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Nutritional Diseases of Camelid Species: Literature Review

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Abstract

Although camelids are considered susceptible to all the nutrient deficiency and toxicity diseases described, few published studies are available. Nutrient-related deficiency and toxicity diseases are well recognized and appropriately treated, resulting in an under-reporting of these conditions. This thesis will review nutritional diseases in camelids, focusing primarily on those diseases for which literature reports have been documented.

Összefoglaló

A tevefélék érzékenyek az összes leírt tápanyaghiányos és toxicitási betegséggel szemben, ennek ellenére kevés publikált tanulmány áll rendelkezésre. Sok esetben ezeket a tápanyag-hiánnyal és toxicitással összefüggő betegségeket időben felismerik és megfelelően kezelik, ami azt eredményezi, hogy ezekről a problémákról kevés információ található. Ez a dolgozat a tevefélék takarmányozási eredetű betegségeit tekinti át, elsősorban azokra a betegségekre összpontosítva, amelyeket a szakirodalomban már dokumentáltak.

Contents

Abbreviations:

AST: Aspartate aminotransferase

BCS: Body condition score

DM: Dry matter

EDTA: Ethylenediaminetetraacetic acid

GGT: Gamma-glutamyl transferase

IM: Intramuscular

IV: Intravenous

LDH: Lactate dehydrogenase

NPN: Non-protein nitrogen

NSAIDs: Non-steroidal anti-inflammatory drugs

NEFA: Non-esterified fatty acids

NWC: New World Camelids

OWC: Old World Camelids

PEM: Protein energy malnutrition

RBC: Red blood cells

SDH: Succinate dehydrogenase

VFA: Volatile fatty acids

WBC: White blood cells

1. Introduction to the Camelids of the world

1.1 What and who are camelids?

In this review, the term "camelids" will be used inclusively to address both New World camelids (NWC) and Old World camelids (OWC). Camels from the Old World are classified as Camelus, while camels from the New World are classified as Lama. Camels live in Asia and Africa, whereas llamas, alpacas, guanacos, and vicugna live in South America. The species in this family are large, and most of them have been domesticated. They are herbivores and have three-chambered stomachs. Their top lip is split into two, and they can move each part separately! They have long necks, a small head, and long legs with padded feet and two large toes. They live in small groups made up of females and their young, with one adult male leading the herd.

1.2 History

Although there are no living species of the camelid family in North America today, the fossil record shows that the family started and evolved there, as preserved remains have been found in the La Brea Tar Pits near Los Angeles. The members of the camelid family that were in North America are thought to have resembled modern day llamas. Members of this family migrated to South America and Asia over land bridges. By the end of the last glacial age, they had become extinct in North America.

1.3 New world camelids (NWC)

All NW camelids are collectively known as lamoids and origin from South America.

- The vicugna (Vicugna vicugna), smallest member of the camel family (Figure 1).
- The alpaca (Vicugna pacos), is a domesticated species that derives from the vicugna (Figure 2).
- The guanaco (Lama guanicoe) (Figure 3).
- The llama (Lama glama), is a domesticated species, descendant of the guanaco (Figure 4).

Figure 1: Image of a vicugna

Figure 2: Image of an alpaca

Figure 3: Image of a guanaco

Figure 4: Image of a llama

1.4 Old world camelids (OWC)

Bactrian camel, Wild Bactrian Camel and Dromedary/Arabian Camel camels

- The Bactrian camel (Camelus Bactrianus) (Figure 5), also called domestic Bactrian camel or Mongolian camel, either of two of the three living species of camels inhabiting the steppes and arid regions of Eurasia.
- The wild Bactrian camel (Camelus Ferus) (Figure 7), is a close relative to the Bactrian camel but was never domesticated. This wild species lives in north-western China and south-western Mongolia. Their look consists of a double-hump with even-toed ungulates.

- Dromedary/Arabian Camel (Camelus Dromedarius) (Figure 6). These domestic camels range from North Africa, Ethiopia, and western and central Africa and are the tallest camel of the three species and can reach up to 7ft 10 (2.4 m). [20]

Figure 5: Image of a bactrian camel

Figure 6: Image of a dromedary

Figure 7: Image of a wild bactrian camel

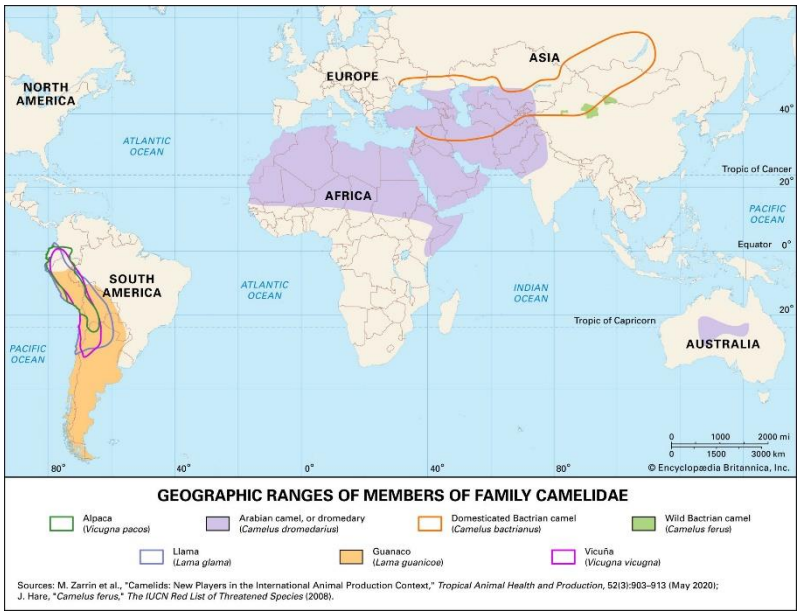

Figure 8: Present-day ranges of living members of the family Camelidae.

2. Herd Health & Nutrition

Purpose of camelid domestication

Humans have managed to establish many reasons why they domesticated camelids. Such reasons range from companion animals ‘pets’, fiber production, exhibit animals, working class animals, and reproductive purposes, including gestation and milk production. The keeping of such animals may be due to all the reasons mentioned above or for only one purpose.

Natural feeding options

Camelid practices are similar to those for sheep. Water needs to be accessible at all times. These animals are adaptive feeders, eating grasses, forbs, shrubs, and trees. They can be kept on a variety of pastures and hay, but due to the limited pastures for larger sized herds, as well as seasonal depletion of forage, a dry lot option is typically used as a backup. Not many herds have access to year-round grazing, and even when it is possible, farmers and owners need to understand that the nutrition content of the feed varies by the day. Such instability is affected by the use of fertilizers, the maturity of vegetation, individual animal selective grazing, or even the type of moisture received (rain or irrigation). Because of the animals’ high feed conversion, hays with high protein content, like alfalfa, are not recommended because the animals can easily become overweight.

Geographic influences

Camelid farmers and owners should be aware of or informed by local veterinary practitioners that all types of camelid supplements may not be applicable to their animals due to geographic influence. The local veterinary practitioner shall inform the owners regarding potential deficiency or toxicity concerns for other animals that would also apply to camelids. Other information that shall be provided is what forages grow well for pasture and/or stored hay crops in the given geographical region.

Only the free-ranging populations of wild Bactrian camels are endangered, as Bactrian camels kept in zoos are generally of domestic origin. Vicugna are listed as vulnerable. In addition to those camelids held in captivity, there are domesticated populations of camelids (except vicugna and guanaco) maintained throughout the world. Most are fairly hardy animals, but there are

some specific disease concerns as domesticated llamas and alpacas have become hobby pets, and thus management and genetic issues are an increasing source of disease.

Facility design for feeding

When designing a camelid facility that will utilize pastures, a reliable and consistent procedure for moving the animals from one pasture to the dry holding or dry lot area is of high importance. When the pasture being grazed on is of high quality, restrictions on grazing are required in order to prevent overweight animals. Restrictions are also important in cases of limited pasture space.

If stored forage is being fed, it is essential to store it away from all elements that could and will compromise the nutrient content. Abundant bunk space is required, as timid animals will otherwise end up eating leftovers at best. Feeding hay on the ground is not ideal, except in winter when the floor is covered in snow. Supplementation must be delivered to the animals individually in well separated feeders to ensure that the animal in need will take them.

It is also essential to maintain a consistent clean water supply in order to ensure the health of the camelids. In large herds, it is typical to find a large water tank that must be periodically drained to remove any contaminants, such as dust and plant material. Automatic waterers such as the float regulated drinkers are a very efficient way to deliver water to the animals but they need to be cleaned often and checked daily for function. Farms in colder climates will need to have a heating system not to let the water freeze or the water can be freshened up multiple times daily. Failure to consume adequate amount of water daily will have a negative impact on the nutrition and digestion of the animal resulting in poor thermoregulation, lactation and general health.

Feed procurement and storage

Having a reliable source from which to obtain high-quality forage is very important. It is also necessary to have an idea of other suppliers in the area, as shortages are unpredictable due to weather factors. A responsible supplier will provide a detailed forage analysis that can be obtained prior to purchase, as it is an essential factor for the foundation of a comprehensive nutrition program. Once the forage analysis is obtained, it is possible to dictate, ideally with the help of a veterinary nutritionist, any need for macro- and micronutrient supplementation. If the facility has enough storage space, it is recommended to stock as much forage as possible from

the same source to avoid any imbalances. Most supplements come as concentrated powders or pellets in bags, so these do not require a yearly supply, but the production batch shall always be taken into consideration.

It is important to keep the stored supplies safe from environmental factors as well as animals. If not secured, animals may find their way to the store, causing them to accidentally overeat. This will lead to cases of gastric acidosis that can be very easily prevented.

3. Applied digestive anatomy and feeding behaviour

Applied anatomy and function

Both NW and OW species are related and have many similar anatomical features and disease susceptibilities, even though they may also be very different. Understanding the appropriate feeding practices and nutrition for any given species is critical, and in order to do so, we must first understand their digestive system and its respective metabolic and physiological responses. Most camelid farmers tend to approach camelids with the same techniques used for cattle and sheep. Though the basics may be very similar, the unique anatomy and metabolism of camelids, which allow them to thrive in their harsh native environment, require different feeding and nutritional approaches. Understanding this will allow us to develop a more tailored feeding management practice.

Some of the main concerns include obesity, vitamin and mineral deficiencies and intoxications, metabolic derangements, and congenital malformations (particularly in the young). Domesticated animals are also more susceptible to degenerative arthropathy and dental disease as they age than wild camelids.

Camelids are often misrepresented as "pseudo-ruminants" due to differences in foregut anatomy in comparison with species from the suborder Ruminantia. The camelid digestive process is similar to that of a moderately-sized browsing or grazing ruminant species, with modifications resulting from their unique metabolic adaptations. Camelids, both NWC and OWC, are potentially susceptible to the full range of ruminant animal nutritional diseases. This review will provide the most common camelid nutritional diseases, including: protein-energy malnutrition, obesity, fore-stomach acidosis, hypophosphatemia rickets, selenium deficiency/toxicity, copper deficiency/toxicity, zinc-responsive dermatitis, and plant poisonings.

Important infectious diseases to take into consideration include coccidiosis, bovine viral diarrhoea virus, alpaca fever, and meningeal worm in NW camelids; trypanosomiasis and camel pox in OW camels; and foot-and-mouth disease in Bactrian camels and NW camelids.

Forestomach Anatomy

All species of NW and OW camelids have demonstrated the ability to thrive in harsh, dry, nutrient-poor environments where domestic ruminants are unable to thrive. Their ability to extract the maximum amount of water and nutrients from their feed is the main reason for their survival. Understanding this lays the groundwork for recognizing that the camelid gut is different from that of a ruminant.

Like adult ruminants, camelids rely on bacterial fermentative digestion in the forestomach (pH 6.4–6.8) to extract nutrients from plant material. This process requires a warm, moist anaerobic environment with a large capacity for long fiber retention time and the ability to mechanically and enzymatically break down large feed particles. Mechanical breakdown by chewing and gastric contractions also play an important role.

The forestomach enzymes are mainly of microbial origin, and the bacterial fermentation of plant-based carbohydrates results in the production of organic volatile fatty acids (VFAs). Under normal conditions, the VFAs produce acetate, propionate, and butyrate in a 4:1:1 ratio before feeding and a 7:2:1 ratio a few hours after feeding. The absolute VFA concentration after feeding is about 140 mmol/L, which is 30–40% higher when compared to ruminants under similar conditions.

Fermentation occurs in the first two gastric compartments (C1 and C2) (Figure 9) and the cranial four fifths of the third gastric compartment (C3). C1 occupies most of the left side of the abdomen, consisting of a large cranial and caudal sacs that are divided ventrally by a transverse pillar. C1 makes up about 83% of the gastric volume and about 50% of the abdominal volume. C2 is small, making up only 6% of the gastric volume, and lays atop C3 (11% of the gastric volume) medially to C1 (Figure 9).

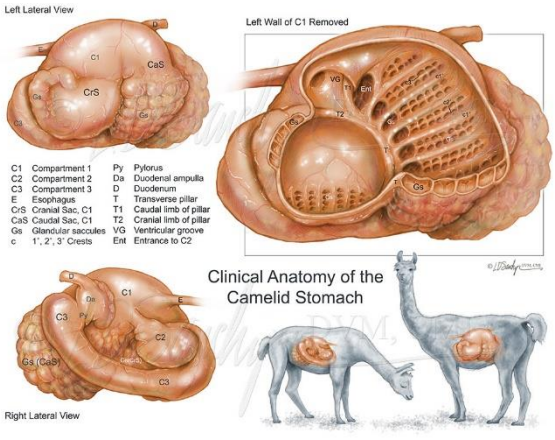

Figure 9: Stomach compartments of camelids

4. Nutritional diseases: literature review

4.1 Sources and methods

For the general understanding and history of the camelids, a study of the book titled Llama and Alpaca Care book [24], the Veterinary Treatment of Llamas and Alpacas book [25], and other scientific studies have played crucial roles in this literature review.

These literature resources provided the basic theory for understanding the anatomy and physiology of camelids. It also provided collective information on the nutritional deficiencies and toxicities of new world camelids.

Several publications were also utilised to describe the relation between the New World and Old World camelids and methods used to identify their nutritional and metabolic diseases.

Any additional knowledge was obtained from discussing camelid nutrition (mainly with New World camelids) with veterinarians who work in this field. The main goal of the research has been to gain an overview of the common and uncommon metabolic diseases in all camelid species and how to treat and/or prevent them.

The aim is to keep improving our knowledge with regards to the nutritional requirements of these animals and to foresee/prevent any nutritional imbalances, especially with their domestic population numbers increasing.

4.2 Results

4.2.1 Macronutrient diseases

By definition, starvation means a prolonged and complete deprivation of food intake. True starvation is most likely underreported, but incomplete starvation, or protein energy malnutrition (PEM), is a more frequent problem. PEM severity depends on the degree and timing of the dietary insufficiency.

Protein-energy malnutrition

Epidemiology

The main cause of PEM is the use of poor-quality forages together with the animals' inability to consume sufficient amounts relative to their requirements. Animals going through a life stage with high protein requirements, such as growing animals, gestating animals, and milking animals, are the most susceptible to PEM. In natural conditions, the animals' body condition score (BCS) mimics the seasonal forage growth. This means that animals tend to give birth during the rainy season, when there is an abundance of high-quality forage. During the dry season, the animals lose a considerable amount of weight and end up in various stages of malnutrition. Animals that end up giving birth during the dry season are highly prone to PEM, secondary infections, and parasitic problems that often lead to death. Animals raised outside of their native environment bring along new challenges such as heat stress and the increased energy requirements for the colder climates (if wet, maintenance energy requirements may increase by up to 75% more). Younger animals have a greater surface area to volume ratio and, thus, a greater heat loss and a lower critical temperature compared with the adults [4, 9, 12, 22].

Clinical findings

Weight loss and a decline in the body condition score are the main clinical signs. Growing animals may also show a slowing or a cessation in growth rates. Other findings may include delayed puberty, anoestrus, irregular oestrus, decreased birth weights, and also embryonic death. Lactating females will show a reduced or complete stop in milk production. Females in gestation or lactation may experience hepatic lipidosis as a secondary complication. Animals suffering from PEM will maintain a healthy appetite until near the terminal stages, compared to animals suffering from infections or parasitic diseases, which will start by showing a reduced appetite.

Diagnosis and Treatment

Direct palpation is required to determine the BCS, as a thick fleece may easily hide body weight and condition changes. Once an unexplained BW loss or BCS loss is identified, a cause needs to be determined. Chronic infections and dental diseases may induce BW loss and BCS loss, such as PEM. Protein-energy malnutrition is often a secondary process in chronic diseases.

Dietary malnutrition and/or starvation result in an increase in blood non-esterified fatty acids (NEFA) up to 0.6–1.0 mEq/L, a mild increase in blood B-hydroxybutyrate up to 1-2 mg/dL, and mild to no changes in blood triglycerides. The increase in NEFA concentrations is not directly associated with any clinical signs but may indicate risk. Pregnant and lactating camelids on restricted diets may develop more rapid and severe fat mobilization, with the possibility of resulting in hepatic lipidosis. Hypoalbuminemia and hypoproteinaemia are often observed, but serum chemistry parameters may vary according to any underlying secondary condition. Anaemia, neutropenia, and lymphopenia may also be associated with PEM, but a low white blood cell count (WBC) may reflect the underlying infection. Animals that have been diagnosed in the early stages of the disease process may be easily recovered with appropriate feeding therapy and supportive care, while animals in an advanced stage of the disease have a very poor prognosis even with aggressive therapy.

Prevention

PEM prevention is mainly done by appropriate feeding regimes where forage quality is matched to the nutrient needs of the animal. If the forage quality is insufficient, supplemental feeds are necessary, especially in colder climates. Dry matter is reasonably increased in colder climates in order to meet the high energy demands on the animal. To ensure proper nutrition, forage chemical analysis is required, and frequent BW and BCS shall be taken. Critical times to monitor BCS would be during early to mid-pregnancy, early to mid-lactation, and periodically (4-6 times per year) for other herd animals [10].

Obesity

Epidemiology

Obesity is considered one of the main nutritional disorders in camelids, mainly in the NW. The consumption of excess energy is what leads to animal obesity. Ad libitum feeding of high-quality forages or overfeeding of supplements is the main cause of obesity, and recent studies also show that excess protein intake may cause obesity in alpacas. Many readily available commercial supplements contain substantial amounts of cereal grains or any other readily fermentable fiber, but even low-energy fibers may contain unwanted caloric intake due to the camelids highly efficient fiber fermentation process. Obesity can lead to increased susceptibility to heat stress, metabolic imbalances, infertility, and problems with locomotion [15, 16].

Diagnosis

Access fat accumulation may be diagnosed by increasing body weight beyond what would be expected during pregnancy or growth, coupled with a BCS assessment exceeding 4 (on a 5-point scale) or 7 (on a 9-point scale). The ideal BCS would be 3 on a 5-point scale and 5 on a 9-point scale. Obesity is identified as having the highest number on both scales (5 and 9). Weight accumulation or weight loss has not yet been quantified for camelids.

Treatment and prevention

An animal's energy requirement consists of its function of lean body mass and its physiologic state (maintenance, growth, pregnancy, lactation). Reduced energy intake and/or increased energy expenditure are methods to both treat and prevent obesity. Animals that identify as obese should have their caloric intake regulated by reducing or limiting supplementation, improving forage availability and quality, or a combination of both. The lower-quality forage should be provided prior to grazing in order to reduce the intake of pasture. These animals should be kept isolated from other non-obese animals to prevent them from stealing their food. It is suggested to pasture obese animals on mature pastures of lower quality. Larger paddocks will encourage animals to move around more, which in turn increases their energy expenditure [22].

For weight reduction to be successful, required energy intake is calculated based on the ideal body weight, then reduced fractionally between 60% and 75%, as it is done in dogs and cats. If weight loss happens too fast, the animals may be at risk of hepatic lipidosis. A study has shown

that llamas that have lost 15-20% of their body weight within a 10- to 14-day period are at about 50% higher risk of some degree of hepatic lipidosis. The unique metabolic adaptation of camelids is similar to that of cats, thus making them more susceptible to hepatic lipidosis. Thus, the approach to weight loss should be tackled with a 70–75% caloric intake restriction in order to achieve a 0.5–1% weight loss per week in order to prevent complications. Ongoing monitoring is essential to maintaining a herd at an optimal weight and preventing obesity.

Protein excess

Epidemiology

The forestomach microbial populations are capable of utilizing non-protein nitrogen (NPN) sources to synthesize microbial protein. This microbial protein is ultimately digested and absorbed by the host animal. Dietary NPN is eventually converted to ammonia, which aids in the fermentation of forestomach microbial fibers. This mechanism is the primary reason the camelids are able to utilize poor quality feeds. NPN toxicosis is a disease unique to ruminants. Compounds of concern are urea, nitrates, and nitrites. Urea is a common fertilizer and ruminant feed supplement that is rapidly cleaved into carbon dioxide and two ammonia molecules. In order to convert these ammonia molecules into microbial proteins, fermentation of carbohydrates is required. If the forage quality level is low, the carbohydrate demand is not met in order to support the NPN formation. This will result in ammonia not being utilized by the microbes and will eventually diffuse across the stomach, eventually ending up in the portal blood circulation. The liver is normally capable of converting excess ammonia back into urea for recycling or excretion, but if its capacity is exceeded, the free ammonia will cause cellular toxicosis, thus disrupting energy metabolism and potassium homeostasis [1].

Clinical presentation

Excess levels of NPN will overwhelm the liver, resulting in a dramatic increase in blood ammonia concentrations and subsequent clinical signs. Urea toxicosis is a rapid consequence, typically occurring as early as 10 minutes and up to 2 hours after excess consumption. Clinic signs may include frothy salivation, bruxism (teeth grinding), colic, muscle tremors, incoordination, and recumbency, followed by death in rapid progression.

Diagnosis and treatment

Ammonia can be smelt off the animal, but the quantification of ammonia shall be determined by blood (1-4 mg/dL) or stomach (> 80 mg/dL) concentrations. Treatment is useless for the most part unless diagnosis happens in the early stages. The main objective would be to reduce the ammonia production and absorption, which can be done by drenching with weak acids (vinegar or 5% acetic acid) and cold water, although the most effective treatment would be emptying the stomach.

Prevention

The best way to prevent protein excess is to minimize the risk of excess exposure to NPN sources. Forestomach microbes may be slowly adapted to utilizing NPN by slowly incorporating it into the animals' diet and ensuring an adequate amount of fermentable carbohydrates, if given. It is recommended to keep the urea concentration in feed under 3%, although many commercial ruminant diets have greater amounts as they aim to cater for cattle and sheep. The amount of urea in a supplement can be determined by dividing the percent protein from NPN provided on the product label by 281. The NPN should not make up more than 30% of the total dietary crude protein content.

4.2.2 Mineral Deficiency Diseases

Macro-mineral deficiency

Epidemiology

Insufficient intake of salts such as sodium and potassium may lead to pica, which is a feeding behaviour characterized by chewing on sticks, pipes, other objects, or eating dirt. In general, forages have a low content of sodium but a sufficient amount of potassium. The provision of salt, either as table salt or trace mineralized salt, minimizes the risks of sodium deficiency.

Diagnosis and treatment

Hypocalcaemia and hypomagnesaemia both show very similar clinical signs. Diagnosis is done, as in ruminants, by analysing the history, signalling, and response to therapy with means of blood analysis to confirm the diagnosis.

Hypocalcaemia mainly occurs during pregnancy or lactation or as a secondary complication of anorexia or illness, but rarely progresses to cause physical weakness. Smooth muscle function may be affected, resulting in ileus. Diagnosis by blood analysis may be used to justify treatment with intravenous calcium gluconate. Calcium gluconate should be administered slowly and carefully, as it is in other ruminant species. A suggested dose would be 1 g of calcium per 45 kg of body weight. Cardiac auscultation is necessary to identify any changes in the heart's rhythm and rate.

Hypomagnesaemia (extremely rare in camelids) and hypoalbuminemia may occur simultaneously, as they do in ruminants. This is most commonly seen in camelids grazing on lush pasture, as it causes a rapid transit time of ingesta through the bowel and so can provoke the condition because if the transit time through the bowel is too rapid, then insufficient magnesium will be absorbed. Clinical signs such as ataxia, tetany, weakness, and recumbency may be observed. Blood evaluation is required in order to provide confirmation for diagnostics. Treatment is rarely successful if they are convulsing. Although blood magnesium levels can be restored to normal, there is usually irreparable brain damage. Intravenous treatment consisting of a mix of calcium gluconate and magnesium is usually enough to reduce the clinical signs. A subsequent subcutaneous injection of 50% magnesium sulphate (10–50 mL) or 100 mL of 25% will minimize relapses. It may be prudent to give some other sort of supportive treatment, such

as a mixture of 20% calcium borogluconate, 5% magnesium hypophosphite, and 20% glucose given intravenously, coupled with NSAIDs. It should be kept in mind that these cases are on a knife edge, and so any treatment by any route may well cause death [7, 11].

Prevention

Prevention is based on appropriate mineral supplementation. Excess dietary phosphorus and potassium, along with inadequate dietary magnesium, adversely affect calcium homeostasis (potassium being the main influence).

Copper deficiency

Epidemiology

Copper (Cu) is an essential constituent of the diet and is required for the harvesting of energy from digested feeds and, together with iron, is required in the production of haemoglobin. It is an essential mineral for bone, tendon, and cartilage and melanin production. It is also needed by the body for protection against certain toxins. Copper is absorbed into the body from the small intestine and is stored in the liver. Beta-carotene is a required component to aid copper absorption. Copper availability is depressed when there is an increase in molybdenum, sulphur, iron, zinc, cadmium, selenium, and calcium in the diet. Foregut microbes metabolize dietary molybdenum and sulphates, generating a range of chelating thiomolybdate compounds. These thiomolybdates bind to dietary copper, making it unavailable for incorporation into copper-sensitive metalloenzymes. Some of these thiomolybdates may be absorbed and bind to the copper-containing enzymes, thus reducing their bioavailability. Dietary copper is usually provided in the forage or as a mineral supplement to camelids. In many regions, forage is low in copper due to the alkaline soil that is used or high in substances that may interfere with its absorption, so copper supplementation is suggested.

Clinical presentation

Typical clinical signs seen in domestic ruminants are ataxia (hind limb, posterior paresis), anaemia, immunodeficiency, and infertility; loss of hair (poor fiber quality); dilute hair colour (achromotrichia); skin pigments; chronic diarrhoea; and leukoencephalomalacia. It will also cause the growth of abnormal bone, connective, and tendon tissue, which will cause lameness and reduced growth rates. The production of stringy fibre and excessive shedding are usually

the first symptoms noticed by the owner. Where all of the above-mentioned clinical signs have been seen in sick camelids, their connection to copper deficiency is difficult to confirm [11].

Diagnosis

The diagnosis is not as straight forward in camelids as compared with sheep because the plasma copper levels are less reliable. History may help to suggest copper deficiency, but more supportive evidence is required by the determination of feed copper content and animal copper status. If a feed has a copper value below 4 ppm dry weight, it strongly indicates deficiency, while feed levels between 4 and 7 ppm may lead to deficiency. The ultimate measure for copper deficiency is a liver biopsy. Copper concentrations in the liver less than 10 mcg/g indicate deficiency, while those between 10 and 90 mcg/g are marginal. Serum copper levels are not reliable for diagnostic purposes.

Treatment and prevention

Treatments may include the oral administration of copper supplements, injecting copper calcium ethylenediaminetetraacetic acid (EDTA) up to 2 mg/kg of copper, and reducing dietary copper inhibitors (mainly zinc). Improvement should be observed within 2–4 weeks. The recommended daily dietary intake is 9–12 ppm (DM basis); however, higher intake will be required in the presence of higher inhibitor concentrations.

When assessing copper requirements in the diet, the practitioner should try to maintain a copper to molybdenum ratio of between 6:1 and 10:1, whereas in sheep it should be between 6:1 and 8:1. Ratios of 15:1 may lead to copper toxicity and 4:1 to copper deficiency. It is typically suggested to use sheep feed for camelids, but not all copper sources are readily available to ruminants. Copper Oxide is basically unavailable for absorption, so copper sulphate or chloride is preferred.

Iron Deficiency

Epidemiology

Iron deficiency is commonly seen when there isn’t enough iron intake, as seen in growing animals feeding on a milk based diet or in animals suffering from chronic blood loss such as parasitic infestation. Younger animals tend to be more susceptible to these iron deficiencies due

to their higher iron demand, lower intake, and being more prone to parasites. This is seen less in adults as the grass-fed diet provides sufficient amounts of iron, even if there is low availability of dietary iron [18].

Clinical presentation

The first and most noticeable clinical sign is anaemia. Camelids that present with diarrhoea, non-regenerative microcytic disorders, hypochromic anaemia, and a poor growth rate are considered to have an iron deficiency. These animals typically respond well to iron supplementation.

Diagnosis

Hematologic indices measuring red blood cells (RBC) number, volume, and haemoglobin content and concentration characterize the state of anaemia but the ultimate diagnostic tool is a bone marrow biopsy smear.

Treatment and Prevention

According to the level of deficiency, iron supplementation may be implemented via the administration of whole blood or via parenteral iron dextran (a safe dose for camelids has yet to be established). A cautious dose of 150 mg per animal at 2–3 week intervals is indicated. The dose of 600 mg of iron as gleptoferron should be divided into three doses and administered on alternate days as three injections of 1 ml containing 200 mg each. In most cases, iron supplementation results in being enough (ferrous sulphate or calcium phosphate minerals are readily available iron sources). It is required to check that the iron content of mineral salts is not just ferric oxide, as it is not readily available, but most forages and commercial diets typically contain enough iron.

Selenium deficiency

Epidemiology

Selenium deficiency is commonly associated with lameness and nutritional myodegeneration, also known as white muscle disease. It is also associated with infertility, stillbirths, and very weak new-born animals. Young animals are more likely to experience clinical signs. Selenium, in the form of selenium-dependent glutathione peroxidase, plays an important intracellular

antioxidant role and has complementary actions with the membrane-bound antioxidant vitamin E. It is important for reducing the damage caused by superoxide bursts of free radicals. An abundance of either selenium or vitamin E may partially or completely compensate for the deficiency of the other [7, 11]. The main cause of deficiency is the lack of available selenium in the feed. This problem is seen regionally as a result of a lack of selenium in the soil, low soil pH, and the presence of iron or aluminium complexes, which impact the selenium content of the plants. Plants incorporate available soil selenium into proteins such as selenomethionine or selenocysteine.

In pregnant animals, selenium is efficiently transferred across the placenta and concentrated in the foetal liver. This is critical because milk has a low selenium content, so neonatal animals rely heavily on their hepatic selenium reserves to support their metabolic activities.

Clinical signs

Severe selenium deficiency will result in pathological degeneration of skeletal muscle fibers with secondary fibrosis. Signs are seen depending on the specific muscles affected and the severity of the condition. Both hind legs are commonly symmetrically affected; however, the heart and tongue muscles are typically affected in young animals. The degeneration of skeletal muscles will be seen as lameness, weakness, and movement difficulties. Young animals with damaged heart muscles may suffer from sudden death. New-borns with affected tongue muscles will experience difficulties in nursing and may be diagnosed as ‘dummy’ animals. Stillbirth and abortion may be seen in pregnant animals [6].

Diagnosis

The diagnosis is straight forward with plasma samples to measure glutathione peroxidase or by using hepatic selenium concentrations. Heparin is the anticoagulant required (normally a green-topped vaccutainer). With regards to blood selenium, camelids differ from other ruminants by having higher concentrations of glutathione peroxidase activity in the serum, which results in fewer differences between serum and whole blood selenium concentrations. Deficient blood concentrations are defined as being below 120 ng/ml (nanograms per millilitre), while normal concentrations are between 150 and 220 ng/ml. Expected hepatic selenium concentrations are valued at 1.0–2.5 mcg/g of dry weight, as seen in other animals. Foetal hepatic concentrations

are higher, sitting at about 5.25 mcg/g of dry weight, due to the concentrating ability. Hepatic selenium is to be considered deficient when the value reaches below 0.4 mcg/g.

Treatment and prevention

Treatment of selenium deficiency involves supportive care such as oral supplementation or parenteral selenium injection. If a patient is diagnosed with a blood or liver selenium deficiency, parenteral supplementation will immediately increase the selenium status. The label dosage of selenium concentration is 55 mcg/kg body weight, this needs to be calculated properly according to the product's concentration. Dietary supplementation of selenium on a daily basis is the ideal method for preventing such a deficiency. According to the FDA, selenium supplement concentration shall be no more than 0.3 ppm (DM basis) [4]. The daily requirements of selenium are 0.74 mg/day (although some studies have shown that 1 mg/day has resulted in better blood selenium levels), to obtain this, free choice trace mineral salt containing 90 ppm selenium is needed, assuming that the consumption is 7-9 g/day. Another method of selenium supplementation is via grain or commercial pellets. All the supplementation methods mentioned must be adjusted according to the selenium levels present in the forage being used [4].

Organic selenium supplements such as selenomethionine are more readily available compared to inorganic sources as they are not altered by the stomachs. Selenomethionine is incorporated into the tissue proteins, resulting in higher tissue and milk selenium concentrations. This may be beneficial to females during late pregnancy and lactation.

Zinc deficiency

Epidemiology

Zinc deficiency affects almost every aspect of metabolism, cellular growth, reproduction, performance, and immune responses and is linked to over 200 metalloenzymes. Zinc availability is reduced by excess dietary copper and calcium. Because zinc is required for the synthesis of retinol-binding proteins, its availability is related to vitamin A status (but also citrate, histidine, and lactose). Forages containing low amounts of zinc (under 22 ppm) require zinc supplementation by means of minerals or concentrates. Zinc deficiency is strongly correlated with skin lesions in camelids, and many cases have responded well to pharmacologic doses of zinc [3].

Clinical presentation

Zinc responsive dermatitis is normally described as non-pruritic. Idiopathic hyperkeratosis is typical in camelids aging from 1-2 years, and coloured fleece animals tend to be more susceptible. Such lesions are more commonly seen in hairless areas of the body such as the ventral abdomen, medial thighs, axilla, and inguinal region. These lesions are characterized by the thickening of the skin with adhering crusts [21].

Diagnosis

Parasitic diseases shall be ruled out by means of a full-thickness skin biopsy with histologic examination. Zinc status shall be determined by serum zinc concentrations or hepatic concentrations; both are equally effective. Serum sampling may be altered by rubber stoppers (plastic stoppers are essential), as they contain zinc, and any blood samples collected in rubber-topped bottles will not indicate any zinc deficiency. Serum zinc levels may also be reduced in the presence of physical changes such as inflammatory reactions that release macrophage cytokines. Compared to other ruminants, camelids have lower serum zinc levels, which may result in more frequent false-positive results [3].

Treatment and prevention

Treatment is simple: with oral supplementation of 1g of zinc sulphate (36.3% zinc) daily or 2-4g of zinc methionine (10% zinc), animals tend to respond well within a 2–3 month period. Zinc methionine is generally preferred, as it is thought to be more available in the presence of interfering substances such as high calcium intake. Zinc does not have good palatability, so it is required to mask its flavour with apple sauce (or anything similar) [3].

Proper supplementation is the first step in preventing or minimizing the risks of zinc deficiency. The daily recommended intake is 45–60 ppm on a DM basis, depending on the physiological state of the animals. It is important to take into consideration the intake of other substances, such as calcium and copper. It should be stressed that this is a very over diagnosed disease, and other causes of the parakeratosis should be investigated [3].

4.2.3 Mineral toxicity diseases

Potassium excess

Epidemiology

Many times, when forages are intensively grown, they result in having a high potassium content due to the intensive use of fertilizers. Excess potassium, as previously stated, reduces the ability to absorb magnesium, increasing the risk of hypomagnesaemia. Excess potassium also affects the body's acid-base response and diminishes the calcium homeostatic mechanisms. The kidneys are in charge of excreting excess potassium, which results in high urine potassium concentrations and an elevated urine pH. If this continues for a while, it will predispose the animals to supersaturated urinary mineral content, leading to the precipitation of uroliths. Male camelids are at higher risks of blocking the urethra as the diameter is narrower than in females and the sigmoid flexure [11, 14].

Clinical presentation

Clinical signs associated with uroliths depend on the degree of blockage the animal is suffering from. If there is a complete blockage, retrograde pressure will build up in the bladder to the point of bladder rapture and subsequent death of the animal. If the blockage is incomplete, the animal may experience stranguria, prolonged urination posture, urine dribbling, and bloody urine. Animals affected may also be depressed, lethargic, or show signs of teeth grinding and abdominal pain and discomfort [11].

Diagnosis

Mainly based on history, signalment, and clinical signs.

Treatment and prevention

Treatment strongly depends on the severity of the condition, its duration, and whether there are any secondary complications. Camelids that have a single stone or plug at the tip of the penis have a higher chance of survival compared to ones that have an obstruction in the sigmoid flexure. Catheterization can usually relieve the obstruction, but if that fails, retrograde hydro propulsion or surgery may be required. If, after treatment, the urinary function is not restored, the prognosis is grave.

Medical treatment during the acute phase involves the use of IV fluids, anti-inflammatory medications, urethral relaxants, and antibiotics. Fluids help stabilize the acid-base balance and correct azotaemia. Furosemide can be given at 2–5 mg/kg IV or 0.66 mg/kg IM. A tube may be placed into the bladder, and anti-inflammatories and antibiotics can be administered. Dimethyl sulfoxide (DMSO) used in the flush can decrease inflammation and bring some debris into solution.

It is assumed that the pathology of such disease in camelids is similar to that of ruminants, so the approach to prevention is to increase water intake by adding salt to the diet and to maintain an appropriate calcium-phosphorus ratio of 2:1 to 4:1. Monitoring the levels of forage potassium, calcium, phosphorus, magnesium, and acid insoluble ash (silica content) is strongly suggested.

Struvite crystals can be prevented by altering the diet in a way to induce urine acidification. Ammonium chloride supplementation (5–10 g/kg DM) is a common way to induce such acidification, and there are also available commercial products that are used in cattle farms that can be used.

Sulphur intoxication

Epidemiology

High sulphate intake has recently been associated with polioencephalomalacia. The pathogenesis is not fully understood, but there is a link with excessive dietary sulphate intake. It seems that the dietary sulphate is reduced to sulphur dioxide, thus forming H_2S gas. The excess H_2S gas is eventually eructated and inhaled, thus increasing the blood H_2S levels. This is a potent cellular toxin that affects the cells respiration capabilities and acts like a pro-oxidant. Low rumen pH promotes the formation of such gas as a result of a high-grain diet.

Clinical presentation

It is suspected that camelids may suffer from this condition due to their stomach capabilities, but there have been no cases reported as of yet. Affected ruminants show signs such as blindness, recumbency, seizures, and sudden death in the acute form, while visual impairment and ataxia may be seen in less severe cases. Animals can recover from the less severe form if treated in time, or progress to the acute form.

Diagnosis

Clinical signs and lack of response to thiamine therapy. Definitive diagnosis is done during necroscopy, when microscopic lesions of cerebrocortical necrosis along with polioencephalomalacia are identified.

Treatment and Prevention

If the disease is in an advanced stage, treatment with thiamine will not be successful. Prevention is the best way to go along by monitoring the feed and water sulphate content and minimizing low rumen pH conditions. Levels higher than 1000 mg/L of sulphate in water have been linked to such disease in ruminants. Polioencephalomalacia has been linked to water sulphate levels of 2500 mg/L. Safe levels of sulphate in water are considered to be below 600 mg/L [8].

Copper toxicity

Epidemiology

Copper accumulation in the liver is the main cause of mineral hepatotoxicity in camelids. Many supplements may contain too much copper as they would be formulated for other animal species such as poultry, horses, or cattle. Errors in the formulation or mixing of these supplements will make them toxic to camelids. Copper is stored in hepatic lysozymes; once these have reached full capacity or the animal finds itself in a stressful situation, the copper is released into the cytoplasm, leading to the death of the hepatocytes. Free copper ions will also lead to lipid peroxidation and cell membrane damage [2].

Clinical presentation

Signs such as jaundice, progressive anorexia, lethargy, dyspnoea, weakness, recumbency, and colic may be seen, often leading to death within two days. Gun-metal discoloration of the kidneys and other organs is not typically seen in camelids as it is in other ruminants. There will be an increase in serum aspartate aminotransferase (AST), lactate dehydrogenase (LDH), succinate dehydrogenase (SDH), and y-glutamyl transferase (GGT) [23].

Diagnosis

For a definitive diagnosis at post-mortem, liver copper levels need to be above 8000 mmol/kg DM or kidney levels higher than 650 mmol/kg DM. Kidney samples are more reliable as there

is not the same interference by iron as in the liver. A dietary analysis may result in having high copper concentrations compared to molybdenum and other blocking minerals. Dietary copper content has a toxicity concentration range of 25–36.6 mg/kg. Diets having over 20 mg/kg and 10:1 Cu:Mo are considered to increase the risk of inducing copper toxicity [2].

Treatment and prevention

If there is a suspected acute ingestion of copper, oral absorption inhibitors or chelators such as ammonium molybdate, sodium sulphate, penicillamine, or zinc acetate may be given as drenches daily for up to a week or more. Penicillamine will also gradually remove the excess liver copper in chronically accumulating patients. In the case of animals surviving the acute phase (48 hours), they may recover but may also develop chronic ill-thrift due to severe liver damage. There is no known reliable antidote for when the disease has developed too far. However, a subcutaneous injection of 3.4 mg/kg ammonium tetrathiomolibdate on 3 alternate days has been successfully used for treatment of copper poisoning in sheep and can potentially be used in camelids.

The required dietary copper is 9–12 ppm (DM), although it may exceed these values. All types of feed should be evaluated for copper content, with any excess removed. Foraged foods typically contain 6–12 ppm copper, but they may differ geographically. Horse or cattle supplements must be avoided, as they contain higher copper concentrations than required. On the other hand, sheep supplements generally contain no copper or values under 30 ppm. Copper availability due to molybdenum, iron, and sulphur should be taken into consideration. Liver copper concentrations should be routinely monitored by examining deceased animals or performing liver biopsies on selected animals.

Selenium toxicity

Epidemiology

Herbivorous species are most often affected due to a stomach microbial imbalance. Acute selenium intoxication is typically caused by the increased ingestion of the supplement sodium selenite, which is highly available, will induce acute selenium intoxication. This is seen in regions where selenium deficiency is common, so the use of such supplements may be abused [19].

Alkali disease is a regional disease of the Great Plains of North America. It is a chronic disease resulting from the consumption of non-accumulator and some secondary accumulator plants over time. Acute toxicosis is caused by the consumption of primary and secondary-accumulating plants or by the inappropriate use of supplements [19].

Clinical presentation

Animals effected by acute selenium toxicosis generally show signs of abdominal pain, abnormal posture, ataxia, and depression, later followed by dyspnoea, tachypnea, salivation, tachycardia, and cardiovascular collapse within minutes/hours following an excess amount of selenium either consumed or injected. The breath may have a garlic scent.

Animals with chronic selenium toxicosis may present themselves with rough coats, hair loss, and dystrophic hoof growths. These animals will appear dull, lacking in vitality, and emaciated. Congenital hoof malformations may be seen due to the highly efficient placental transfer of selenoproteins [17].

Diagnosis

The selenium concentrations in the blood, liver, and kidneys are important diagnostic tools as their values increase with prolonged exposure.

Treatment and prevention

There is no available antidote for acute or chronic selenium toxicosis. Animals will recover totally when removed from the selenium-rich diet; however, if the animals are left on these pastures, they will be found dead. Prevention is mainly achieved by implementing proper use of parenteral products and proper monitoring of dietary supplements.

4.2.4 Vitamin deficiency and toxicity diseases

Vitamin A

It is extremely rare for camelids to be fed a diet deficient in vitamin A. The coat will show a generalized seborrhoea. However, the main sign is irreversible retinal atrophy, which causes blindness. The animals have fixed, dilated pupils. In advanced cases, they will be ataxic and may show head pressing. The condition is irreversible [5].

Vitamin E

Epidemiology

This condition is seen in camelids. It is not a real deficiency but rather a vitamin E/selenium-responsive dermatitis. The animals appear healthy and are non-pruritic, but they lose their hair.

Clinical presentation

Various degrees of lameness, weakness, and movement difficulties may be due to a vitamin E deficiency as a result of myopathy, but neuro-degeneration is a more common finding.

Diagnosis

An animal's vitamin E status can be determined by measuring the a-tocopherol levels in serum or liver biopsy. The question of whether vitamin E is solely responsible must be determined [5].

Treatment and prevention

Treatment of such deficiency may be done by subcutaneous administration of a parenteral vitamin E solution dosed according to the animals' weight. Such administration shall be done every 2–3 weeks in order to bring the levels of vitamin E back to normal. Prevention and maintenance can be accomplished by offering the appropriate daily dietary supplementation. Animals on fresh pasture do not require any added supplements, only animals that are mainly fed mature and dry pasture require additional supplementation. Many readily available commercial supplements made for sheep and goats do not contain sufficient amounts of vitamin E required in camelid species.

Vitamin D deficiency

Epidemiology

Camelid species may develop metabolic bone diseases. Such disease is secondary to many factors, mainly hypophosphatemia. The main reason being insufficient dietary vitamin D along with its endogenous synthesis and other mineral imbalances. Mineral imbalances may occur due to a lack of dietary phosphorus or an abundance of calcium that interferes with its absorption. Lack of vitamin D3 has been linked to short periods of daylight or low exposure to sunlight. Darker fleeced animals tend to have less vitamin D concentrations, but shearing these animals will increase their exposure to sunlight, thus increasing the vitamin D levels. During the winter period, animals are more susceptible to vitamin D deficiency as the blood levels drop by up to a third of the summer level. Maintaining appropriate vitamin D levels is essential to ensure the proper absorption of calcium and phosphorus from the gut, calcium reabsorption from the urine, and normal bone mineralization.

Reduced activated vitamin D in the blood will promote parathyroid hormone ability, resulting in mobilization of bone calcium and increasing calcium conservation from urine in order to keep the blood calcium homeostasis. This will result in the weakening of bones and the loss of phosphorus. Such diseases are more commonly seen in young, growing animals where the bones are still fragile and under development. Adult animals are usually less susceptible due to a sufficient bone calcium reserve to survive seasonal deficiencies without showing any clinical signs [13].

Crias born to calcium-deficient dams will have a higher risk of developing rickets or angular limb deformities. Newborn animals heavily rely on colostral vitamin D intake because, as a fat-soluble vitamin, it does not cross the placenta well. The efficiency of the colostral uptake depends on the vitamin D status of the mother. Animals born during the fall season are the ones more susceptible to this deficiency due to the reduced sunlight for their first six months of life.

Clinical presentation

Clinical signs are mostly seen right after the dark months and may vary from subtle to severe. The first sign that may be observed is carpal or tarsal angulation, a change in the stride from short striding to a bunny hop gait. Angular deformities are most likely seen in animals under 6

months of age. As the condition becomes more severe, postural abnormalities may appear, such as the animal being in a standing position with its hind legs tucked under the body as a method to shift the weight off the painful carpi. In some cases, we may notice swelling of joints that are painful to the touch. Other signs may include poor growth without any evidence of rickets.

Diagnosis

Affected or at-risk camelids can be diagnosed by obtaining the serum inorganic phosphorus and vitamin D concentrations. A variety of bone abnormalities, such as angular limb deformities, thinning of long bone cortex, and rickets, have been associated with hypophosphatemia. Some of the above abnormalities may be diagnosed by a simple physical inspection, but radiographic examination will present a more defined result. Rickets, which is the widening of the ossification failure of long bones, is linked to seasonal low blood vitamin D levels as it is not only required for bone mineralization but also to facilitate the intestinal absorption of phosphorus.

Treatment and prevention

Vitamin D supplementation will prevent the disease's occurrence and even reverse any pathological changes. The goal is to keep the levels of vitamin D and phosphorus balanced, mainly in growing or pregnant animals, especially during the high-risk months. Vitamin D may be administered through feed supplementation or via injectable solutions. Vitamin D injections at a dose of 1000–2000 IU/kg body weight have shown to maintain adequate vitamin D levels for up to 3 months. Such injections are of high efficacy in growing crias, especially for the ones growing during the winter period. Oral vitamin D intake has a higher daily requirement for camelids compared to other animals as they have a lower bioavailability. Such administrations shall be taken with care, as camelids are prone to vitamin D toxicity.

Patients that do not show any response to vitamin D supplementation may require phosphorus supplementation, which must be balanced with the calcium intake. Monosodium phosphate may be added to the feed, or in severe cases, sodium phosphate may be administered intravenously.

Affected animals will often show clinical improvements within 2–4 weeks after phosphorus supplementation or 1 week after vitamin D supplementation.

Vitamin D toxicity

Epidemiology

Vitamin D is known to be one of the more toxic supplements, with a narrow range between requirement and toxicity. The toxicity levels depend on the length and exposure to the vitamin. If vitamin D (cholecalciferol) is used inappropriately, it will lead to intoxication. Some plants, such as Solanum malacoxylon and Cestrum diurnum, contain high levels of vitamin D. Even though vitamin D toxicity may occur through natural feeding, excess supplementation is usually the main cause of toxicity.

Clinical presentation

Non-specific clinical signs such as anorexia, depression, weight loss, lameness (by dystrophic skeletal changes), cardiac arrhythmias, and renal dysfunction may be observed in cases of vitamin D hypervitaminosis. Other clinical signs related to the respiratory and cardiac functions may be seen if mineralization of the heart, lung parenchyma, and major blood vessels occurs.

Diagnosis

The diagnosis is mainly based on the clinical signs presented. The suggested exposure to excess vitamin D shall be determined. Hepatic and renal vitamin D concentrations will appear elevated. Elevated creatinine, azotaemia, and other blood parameters may be related to the renal dysfunction. Mineralization of soft tissue may be detected via ultrasonography, radiography, or histologically with special stains such as von Kossa-staining.

Treatment and prevention

Treatment is limited beyond supportive care. Kidney function must be maintained. Any sort of vitamin D administration shall be stopped immediately, and the intake of calcium and phosphorus should be reduced to a minimum. The treatment approach is determined by the severity of renal dysfunction.

Dietary management is the primary method of prevention. Proper dosing of vitamin D in crias is also essential to prevent rickets without causing any degree of toxicity. The current level of vitamin D recommended is 30 IU/kg; anything above this dose will not provide any additional benefits to the animal.

4.2.5 Pregnancy toxaemia

This is a rare condition in camelids. Also seen in sheep, it occurs during late pregnancy. However, unlike sheep, it occurs when there is only a single offspring (cria). It is thought that stress causes inappetence, which brings on the disease, which is then self-perpetuating. The less the animal eats, the worse the condition progresses. At first, animals will appear to be slightly depressed and weak and will later appear to be reluctant to move. They then become ataxic, which will lead to recumbency. There may also be neurological signs. Azotaemia, lipaemia, or hyperlipaemia may be visible on a blood sample. There will be an elevated GGT. There will be ketonuria.

Everything possible must be done in order to encourage the animal to eat. In critical cases, a drip line will need to be set up with normal saline, which can then be spiked with glucose and vitamin B complex. Corticosteroids should not be given as they will induce abortion of the foetus, which will also result in the death of the mother. Propylene glycol by mouth is not suggested as there have been reports of toxicity in camelids together with the potential danger of inhalation pneumonia. The prognosis for recumbent patients is poor.

4.2.6 Chronic wasting disease

It is the body condition scores that will need to be examined, as fleeces may deceive the eye. Practitioners should be on the lookout for tuberculosis in any animal showing chronic wasting signs. However, there are many other causes that can be ruled out by a careful clinical examination. It should be remembered that Johne's disease in camelids is not usually associated with diarrhoea, nor is chronic liver fluke infection. Lymphosarcoma is the most common tumour seen in camelids. The diagnosis is typically in the terminal stages due to a failure to show clear clinical signs earlier. There is often weight loss over a period of months, and then lethargy sets in after the animal shows a reduced appetite and becomes rapidly recumbent. Most cases have ascites on abdominal ultrasound. Abdominal masses may be seen to confirm the diagnosis. Peritoneal taps are not helpful, as these tumours rarely shed cells. Obviously, if the tumour invades the liver, the liver enzymes will be raised. Haemangiosarcomas and adenosarcomas will affect the liver and raise levels of live enzymes, but they are extremely rare. Liver abscesses are much more common than tumours. There will be a raised white blood cell count and fibrinogen. The abscess may be visible on ultrasound. Prolonged antibiotic treatment is required, i.e., for 1

month. Liver enzymes may not be markedly affected. On the other hand, with the very rare condition of cholangiohepatitis, the liver enzymes will be greatly increased.

5. Conclusion

This literature review has compared a great number of studies that have been conducted to date with regards to camelid nutrition. From this research, the following conclusions can be made:

- Forage analysis: this is the foundation for building a comprehensive nutrition program that will dictate the need for macro- or micronutrient supplementation. It is ideal to purchase large amounts of high-quality forage in a single lot to ensure a consistent nutritional plan.
- Knowledge of local deficiency and toxicity concerns: educating the owners so as to prevent these conditions from occurring.
- Determination of needed supplementation: a solely foraged diet is not necessarily complete and balanced, especially for animals needing a higher requirement. Supplements can be used to enhance such diets by providing additional energy, protein, minerals, vitamins, or a combination of any of these.
- Maintenance of nutrition quality through proper storage: even a reliable source of feed may not be able to meet expectations due to weather factors altering its nutritional composition. Supplements should also be kept in a protected space, mainly to be out of reach from the animals, as overeating may happen and lead to gastric acidosis and other forms of toxicosis.
- Adequate feeding facilities are required to ensure proper nutrition for all members of the herd.
- Water plays an important role in temperature regulation and provides an aqueous medium to sustain all of the metabolic reactions that occur in the body. The water requirements are determined by body weight, physiological state, level of activity, production state, dietary composition, and environmental conditions.
- Routine body condition scoring: allows us to evaluate the energy status of a nutritional program by grading animals by measuring the amount of subcutaneous fat stores.

From the survey carried out, I can conclude that with a detailed nutritional program (targeted for the geographical area in question), we can likely pinpoint excess, adequacy, or deficiency in camelids of all ages and their reproductive or exercise status.

Having said this, the reluctance of owners to spend money on forage analysis and overall nutritional consultation continues to be prevalent in veterinary practice. Owners do consider their animals valuable, but spending money to ensure their nutritional wellbeing is deemed expensive. The attitude of opting for the lowest-cost nutrition is becoming increasingly popular among camelid owners. In such a scenario, nutritional consultation is probably an even more pressing need.

6. Summary

This literature review tackled the topic of nutritional diseases in camelids. First, a detailed comparison was made between the different camelid species found around the globe. Their feeding requirements and the type of feed used in the geographical region were discussed, as not every country has the same natural feeding options. Typically, a mixture of mixed grasses works best, but not every region may offer the same ratios of such forage.

This study then continues to describe how the geographical areas influence the type of additional supplementation applicable to the animals. This is something that the camelid owners should be made aware of by their local veterinarian.

This review continues by emphasizing how forage should be bought in bulk and stored adequately so as not to compromise the nutritional value of the product and have to re-examine new forage each time it is bought in small amounts.

This research also took a detailed look at several metabolic diseases, common and uncommon, found in camelid species. Different deficiencies and toxicities were evaluated at a macronutrient and mineral level, including their causes, diagnosis, treatment, and prevention.

Having tackled all of the above issues, the limitation is always set by the economy and the owner's unwillingness to spend money on their herd. Metabolic diseases are a constant pressing matter in these ruminants, and all these can be prevented when owners agree to undergo nutritional consultations.

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Thesis progress report for veterinary students

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Thesis title: Nutritional Disease of Camelid Species – Literature Review

	Timing			Topic / Remarks of the supervisor	Signature of the supervisor
	year	month	day		
1.	2022	02	10	Supplying of collection of literature	
2.	2022	03	08	Discussion of the chapters of thesis	
3.	2022	04	13	Correction of the 1st chapter's manuscript	
4.	2022	04	28	Consultation about the progress in the preparation of thesis	
5.	2022	05	11	Consultation about the 2nd and 3rd chapters	

Grade achieved at the end of the first semester: .....good (4).....

Consultation – 2nd semester

	Timing			Topic / Remarks of the supervisor	Signature of the supervisor
	year	month	day		
1.	2022	09	15	Consultation about the 4th chapter	
2.	2022	10	04	The 1st sketch is prepared	
3.	2022	10	28	Corrected sketch is applied	
4.	2022	11	17	Correction of the manuscript	
5.	2022	12	13	Final version of the thesis is prepared	

Grade achieved at the end of the second semester: excellent (5)

The thesis meets the requirements of the Study and Examination Rules of the University and the Guide to Thesis Writing.

I accept the thesis and found suitable to defence,

Dr. Éva Cadar

.....

signature of the supervisor

Signature of the student: .....

Signature of the secretary of the department: .....

Date of handing the thesis: 22. March 2023

I hereby confirm that I am familiar with the content of the thesis entitled
Nutritional Diseases of Camelid Species – Literature Review
written by Raoul Stafrace which I deem suitable for submission and defence.

Date: Budapest, 22. March, 2023

Dr. Éva Cenkvári

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