1993). In many cases of enterotoxemia, mixed infections with *Salmonella* sp., *E. coli*, or coccidia are likely responsible for increased mortality (Wernery et al., 1991).

***Clostridium perfringens* type C enterotoxemia** is well described in young NW camelids. It causes similar signs to those of *C. perfringens* type A enterotoxemia, but diarrhea is more common. In both types A and C enterotoxemia, pulmonary and tissue edema, cerebral edema, and neuronal necrosis are present. In all cases of clostridial enteritis, isolation of the organism from the ileum is not sufficient for confirmation of disease since this bacterium can be part of the normal flora. Culture should be coupled with appropriate lesions, histologic evidence of intralesional Gram-positive bacilli with morphologically consistent with *Clostridium* spp., and demonstration of the toxin using ELISA or identification of the toxin producing gene by PCR.

***Clostridium perfringens* type D enterotoxemia** has also been suspected in camelids with lesions similar to those in cattle and sheep. Serosal hemorrhages, prominent fibrin clots in the pericardial sac, and pulmonary and cerebral edema are strongly suggestive.

Mycoplasma haemolamae is a highly endemic, hemotropic, intra-erythrocytic Mycoplasma of llamas and alpacas that has been detected in camelids in Europe and North and South America (Guimaraesa et al., 2012). It is similar to the hemotropic mycoplasma organisms reported in cattle, pigs, and cats (Tornquist, 2012). These are small, 0.5 µm diameter bacteria that lack a cell wall and are present at the periphery of red blood cells on blood films (Fig. 7.13). They are usually round but can be ring-shaped or even linear. Electron microscopy suggests the organism does not actually invade the RBC, but rather indents the membrane (Reagan et al., 1990).

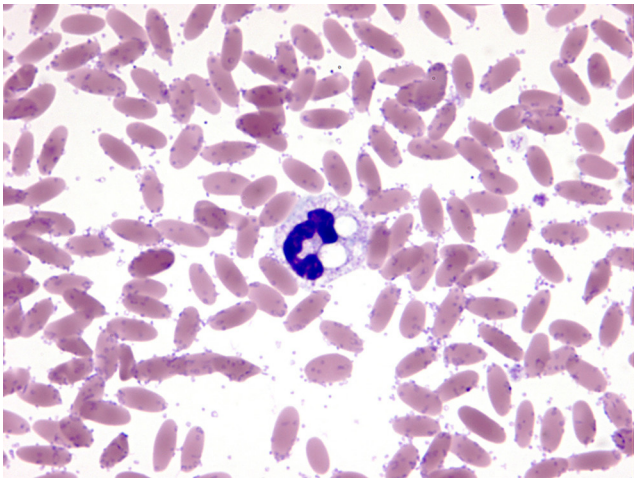

FIGURE 7.13 ***Mycoplasma haemolamae*** **in an alpaca cria**. Numerous, up to 1 µm in diameter, coccoid and ring-shaped bacteria (basophilic in this blood film) are present on and between the characteristic, ovoid camellid erythrocytes. Modified Wright stain. *(Photo Courtesy of M. Scott, Michigan State University)*

Infection is likely transmitted by insects, though specific vectors have not been identified. Vertical transmission has been established, with crias showing infection at 1 day of age before colostrum ingestion. Weight loss, lethargy, and anemia, which can be life-threatening, are commonly reported though the pathogenesis of infection is not well documented. It is likely that this organism predisposes to other diseases. Blood films can be diagnostic, but infection levels may be very low or cyclic. PCR tests are available and are particularly valuable in animals with low level infections.

Fungi

While numerous fungal diseases, including dermatophytosis and aspergillosis, have been reported in captive and free-range NW and OW camelids, **coccidioidomycosis** due to infection with ***Coccidioides immitis*** or ***C. posadasii*** is likely the most significant fungal disease in NW camelids. NW camelids appear to be very susceptible to this infection. It is acquired via inhalation of infectious arthrospores. The fungus is endemic in desert areas of southern California, the San Joaquin Valley, Utah, Nevada, Arizona, New Mexico, and Texas in the United States; as well as desert areas of northern Mexico, Northwest Venezuela, Argentina, Bolivia, and Paraguay, Honduras, Guatemala, and Nicaragua (Fowler, 2010e). Coccidioidomycosis usually presents as a respiratory disease. However, disseminated disease is common and can cause lesions affecting the neurological system, heart, liver, kidney, skin, bone, eyes, or lymphoid tissues; clinical signs reflect the location of the lesions (Coster et al., 2010; Fowler et al., 1992). Granulomas or granulomatous inflammation surrounding intralesional fungal arthroconidia, spherules, and endospores are characteristic, and inflammation can coalesce into large masses that efface organs. Inflammation surrounding freshly ruptured spherules may be suppurative. Multinucleated giant cells may also be present. In its parasitic form, infective arthroconidia transform into immature spherules (10 µm -20 µm in diameter) that mature into large, up to 200 µm diameter mature spherules (or sporangia) filled with 2 µm - 5 µm endospores. Sporangia subsequently rupture, releasing endospores into adjacent tissue that form new spherules, or if they are released into the environment, the endospores form mycelia. Aerosolized endospores can be a significant zoonotic threat, especially in a laboratory setting if appropriate precautions are not followed (Caswell and Williams, 2016).

Metazoa

Parasitic diseases of OW and NW camelids have a significant impact on meat, milk, and fleece production in South America and Africa and on the management of breeding herds and individuals in North America and Europe. Parasites of camelids have been exhaustively reviewed by Fowler (Fowler, 2010h). A few of the most important will be discussed here.

Lamanema chavezi**,** a trichostrongylid nematode, causes severe clinical signs, including high mortality in NW camelids (Fowler, 2010h). Its range was previously restricted to South America, but recently reports of infections in North America and New Zealand indicate the range has expanded with exportation of camelids from endemic areas (Jarvinen et al., 2014; McKenna et al., 2009). The life cycle includes ingestion of larvae, which then penetrate the intestinal wall and migrate to the liver and lung. Maturation occurs after migration up the trachea, re-ingestion, and migration back to the small intestine. Migration through the intestinal wall leads to severe catarrhal and hemorrhagic enteritis. Hepatic lesions include coagulative necrosis, hemorrhage, and multiple small abscesses, which if the animal survives will become fibrotic and mineralized. The mountain viscacha, a South American rodent, is considered the definitive host. How this infection might be maintained outside of South America is unknown (Fowler, 2010h).

Other trichostrongyles that infect camelids are similar to those of small ruminants and cattle. These include ***Ostertagia*** **spp**. and ***Haemonchus contortus.*** Both cause significant disease. Ostertagiasis causes similar proliferative gastritis as in domestic ruminants. *H. contortus* causes blood and protein loss though traumatic damage to the C-3 mucosa, which leads to ill-thrift and weight loss in addition to severe anemia and hypoproteinemia. Haemonchiasis causes multifocal to diffuse micro-ulcerations in the intestinal mucosa, as well as anemia related hepatic centrilobular degeneration and necrosis and tissue hypoxia.

Whipworms, especially ***Trichuris*** spp., are also a significant source of unthriftiness in NW and OW camelids.

Ova are remarkably resistant to environmental degradation, and thus *Trichuris* spp. are a particular problem in intensively managed camelids such as in small zoo exhibits. They have a direct life cycle. Eggs passed in the feces become infective on the pasture/substrate within three weeks. Upon ingestion, the eggs hatch and larvae invade the small intestinal mucosa, mature, then migrate to the cecum and colon to complete their life cycle. Whipworms have a characteristic tapered appearance, and the slender tapered end burrows into the intestinal mucosa. Catarrhal and hemorrhagic enterocolitis are typically seen grossly and histologically.

Parelaphostrongylus tenuis, the **meningeal worm**, and abberant larval migrans are a significant differential diagnosis for neurologic disease in camelids in North America. The white-tailed deer is the definitive host for the meningeal worm and the life cycle is complex (Fowler, 2010h). In the host, adult worms live in the veins of the dura mater where the eggs and larvae enter the blood vessels, flow to the lungs, and infiltrate the alveoli. They are then expectorated, swallowed, and are defecated into the environment. Terrestrial snails and slugs ingest the larvae, which are subsequently ingested again by the host. Larvae released from the snail in the host's stomach penetrate the peritoneum and migrate to the spinal cord via the spinal nerves. Larvae mature in the gray matter dorsal horns and adults then migrate to the brain via the subdural space and finally through the dura mater to the venous sinuses. In deer, the definitive host, there are few lesions and little clinical disease; however, in aberrant hosts, such as NW camelids, damage to the neurologic system is often severe and clinical signs reflect sites of damage. The presence of peripheral eosinophilia and increased eosinophils in the cerebrospinal fluid is variable.

There are typically no to very rare visible gross lesions in *P. tenuius* infections, and nematode larvae are only very rarely identified in the meninges of the brain. Microscopic lesions include lymphoplasmacytic perivascular cuffs axonal degeneration, hemosiderin-laden macrophages, eosinophils, meningitis, mineralization, hemorrhage, multinucleated giant cells, glial scars (linear tracts of glial cells), linear tracts of necrosis and mixed inflammation consistent with parasitic migration, and typically upon exhaustive histologic sectioning and examination, cross sections of nematodes (Dobey et al., 2014). Histologically, nematodes are usually degenerate, 100 to 200 mm in diameter, with a prominent thin, smooth cuticle, coelomyarian musculature, accessory hypodermal chords, and a gastrointestinal tract composed of multinucleate cells. A reproductive tract is often present and contains sperm or thin-shelled eggs.

Liver flukes, ***Fasciola hepatica*** and ***Dicrocoelium dendriticum***, are commonly found in NW camelids and cause subclinical and clinical disease. Acute massive infections can lead to bile stasis, liver degeneration and necrosis, hepatic insufficiency, and death. More commonly, chronic infections are characterized by bile duct obstruction and bile stasis with cholangitis and bile epithelial hyperplasia (Hilbe et al., 2015). Anemia and hypoproteinemia are common, and an unthrifty condition and particularly poor wool quality develops. Grossly, hepatic fibrosis and ascites are prominent. Regenerative hepatic nodules, mineralized foci, and abscesses may also be present. Histologically, in chronically infected animals, the liver contains multifocal to regionally extensive and occasionally coalescing areas of abundant fibrosis with biliary hyperplasia that surrounds regenerative nodules. Neutrophils, lymphocytes and plasma cells, and some macrophages are free in the parenchyma, periportal, and in the lumina of bile ducts. Flukes and their ova are often found in affected areas in the bile ducts and within the parenchyma. Characteristic features of trematodes are their tegument, lack of body cavity, and the presence of both gonads (hermaphrodites). Flukes found in camels can be differentiated by their size and the presence of spines. *Fasciola* spp. are the largest flukes in camelids (cm vs. mm) and have spines on their tegument. *F. hepatica* ova are large (120 mm –140 mm) and lack a miracidium. *D. dendriticum* ova are small (40 mm x 25 mm) oval, have a golden wall, are operculated, and contain a miracidium. Pulmonary trematodiasis in NW camelids can be associated with severe intimal and adventitial thickening and mild to moderate medial thickening (Hilbe et al., 2015).

Protozoa

In camelids, ***Eimeria macusaniensis***, an apicomplexan coccidian parasite that primarily affects young NW camelids is considered the most significant. At least 4 other species of Eimeria, ***E. alpacae, E. lamae, E. punoensis,*** **and** ***E. peruviana,*** also infect NW camelids, but none have been associated with the severe clinical signs seen in *E. macusaniensis* infection (Cebra et al., 2007; Fowler, 2010h). *E. macusaniensis* is by far the largest camelid coccidian. Its oocytes are 81-107 µm x 61-80 µm and tissue meronts are greater than 100 µm in diameter. Other *Eimeria* are typically a quarter of this size (Cebra et al., 2007; Fowler, 2010h). In OW camelids, 4 species of *Eimeria* have been characterized: ***E. bactriani, E. cameli, E. dromedarii*****,** and ***E. pellerdyi***. Similar to *E. macusaniensis, E. cameli* is the largest by far, with oocytes that are three to four fold larger than other species. All have been associated with severe disease, particularly in young camels (Hussein et al., 1987).

Clinical signs of *E. macusaniensis* infection include lethargy, weight loss, anorexia, diarrhea, and in some cases sudden death. The number of organisms in a given infection is important and heavily contaminated pastures can be a source of high infective doses and a significant factor in clinical outcomes. Co-infections are also likely important, since the destruction of enteric epithelium, particularly in the small intestinal crypts, can predispose to other infections, particularly with clostridial organisms (Rosadio et al., 2010).

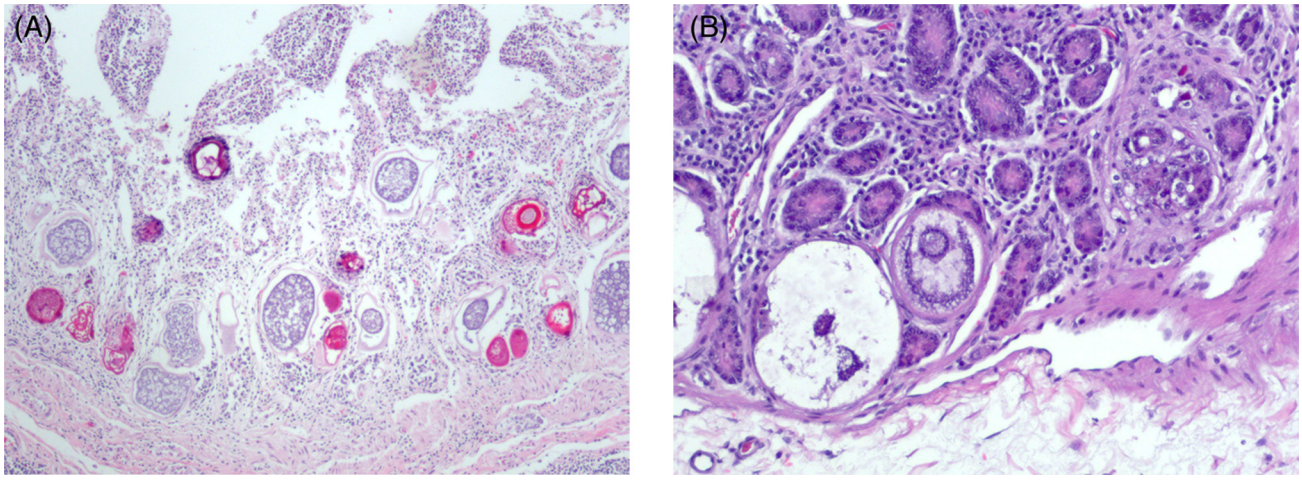

FIGURE 7.14 ***Eimeria macusaniensis* in the ileum of a llama**. (A) Myriad, round, coccidian parasites of multiple life-stages including meronts, microgamonts, macrogamonts, and oocysts with thickened eosinophilic walls are present in the mucosa. The number of organisms can completely efface the intestinal architecture. (B) An area of fibrosis in the lamina propria (right side of image) is associated with multiple degenerating coccidial schizonts and cysts in the lamina propria. Others compress or rupture into crypts.

Histologically, the density of meronts and gamonts within the mucosa of the jejunum and ileum is often remarkable and they may efface the parenchyma leaving little remaining unaffected tissue (Fig. 7.14A, B). Necrosis and loss of villi are the primary lesions, though in more chronic cases, severe fibrosis in the lamina propria may occur. The colon and duodenum are rarely affected. Coccidia are typically most dense in the deep crypts, but in heavy infections, parasites occupy the entire villus. Despite such massive infections, fecal examinations for *E. macusaniensis* are often negative, at least initially, in many animals.

Cryptosporidia are important apicomplexan protozoal pathogens in both young NW and OW camelids. They are an important cause of neonatal diarrhea that must be differentiated from other causes of neonatal diarrhea including bacterial endotoxemia or enterotoxemia, coccidiosis, coronavirus, rotavirus, or BVDV (Fowler, 2010h; Whitehead, 2009). Fecal smears and histologic sections can be examined with carbol-fuchsin modified Ziehl-Nielsen stain or periodic acid-Schiff reaction, for which the parasite will be positive. Villus atrophy is the primary histologic feature, with small 4-5 µm diameter basophilic round organisms that are intracellular but extracytoplasmic, and are typically most apparent superficially in cells at the tips of small intestinal villus epithelium.

Trypanosomiasis (also known as **nagana** or **surra**) is a severe disease of OW camels that affects productivity in regions where these animals are kept. ***Trypanosoma evansii*** causes surra in camels and is found in Southeast, East, and Central Asia, as well as North Africa (***T. evansii*** has been reported from South America; it has not been identified in NW camelids). The tsetse fly (*Glossinia spp.)* is the intermediate host and after feeding on the blood of an infected animal, the organisms multiply in the fly's hindgut and are deposited as metamorphosed trypanosome in the salivary secretions when the fly takes its next blood meal. Two syndromes have been reported in camels: an acute and chronic form. In acute surra, there is fever, anemia, edema, and weakness that progresses to paralysis. Pulmonary edema may lead to secondary pneumonia. Abortion and caseous mastitis have also been reported. Death usually occurs within weeks and there are many parasites in blood smears (Fig. 7.15). In the more chronic form, there is intermittent fever, anemia and edema, and progressive loss of condition. Between febrile episodes, finding organisms in blood smears may be challenging, so sample collection should coincide with periods of fever.

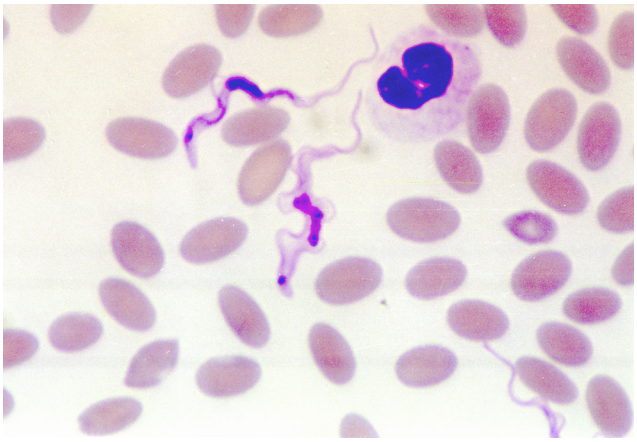

FIGURE 7.15 ***Trypanosoma evansii* (surra) in a camel**. *T. evansii* is characterized by its spindloid body, undulating membrane along one side, anterior flagellum, and kinetoplast. To detect organisms in blood smears, samples should be collected during periods of fever. Organisms may be very rare during the intervening afebrile periods. Wright-Giemsa stain *(Photo Courtesy of A. Tibary, Washington State University)*

T. brucei and ***T. congolensis*** are the causes of human sleeping sickness and can also infect camels. However, they are not a primary cause of surra in camels, though these organisms may prevent camels from being kept in the so-called Tsetse Belt of Africa. ***T. brucei, T. congolense****, and* ***T. vivax*** have been well described in cattle; however, lesions in camels have not been characterized in the literature. It would be expected that lesions might be similar, and consist of interstitial pneumonia, myocardial degeneration and myocarditis, bone marrow atrophy, lymphadenitis with follicular hyperplasia followed by atrophy and sclerosis, thymic atrophy, and renal membranoproliferative glomerulonephritis, and interstitial fibrosis (Valli et al., 2016). In cattle, organisms are most often seen in the liver and the cerebral cortex, though they may be found in any organ (Valli et al., 2016). PCR assays have been developed and can be used in camels.

Ectoparasites

New World camelids are aberrant hosts of the deer and sheep **nasal bot, *Oestrus ovis*;** the deer bot is more common (Fowler, 2010h; Fowler and Paul-Murphy, 1985). Mortality has not been reported, but a significant mucosal reaction in the nasopharynx consisting of granulomatous inflammation surrounding embedded larvae is common. Sneezing, coughing, and nasal discharge are associated with dyspnea since camelids are obligate nasal breathers. Old World camels are primary host of the camel bot, ***Cephalopina titillator***, which can lead to significant morbidity and rare mortality (Oryan et al., 2008). Nasopharyngeal mucous membranes are congested and edematous and covered by adherent layers of hemorrhage, mucus, and fibrin. Histologically, the mucosa is hyperplastic and infiltrated by lymphocytes, plasma cells, macrophages, and eosinophils; fibrosis develops in more chronic cases. Goblet cell hyperplasia and dilation of submucosal secretory glands may also be present. In rare cases, larvae may migrate through the ethmoturbinates resulting in parasitic meningitis and death.

Mange mites infect NW and OW camelids. **Sarcoptic mange (*Sarcoptes scabiei*)** is the most common and has the greatest impact on fiber quality and animal health. Sarcoptic mange is considered host specific and is probably not zoonotic. Camelids can also be infected with **Psoroptic (*Psorpotes* spp.)** and **chorioptic (*Chorioptes bovis*) mange** (Fowler, 2010h). Psorioptic and chorioptic mange mites are not considered host specific, can be transmitted across species, and may be zoonotic. Mange syndromes are similar across species and are described elsewhere in this text. Briefly, sarcoptic mange is characterized by crusted papules and pustules primarily on the limbs and ventrum, with hyperkeratosis and frequent secondary bacterial infection. Thickening of the skin is a differentiating feature of sarcoptic mange. Psoroptic mange produces moist papules progressing to crusts, on the shoulders, back, sides, and tailhead, as well as in the ears with subsequent fiber loss. Dermatitis induces pruritus, but there is little thickening of the skin. Chorioptic mange is less common, but has similar lesions to psoroptic mange. All forms of mange are reportable in the US; they may also be reportable in other jurisdictions.

E-SLIDES

7.e1 **Lama, zinc responsive dermatosis, skin.** This condition is seen on the face, ventral abdomen, thorax, and inguinal region of New World camelids. Note the markedly thickened parakeratotic hyperkeratosis, keratin accumulation, and hair follicle distention. Diffuse perivascular lymphoplasmacytic, eosinophilic, and histiocytic dermatitis are also present. (see Fig. 7.8). eSlide: VM05248

7.e2 **Alpaca, Eimeria macusaniensis, ileum.** Myriad round, coccidian parasites of multiple life stages including meronts, microgamonts, macrogamonts, and oocyts with thickened eosinophilic walls are present in the mucosal lamina propria. The number of organisms can completely efface the intestinal architecture. (see Fig. 7.14). eSlide: VM05249

REFERENCES

Aboellail, T.A., 2013. Pathologic and immunophenotypic characterization of 26 camelid malignant round cell tumors. Journal of Veterinary Diagnostic Investigation 25, 168–172.

Abu Damir, H., Barri, M.E., Tageldin, M.H., Idris, O.F., 1990. Clinical and subclinical colloid goitre in adult camels (*Camelus dromedarius*) at Kordofan region of Sudan. British Veterinary Journal 146, 219–227.

Abubakar, M.S., Fatihu, M.Y., Ibrahim, N.D.G., Oladele, S.B., Abubakar, M.B., 2010. Camel pneumonia in Nigeria: Epidemiology and bacterial flora in normal and diseased lung. African Journal of Microbiology Research 4, 2479–2483.

Adney, D.R., Bielefeldt-Ohmann, H., Hartwig, A.E., Bowen, R.A., 2016. Infection, Replication, and Transmission of Middle East Respiratory Syndrome Coronavirus in Alpacas. Emerging Infectious Diseases 22, 1031–1037.

Ali, A., Mehana, E.E., Ahmed, A.F., El-Tookhy, O., Al-Sobayil, A., Al-Hawas, A., 2011. Ovarian hydrobursitis in female camels (*Camelus dromedarius*): clinical findings, histopathology and fertility after unilateral surgical ablation. Theriogenology 76, 492–499.

Ali, A., Al-Sobayil, F.A., Hassanein, K.M., Al-Hawas, A., 2012. Ovarian hydrobursitis in female camels (*Camelus dromedarius*): the role of *Chlamydophila abortus* and a trial for medical treatment. Theriogenology 77, 1754–1758.

Al-Qarawi, A.A., 2005. Infertility in the dromedary bull: a review of causes, relations and implications. Animal Reproduction Science 87, 73–92.

Al-Qarawi, A.A., Abdel-Rahman, H., Elmougy, S.A., 2001. Impairment in the pituitary-thyroid axis of the *Camelus dromedarius* infected with *Trypanosoma evansi*. Deutche Tierarztliche Wochenschrift 108, 172–174.

Altmann, D., Kronberger, H., Schueppel, K.F., Lippmann, R., Almann, I., 1976. (Enzootic meningo-encephalomyelitis simplex in New World tylopods and equines) Bornasche krankheit (meningo-encephalomyelitis Simplex enzootica equorum) bei Neuwelttylopoden und Equiden. In: Verhandlungsber, 18th International Symposium Erkr. Zootiere, Innsbruck. pp. 127–132.

Balamurugan, V., Venkatesan, G., Bhanuprakash, V., Singh, R.K., 2013. Camelpox, an emerging orthopox viral disease. Indian Journal of Virology 24, 295–305.

Bani-Ismail, Z., Hawkins, J.F., Siems, J.J., 1999. Surgical correction of polydactyly in a camel (*Camelus dromedarius*). Journal of Zoo and Wildlife Medicine 30, 301–304.

Benirschke, K., 2008. Comparative Placentation.

Carman, S., Carr, N., DeLay, J., Baxi, M., Deregt, D., Hazlett, M., 2005. Bovine viral diarrhea virus in alpaca: abortion and persistent infection. Journal of Veterinary Diagnostic Investigation 17, 589–593.

Caswell, J.L., Williams, K.J., 2016. Respiratory system. In: Maxie, M.G. (Ed.), Jubb, Kennedy, and Palmer's Pathology of the Domestic Animals. 6 ed Elsevier, St. Louis, MO, pp. 465–591.

Cebra, C.K., 2009. Disorders of carbohydrate or lipid metabolism in camelids. Veterinary Clinics of North America Food Animal Practice 25, 339–352.

Cebra, C.K., Garry, F.B., Powers, B.E., 1995. Lymphosarcoma in 10 new world camelids. Journal of Veterinary Internal Medicine 9, 381–385.

Cebra, C.K., Valentine, B.A., Schlipf, J.W., Bildfell, R.J., McKenzie, E., Waitt, L.H., Heidel, J.R., Cooper, B.J., Löhr, C.V., Bird, K.E., Saulez, M.N., Firshman, A.M., 2007. Eimeria macusaniensis infection in 15 llamas and 34 alpacas. Journal of the American Veterinary Medical Association 230, 94–100.

Coster, M.E., Ramos-Vara, J.A., Vemulapalli, R., Stiles, J., Krohne, S.G., 2010. *Coccidioides posadasii* keratouveitis in a llama (*Lama glama*). Veterinary Ophthalmology 13, 53–57.

Crossley, B.M., Barr, B.C., Magdesian, K.G., Ing, M., Mora, D., Jensen, D., Loretti, A.P., McConnell, T., Mock, R., 2010. Identification of a novel coronavirus possibly associated with acute respiratory syndrome in alpacas (*Vicugna pacos*) in California, 2007. Journal of Veterinary Diagnostic Investigation 22, 94–97.

Crossley, B.M., Mock, R.E., Callison, S.A., Hietala, S.K., 2012. Identification and characterization of a novel alpaca respiratory coronavirus most closely related to the human coronavirus 229E. Viruses 4, 3689–3700.

Decker, R.A., Hruska, J.C., McDermid, A.M., 1979. Colloid goiter in a newborn dromedary camel and an aborted fetus. Journal of the American Veterinary Medical Association 175, 968–969.

Dobey, C.L., Grunenwald, C., Newman, S.J., Muller, L., Gerhold, R.W., 2014. Retrospective study of central nervous system lesions and association with Parelaphostrongylus species by histology and specific nested polymerase chain reaction in domestic camelids and wild ungulates. Journal of Veterinary Diagnostic Investigation 26, 748–754.

Dunkel, B., Del Piero, F., Wotman, K.L., Johns, I.C., Beech, J., Wilkins, P.A., 2004. Encephalomyelitis from West Nile Flavivirus in 3 Alpacas. Journal of Veterinary Internal Medicine 18, 365–367.

Duraffour, S., Meyer, H., Andrei, G., Snoeck, R., 2011. Camelpox virus. Antiviral Research 92, 167–186.

el Sanousi, S.M., Gameel, A.A., 1993. An outbreak of enterotoxaemia in suckling camels. Zentralblatt veterinarmed A. 40, 525–532.

Fowler, M., 2010a. Congenital/Hereditary conditions. In: Fowler, M. (Ed.), Medicine and surgery of camelids. 3 ed Wiley-Blackwell, Ames, IA.

Fowler, M., 2010b. Feeding and nutrition. In: Fowler, M. (Ed.), Medicine and surgery of camelids. Wiley-Blackwell, Ames, IA.

Fowler, M., 2010c. General Biology and Evolution, 3 ed. Medicine and Surgery of Camelids. Wiley-Blackwell, Ames, IA, 3-16.

Fowler, M., 2010d. Hemic and Lymphatic Systems. In: Fowler, M. (Ed.), Medicine and Surgery of Camlids,. Third ed Wiley-Blackwell, Ames, IA, pp. 407–422.

Fowler, M., 2010e. Infectious diseases. In: Fowler, M. (Ed.), Medicine and surgery of camelids. 3 ed Wiley-Blackwell, Ames, IA, pp. 173–230.

Fowler, M., 2010f. Integumentary system. In: Fowler, M. (Ed.), Medicine and surgery of camelids. 3 ed Wiley-Blackwell, Ames, IA.

Fowler, M., 2010g. Multisystemic disorders. In: Fowler, M. (Ed.), Medicine and surgery of camelids. 3 ed Wiley-Blackwell, Ames, IA.

Fowler, M.E., 2010h. Parasites. In: Fowler, M.E. (Ed.), Medicine and surgery of camelids. 3 ed Wiley-Blackwell, Ames, IA, pp. 231–269.

Fowler, M., Bravo, P.W., 2010. Reproduction. In: Fowler, M. (Ed.), Medicine and Surgery of Camelids. Third ed Wiley-Blackwell Publishing, Ltd, Ames, IA, pp. 429–478.

Fowler, M.E., Paul-Murphy, J., 1985. *Cephenemyia sp.* infestation in the llama. California Veterinarian 39, 10–12.

Fowler, M.E., Pappagianis, D., Ingram, I., 1992. Coccidioidomycosis in llamas in the United States: 19 cases (1981-1989). Journal of the American Veterinary Medical Association 201, 1609–1614.

Gao, S., Luo, J., Du, J., Lang, Y., Cong, G., Shao, J., Lin, T., Zhao, F., Belák, S., Liu, L., Chang, H., Yin, H., 2013. Serological and molecular evidence for natural infection of Bactrian camels with multiple subgenotypes of bovine viral diarrhea virus in Western China. Veterinary Microbiology 163, 172–176.

Gauly, M., Vaughan, J., Hogreve, S.K., Erhardt, G., 2005. Brainstem auditory-evoked potential assessment of auditory function and congenital

deafness in llamas (*Lama glama*) and alpacas (*L. pacos*). Journal of Veterinary Internal Medicine 19, 756–760.

Gerros, T.C., Stone, W.C., 1994. What is your diagnosis? Complete bilateral choanal atresia in a llama. Journal of the American Veterinary Medical Association 205, 179–180.

Guimaraesa, A.M.S., Tothc, B., Santosa, A.P., do Nascimentoa, N.C., Kritchevskyc, J.E., Messick, J.B., 2012. Genome Sequence of "Candidatus Mycoplasma haemolamae" Strain Purdue, a Red Blood Cell Pathogen of Alpacas (*Vicugna pacos*) and Llamas (*Lama glama*). Journal of Bacteriology 194, 6312–6313.

Henningson, J.N., Steffen, D.J., Topliff, C.L., Kurth, K.L., Dubielzig, R.R., Brodersen, B.W., Bedenice, D., Eskridge, K.M., Callan, R.J., Reggiardo, C., Rupp, G.P., Kelling, C.L., 2012. Systemic distribution of viral antigen in alpacas persistently infected with bovine pestivirus. Veterinary Pathology 50, 308–317.

Hilbe, M., Robert, N., Pospischil, A., Gerspach, C., 2015. Pulmonary Arterial Lesions in New World Camelids in Association With *Dicrocoelium dendriticum* and *Fasciola hepatica* Infection. Veterinary Pathology 52, 1202–1209.

House, J.A., Gregg, D.A., Lubroth, J., Dubovi, E.J., Torres, A., 1991. Experimental equine herpesvirus-1 infection in llamas (*Lama glama*). Journal of Veterinary Diagnostic Investigation 3, 137–143.

Hussein, H.S., Kasim, A.A., Shawa, Y.R., 1987. The prevalence and pathology of Eimeria infections in camels in Saudi Arabia. Journal of Comparative Pathology 97, 293–297.

Intisar, K.S., Ali, Y.H., Khalafalla, A.I., Mahasin, E.A., Amin, A.S., Taha, K.M., 2010. The first report on the prevalence of pestivirus infection in camels in Sudan. Tropical Animal Health and Production 42, 1203–1207.

Irwin, J.A., 2001. Lymphosarcoma in an alpaca. Canadian Veterinary Journal 42, 805–806.

Jacobsen, B., Algermissen, D., Schaudien, D., Venner, M., Herzog, S., Wentz, E., Hewicker-Trautwein, M., Baumgärtner, W., Herden, C., 2010. Borna disease in an adult alpaca stallion (*Lama pacos*). Journal of Comparative Pathology 143, 203–208.

Jarvinen, J.A., Whitley, E.M., Kreuder, A.J., Schleining, J.A., 2014. Identification of Lamanema chavezi Becklund 1963 infection in a llama (*Lama glama*) in the United States. Journal of Veterinary Diagnostic Investigation 26, 178–183.

Jones, M., Miesner, M., Grondin, T., 2009. Outbreak of *Streptococcus equi* ssp. zooepidemicus polyserositis in an alpaca herd. Journal of Veterinary Internal Medicine 23, 220–223.

Kapil, S., Yeary, T., Evermann, J.F., 2009. Viral diseases of new world camelids. Veterinary Clinics of North America Food Animal Practice 25, 323–337.

Khalafalla, A.I., Saeed, I.K., Ali, Y.H., Abdurrahman, M.B., Kwiatek, O., Libeau, G., Obeida, A.A., Abbas, Z., 2010. An outbreak of peste des petits ruminants (PPR) in camels in the Sudan. Acta Tropica 116, 161–165.

Kiupel, M., VanAlstine, W., Chilcoat, C., 2003. Gross and microscopic lesions of polioencephalomalacia in a llama (*Lama glama*). Journal of Zoo and Wildlife Medicine 34, 309–313.

Lamoureux, J.L., Fitzgerald, S.D., Church, M.K., Agnew, D.W., 2011. The effect of environmental storage conditions on bone marrow fat determination in three species. Journal of Veterinary Diagnostic Investigation 23, 312–315.

Lee, L.C., Scarratt, W.K., Buehring, G.C., Saunders, G.K., 2012. Bovine leukemia virus infection in a juvenile alpaca with multicentric lymphoma. Canadian Veterinary Journal 53, 283–286.

Martin, J.M., Valentine, B.A., Cebra, C.K., Bildfell, R.J., Löhr, C.V., Fischer, K.A., 2009. Malignant round cell neoplasia in llamas and alpacas. Veterinary Pathology 46, 288–298.

Mattson, D.E., Baker, R.J., Catania, J.E., Imbur, S.R., Wellejus, K.M., Bell, R.B., 2006. Persistent infection with bovine viral diarrhea virus in an alpaca. Journal of the American Veterinary Medical Association 228, 1762–1765.

McKenna, P.B., Morley, C., Koning, M., Tahana, J.S., Taylor, M.J., 2009. Confirmation of the occurrence of the nematode parasite Lamanema chavezi Becklund in South American camelids in New Zealand. New Zealand Veterinary Journal 57, 395–396.

Milad, K.E., Ridha, G.S., 2009. The occurrence of thiamine-responsive polioencephalomalacia in dromedary breeding camels in Libya: preliminary investigation and diagnosis. Iraqi Journal of Veterinary Sciences 23 (Supplement I), 119–122.

Moore, C.P., Shaner, J.B., Halenda, R.M., Rosenfeld, C.S., Suedmeyer, W.K., 1999. Congenital ocular anomalies and ventricular septal defect in a dromedary camel (*Camelus dromedarius*). Journal of Zoo and Wildlife Medicine 30, 423–430.

Narnaware, S.D., Dahiya, S.S., Tuteja, F.C., Nagarajan, G., Nath, K., Patil, N.V., 2015. Pathology and diagnosis of *Mycobacterium bovis* in naturally infected dromedary camels (*Camelus dromedarius*) in India. Tropical Animal Health and Production 47, 1633–1636.

Nelson, D.D., Duprau, J.L., Wolff, P.L., Evermann, J.F., 2015. Persistent Bovine Viral Diarrhea Virus Infection in Domestic and Wild Small Ruminants and Camelids Including the Mountain Goat (*Oreamnos americanus*). Frontiers in microbiology 6, 1415.

Nolen-Walston, R., Bedenice, D., Rodriguez, C., Rushton, S., Bright, A., Fecteau, M.E., Short, D., Majdalany, R., Tewari, D., Pedersen, D., Kiupel, M., Maes, R., Del Piero, F., 2007. Eastern equine encephalitis in 9 South American camelids. Journal of Veterinary Internal Medicine 21, 846–852.

Oryan, A., Valinezhad, A., Moraveji, M., 2008. Prevalence and pathology of camel nasal myiasis in eastern areas of Iran. Tropical Biomedicine 25, 30–36.

Pillitteri, C.A., Craig, L.E., 2012. Hepatic encephalopathy associated with hepatic lipidosis in llamas (*Lama glama*). Veterinary Pathology 50, 177–181.

Powers, B.E., Johnson, L.W., Linton, L.B., Garry, F., Smith, J., 1990. Endometrial biopsy technique and uterine pathologic findings in llamas. Journal of the American Veterinary Medical Association 197, 1157–1162.

Reagan, W.J., Garry, F., Thrall, M.A., Colgan, S., Hutchison, J., Weiser, M.G., 1990. The clinicopathologic, light, and scanning electron microscopic features of eperythrozoonosis in four naturally infected llamas. Veterinary Pathology 27, 426–431.

Rebhun, W.C., Jenkins, D.H., Riis, R.C., Dill, S.G., Dubovi, E.J., Torres, A., 1988. An epizootic of blindness and encephalitis associated with a herpesvirus indistinguishable from equine herpesvirus I in a herd of alpacas and llamas. Journal of the American Veterinary Medical Association 192, 953–956.

Reed, S.K., Sermevolos, S.A., Rist, P.K., Valentine, B.A., 2007. Morphologic and biochemical characterization of hyperextension of the metacarpophalangeal and metatarsophalangeal joints in llamas. American Journal of Veterinary Research 68, 879–885.

Ribadu, A.Y., Ogwu, D., Njoku, C.O., Eduvie, L.O., 1991. An abattoir survey of female genital disorders of imported camels (*Camelus dromedarius*) in Kano. Nigeria. British Veterinary Journal 147, 290–292.

Ridge, S.E., Harkin, J.T., Badman, R.T., Mellor, A.M., Larsen, J.W.A., 1995. Johnes disease in alpacas (*Lama pacos*) in Australia. Australian Veterinary Journal 72, 150–153.

Rosa, F.B., Rissi, D.R., 2013. Pathology in Practice. Multicentric B-cell lymphoma. Journal of the American Veterinary Medical Association 243, 645–647.

Rosadio, R., Londoñe, P., Pérez, D., Castillo, H., Véliz, A., Llanco, L., Yaya, K., Maturrano, L., 2010. Eimeria macusaniensis associated lesions in neonate alpacas dying from enterotoxemia. Veterinary Parasitology 168, 116–120.

Rosychuk, R.A., 1994. Llama dermatology. Veterinary Clinics of North America Food Animal Practice 10, 228–239.

Sakamoto, K., Kiupel, M., Frank, N., March, P.A., 2004. Vertebral malformation, syringomyelia, and ventricular septal defect in a dromedary camel (*Camelus dromedarius*). Journal of Veterinary Diagnostic Investigation 16, 337–340.

Sartin, E.A., Crowe, D.R., Whitley, E.M., Treat, R.E., Purdy, S.R., Belknap, E.B., 2004. Malignant neoplasia in four alpacas. Journal of Veterinary Diagnostic Investigation 16, 226–229.

Schulman, F.Y., Krafft, A.E., Janczewski, T., Reupert, R., Jackson, K., Garner, M.M., 2003. Camelid mucoutaneous fibropapillomas: clinicopathologic findings and association with papillomavirus. Veterinary Pathology 40, 103–107.

Shapiro, J.L., Watson, P., McEwen, B., Carman, S., 2005. Highlights of camelid diagnoses from necropsy submissions to the Animal Health Laboratory, University of Guelph, from 1998 to 2004. Canadian Veterinary Journal 46, 317–318.

Simmons, H.A., Fitzgerald, S.D., Kiupel, M., Rost, D.R., Emery, R.W., 2005. Multicentric T-cell lymphoma in a dromedary camel *Camelus dromedarius*. Journal of Zoo and Wildlife Medicine 36, 727–729.

Steffen, D.J., Topliff, C.L., Schmitz, J.A., Kammerman, J.R., Henningson, J.N., Eskridge, K.M., Kelling, C.L., 2014. Distribution of lymphoid depletion and viral antigen in alpacas experimentally infected with Bovine viral diarrhea virus 1. Journal of Veterinary Diagnostic Investigation 26, 35–41.

Stoughton, W.B., Gold, J., 2015. Streptococcus equi subsp zooepidemicus pleuropneumonia and peritonitis in a dromedary camel (*Camelus dromedarius*) calf in North America. Journal of the American Veterinary Medical Association 247, 300–303.

Sumar, J., 1989. Defectos congenitos y heredetarios en la alpaca-teratologia. Consejo Nacional de Ciencia y Techologia, Cusco, Peru.

Tharwat, M., Al-Sobayil, F., Ali, A., Hashad, M., Buczinski, S., 2012. Clinical, ultrasonographic, and pathologic findings in 70 camels (*Camelus dromedarius*) with Johne's disease. Canadian Veterinary Journal 53, 543–548.

Tibary, A., Anouassi, A., 2001. Retrospective study on an unusual form of ovario-bursal pathology in the camel (*Camelus dromedarius*). Theriogenology 56, 415–424.

Tibary, A., Fite, C., Anouassi, A., Sghiri, A., 2006. Infectious causes of reproductive loss in camelids. Theriogenology 66, 633–647.

Tornquist, S.J., 2012. Mycoplasma haemolamae in New World Camelids. In: Miller, R.E., Fowler, M. (Eds.), Fowler's Zoo and Wild Animal Medicine, Current Therapy. Elsevier Saunders, St. Louis, MO, pp. 646–648.

Underwood, W.J., Bell, T.J., 1993. Multicentric lymphosarcoma in a llama. Journal of Veterinary Diagnostic Investigation 5, 117–121.

Ure, A.E., Elfadl, A.K., Khalafalla, A.I., Gameel, A.A., Dillner, J., Forslund, O., 2011. Characterization of the complete genomes of *Camelus dromedarius* papillomavirus types 1 and 2. Journal of General Virology 92, 1769–1777.

Vallenas, P.A., Cummings, J.F., Munnell, J.F., 1971. A gross study of the compartmentalized stomach of two New World camelids – the llama and guanaco. Journal of Morphology 134, 399–424.

Valli, V.E.O., Kiupel, M., Bienzle, D., 2016. Hematopoietic system. In: Maxie, G. (Ed.), Jubb, Kennedy, and Palmer's Pathology of the Domestic Animals. Elsevier, St. Louis, MO.

Vítovec, J., 1982. Renal cell carcinoma in a camel (*Camelus dromedarius*). Veterinary Pathology 19, 331–333.

Wernery, U., 2014. Camelid brucellosis: a review. Revue Scientifique Et Technique De L Office International Des Epizooties 33, 839–857.

Wernery, U., Kinne, J., 2012a. Foot and mouth disease and similar virus infections in camelids: a review. Revue Scientifique Et Technique De L Office International Des Epizooties 31, 907–918.

Wernery, U., Kinne, J., 2012b. Tuberculosis in camelids: a review. Revue Scientifique Et Technique De L Office International Des Epizooties 31, 899–906.

Wernery, U., Seifert, H.S., Billah, A.M., Ali, M., 1991. Predisposing factors in enterotoxemias of camels (*Camelus dromedarius*) caused by *Clostridium perfringens* type A. Revue d'elevage et de medecine veterinair des pays tropicaux 44, 147–152.

Wernery, U., Nagy, P., Amaral-Doel, C.M., Zhang, Z., 2006. Lack of susceptibility of the dromedary camel Camelus dromedarius to foot and mouth disease virus serotype O. Veterinary Record 158, 201–203.

Whitehead, C.E., 2009. Neonatal diseases in llamas and alpacas. Veterinary Clinics of North America Food Animal Practice 25, 367–384.

Whitehead, C.E., Bedenice, D., 2009. Neurologic diseases in llamas and alpacas. Veterinary Clinics of North America Food Animal Practice 25, 385–405.

Xu, L., Zhang, Z., Wang, L., Feng, D., Zhou, X., Xu, B., Zhao, D., 2012. Cloning and polymorphism analysis of prion protein gene in domestic bactrian camel in China. Gene 491, 256–259.

Younan, M., Bornstein, S., Gluecks, I.V., 2012. MERS and the dromedary camel trade between Africa and the Middle East. Tropical Animal Health and Production 48, 1277–1282.

Zakian, A., Nouri, M., Kahroba, H., Mohammadian, B., Mokhber-Dezfouli, M.R., 2016. The first report of peste des petits ruminants (PPR) in camels (*Camelus dromedarius*) in Iran. Tropical Animal Health and Production 48, 1215–1219.