Nutritional therapy in the management of systemic diseases in dogs

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Nutrition of sick animals is an important factor in the management of many systemic diseases in dogs. In this paper nutritional management of commonly encountered systemic diseases of dogs from the practitioners point is discussed.

Liver Disorder:
Nutritional Management:
Nitrogen Metabolism:

Dietary therapy must aim to provide adequate protein to meet the requirements for hepatic regeneration and repair, and yet minimise nitrogenous waste products of protein catabolism. The type and quantity of protein must be modified to avoid encephalopathy. Hepatic encephalopathy (HE) is of great concern in severe liver dysfunction and portosystemic shunts. Almost all the amino acid levels in plasma increases with exception of branched chain amino acids (BCAA) and arginine.

The BCAA i.e. valine, leucine, isoleucine are essential in the diet and are metabolised outside the liver, predominantly in muscle. BCAA supplementation may have therapeutic benefits in the management of HE by competitively inhibiting transport of aromatic and sulphur containing amino acids across the blood brain barrier.

In severe liver disorders a highly digestible, protein restricted, high biological value (more than 75) protein diet is recommended (e.g. Cottage cheese). Dairy proteins have a high biological value and appropriate BCAA : AAA (Aromatic Amino Acids) ratio but are low in arginine. Arginine is an important component of urea cycle. Fish meal protein is of high biological value which is highly palatable but high in purine. Alternative protein sources are vegetable proteins (soy flour, corn grits, rice etc,) which have a favourable amino acid profile and a low content of methionine and mercaptans.

Ideally the diet should be based on highly digestible carbohydrate (e.g. Rice, pasta) be adequate in vitamin content and highly palatable to combat inappetance associated with hepatic disease.

Lipid requirements:

Fat is restricted where steatorrhoea is clinically evident. Fatty acids may aggravate HE by the direct action of short and medium chain length fatty acids on the CNS and indirectly by reducing the conversion of ammonia to urea aggravating the post-prandial hyperammonaemia.

Carbohydrate:

Highly digestible carbohydrate, such as rice and pasta are absorbed in the proximal gastro intestinal tract and provide a non encephalogenic energy source. Fibre may increase bacterial fermentation, decreases palatability and increases nitrogenous losses due to the abrasive desquamation of epithelial cells.

Vitamins and Minerals:

Vitamin and mineral deficiency may occur due to poor appetite, decreased intestinal absorption and increased demands due to catabolism and regeneration. Water soluble vitamin supplementation at maintenance requirements is enough. However, ascorbic acid supplementation at levels of upto 25 mg/day to compensate for decreased synthesis. Vitamin E supplementation at 500 mg/day provides an important hepatocellular protective effect against copper toxicity and lipid peroxidation injuries. Vitamin A supplementation is dangerous due to risk of synergism between Vit A and cytotoxins in provoking hepatocyte damage. The Bedlington and White Terriers are prone to disorders of hepatic storage of copper leading to hepatotoxicity. This can be managed by restricted levels of dietary copper (0.8 mg/1000 Kcal ME, avoid feeding of offal

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7 – 14 days). A rapid reduction in sodium intake in chronic renal failure may result in extracellular fluid volume contraction and hypotension.

Dietary Lipid:

Though hyperlipidaemia is present in many dogs with renal dysfunction, adverse effects of these have not yet been established. The production of eicosanoids (prostaglandins, thromboxanes, leukotrienes and prostacyclin) by the kidney is enhanced in chronic renal disease and plays a critical role in the genesis of glomerular hypertension. The method to modify this is to feed a diet rich in n-3 fatty acids (e.g. Fish oil) to lower renal eicosanoid production and to shift the production from the usual arachidonic acid metabolites to a different series of eicosanoids. Dietary supplementation with plant oils will provide good quantity of linoleic acid (n-6) the precursor of arachidonic acid and this may actually enhance renal production of eicosanoids.

Potassium:

In hypokalaemic animals i.e. dogs with polyuric renal failure additional supplementation of potassium can be done (1-6 mEq/Kg body weight/day). In hyperkaelemic animals restrict dietary potassium and appropriate medical therapy has to be instituted.

Energy intake:

Inadequate intake of protein and/or energy in renal insult will reduce the renal compensatory response and may prevent the regeneration of adequate renal tissue to support life. Malnutrition may be further compounded by vomiting, malabsorption, maldigestion and disorders of protein and carbohydrate metabolism. Non uremic dog should initially ingest approximately 75 Kcal/Kg body weight/ day (Vegetable oils 9 Kcal/g, Margarine 7 Kcal/g, Cream 4 Kcal g). Carbohydrate sources of energy such as honey, jelly or sugar contain about 3.5 Kcal/ml.

Urolithiasis:

Struvite urolithiasis is the most common type of urolith and the mineral found in these uroliths of dogs is magnesium ammonium phosphate or struvite.. Pure struvite uroliths are uncommon. Calculolytic diets have been formulated to reduce urine concentration of urea, phosphorus and magnesium. Diets contain reduced quantities of high quality protein, phosphorus and magnesium are supplemented with sodium chloride to stimulate thirst. These diets should not be given to patients with heart failure, nephrotic syndrome, hypertension and to growing dogs. To prevent recurrence of calcium oxalate uroliths, a diet moderately restricted in protein, calcium oxalate and sodium is recommended. The diet for urate urolithiasis is to reduce urine concentration of ammonium and urate. A protein restricted diet used for moderate to severe renal insufficiency is quite beneficial.

Congestive heart failure:

In congestive heart failure, in addition to therapy and sodium restriction, specific nutrients of particular concern are potassium, magnesium and taurine. Carnitine is a conditionally essential nutrient in some dogs with myocardial disease.

Potassium deficiency is a potential problem when loop diuretics are used. Hypokaelemia produces electrocardiographic abnormalities, generalised muscle weakness and increases myocardial sensitivity to digoxin contributing to arrhythmia. Magnesium deficiency is more common in congestive heart failure than recognised. Magnesium plays a key role in regulating the movement of ions across

cell membranes. In chronic magnesium deficiency, intracellular potassium decreases, which inturn contributes to hypokalemia and this is not responsive to potassium therapy unless magnesium is supplemented. Magnesium deficiency is difficult to diagnose, as serum magnesium is a poor indicator of magnesium status. Digitalis also promotes magnesium loss by inhibiting renal tubular reabsorption of magnesium. In addition dietary sodium restriction and decreased cardiac output lead to increased aldosterone secretion, which subsequently contributes to further urinary losses of magnesium.

Taurine is not recognised as an essential nutrient in dogs. However, datas suggest that treatment of cardiomyopathy with both taurine(500 mg twice daily orally) and L-carnitine (1.0 g twice daily orally) is quite beneficial. Long chain fatty acids are major sources of energy for cardiac and skeletal muscle. Carnitine plays a critical role in transporting long chain fatty acids across mitochondrial membranes. Carnitine has been claimed to be beneficial in treatment of dilated cardiomyopathy. The recommended dose is 50 – 200 mg/Kg orally every 8 hours as L-carnitine. Other forms of carnitine i.e. D-carnitine or D, L-carnitine are contraindicated.

Skeletal Diseases:

A deficiency but more frequently an excess of one or more nutrients may play an important role in a variety of skeletal diseases.

Osteochondrosis:

It is a disturbance of endochondral ossification and is characterised by an abnormal maturation of chondrocytes and thus a delay in cartilage mineralisation. This condition is more common in males than females. It is commonly seen in Great Dane, Labrador and Golden Retriever, Newfoundland and Rottweiler. Endochondral ossification disturbance in articular cartilage results in osteochondritis dissecans (OCD). In OCD part of the articular cartilage becomes detached and may be fragmented, mineralised or even ossified. OCD lesions are most commonly seen in shoulder, stiffle, elbow and hock joints. OCD results due to chronic intake of too much of a balanced food or of food with calcium with or without other constituents. OCD is characterised by lameness, pain upon extension or flexion of the joint, joint effusion and subchondral sclerosis.

Dietary correction at an early stage may help in the spontaneous resolution. Dietary modification will not normalise OCD cases in which there is detached cartilage or where a more severe curvature of radius exists. Dietary correction should include a decrease in intake of energy (protein, fat, carbohydrate), calcium and vitamins to the minimal requirement.

Decreased skeletal remodelling:

This occurs as two separate entities – Canine Wobbler syndrome (commonly seen in Great Danes, Mastiffs, Irish Wolfhounds and Dobermanns) and Enostosis (also called as Panosteitis eosinophilia) which is seen in variety of dogs at young age and is particularly seen in German Shepherd dogs. The cause of this is chronic excessive calcium intake, which results in high calcium absorption in young dogs of large breeds. Calcium is not significantly excreted in urine or feces but is mainly routed to the bones in these dogs.

Wobbler syndrome:

Great Danes fed with a high calcium show a delayed expansion of the cervical vertebral canal in proportion to the growth of the spinal cord. Compression of spinal cord causes myelin degeneration.

Enostosis: In this a decreased endosteal

osteoclastic resorption together with an increase in new periosteal bone formation has been observed. The nutrient canals and foramina of the cortex are often abnormal in shape. This causes oedema and eventually fibrosis in the medullary cavity. Oedema may also extend throughout the cortex and underneath the periosteum causing a loose periosteal attachment or excessive lamellar bone formation. Early dietary correction may halt the process of disproportionate remodelling of the skeleton.

Dietary correction includes a decrease in the quantity of calcium and vitamins to the minimal requirements. It may be beneficial to feed low calcium diet for a limited period to hasten osteoclast activity.

Excess vitamin in the diet:

Massive intake of vitamin D can cause hypercalcemia, together with hyperphosphataemia, anorexia, polyuria, vomiting, muscle weakness and lameness. Vitamin D intoxication is characterised by mineralisation of soft tissues including blood vessels, alveoli and renal tubules together with pathological changes in the gastrointestinal tract and the heart. Excess vitamin D can cause deleterious effects on skeletal development and probably also on the kidney function also.

Hip Dysplasia:

In this condition additional calcium supplementation may not be beneficial. Excessive calcium intake decreases maturation of hip joint conformation as well as of the vulnerable cartilaginous template of the skeleton. This may coincide with overloading of the hip joint and may play a significant role in deformity of the hip joint at an early age. ❑

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not intended to treat or cure the condition but to aid positively in the treatment process and to improve the quality of life. The patient should receive high quality food through oral or enteral or parenteral route. The diet should provide:

- High quality protein
- Highly digestible protein
- Fat as preferential fuel source
- Nutrient dense diet in small volume
- Moderately fermentable fiber
- Optimum ratio of omega 3 to omega 6 fatty acid
- For nutritional management of inflammation

The diet should be highly palatable because during sickness the appetite is depressed and the patient has anorexic tendencies.

Conclusion

Critically ill dogs whose altered nutritional needs are met early, often respond favourably to therapy, while malnourished patients have increased morbidity and mortality. Hence a desirable nutritional support plan for sick animals be worked out by assessing the nutritional needs and the condition of the patient to ensure ease of administration. The animals should be fed to condition by frequent feeding of small volumes. The diet must provide an optimal caloric distribution with 55% of metabolizable energy from fat, 30% from protein and only, 15% from carbohydrates. By providing a nutrient dense diet, the veterinarian can be assured of providing optimal nutrition that can have a substantial and beneficial impact on the patient's recovery.
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