

Clinical and dietary management of systemic disorders in pet animals

Deepa Ananth

cope of pet animal therapeutics greatly depends upon proper management of disease condition. Veterinarians and clinical nutritionists need to provide required advice to their clients about the disease and suitable diet.

Nutritional assessment, is the first step to identify malnutrition. If it is not present initially, the animal should be evaluated periodically to ensure that the malnutrition does not develop as a sequele to the disease, administered drug, food aversion or deprivation.

Dr.Deepa Ananth, MVSc Veterinary Surgeon, Veterinary Polyclinic, Wadakkancherry, Trichur Nutritional assessment techniques include detailed history taking and physical examination, measurement of body weight, assignment of body condition score and ex-

amination of serum albumen concentration. Moreover the given feed and its acceptance

or rejection will help in determining the need for dietary situations, and help in planning a dietary recommendation. Other factors such as age, physiological status and level of activity should be considered while recommending and formulating diet. Adverse effects of certain drugs may be ascertained.

In the case of management of critically ill patients showing clinical signs of shock, blood volume must be restored first. The volume of fluid required is calculated based on body weight. The most common electrolyte abnormality noticed in veterinary practice is hypokalemia with serum K+ level going < 3 m Eq. per litre as against normal values ranging between 4.1 - 5.5m Eq/l in dogs and in cats 3.7 - 5.4 M Eq/l. The most common cause for hypokalemia is decreased feed intake, increased urinary or gastrointestinal loss, compartmental shifting of potassium into the intracellular space which is affected by catecholamines, insulin and metabolic acidosis. Renal potassium loss occurs during vomiting, cirrhosis, renal failure, and hyperaldosteronism and during therapy using steroids, administration of potassium free crystalloid or glucose and ACE inhibitors. Hyperkalemia occur during massive tissue injury, digitalis toxicity, lack of insulin, urethral obstruction, adrenal insufficiency and when given non steroidal anti inflammatory agents and potassium sparing diuretics, ACE inhibitors and heparin. Decreased heart rate, P wave amplitude and increased QRS duration are noticeable electrocardiographic indicators of hyperkalemia.

Acid base disturbance causes abnormalities in sodium and potassium balance of 25:1 and help in diagnosis of hypo/hyperadrenocortism. Change in arterial pH, Bicarbonate concentration, mineral and organic metabolic acidosis, osmolarity, hormonal activity and metabolic excretory function of liver and kidney also predispose this condition.

Magnesium is the second most abundant intracellular cation and is needed for ATP production and energy metabolism. It is used as adjuct therapy in car-





diac arrhythmia, cardio pulmonary arrest and refractory hypokalemia.

Nitrogen: - Wertheimer et al (1919) has reported increased urinary excretion of nitrogen during metabolic stress. David Cuthbertson observed that source of urinary nitrogen was froms keletal muscle and it is primarily excreted as urea. Thus hypermetabolism seen after the traumatic shock stage, are clinically characterized by evidence of endogenous protein catabolism by increased tissue wastage.

The metabolism due to trauma is different from metabolism of fasting animal as the latter adapts it by reducing activity and thereby energy reserves and body protein are conserved. This is achieved by using fat as fuel when body glucose reserves are depleted. Moreover gluconeogenesis occur in liver from glycerol, lactate and aminoacids derived from breaking down of skeletal muscles and visceral proteins. But in injured or sick animals, endogenous protein catabolism is accelerated beyond the requirement of gluconeogenesis and the amino acids are utilized for energy. The major source of peripheral release of amino acids after injury is skeletal muscles. These amino acids are utilized for synthesis of proteins required for wound healing and host defense mechanisms. It is estimated that excretion of each gram of nitrogen represents loss of 30g of lean tissue. Such drastic loss should be replenished immediately to enhance the rate of wound healing and other recovering processes. Exogenous supplementation of energy and aminoacids would reduce the rate of catabolism to some extent.

Dietary management of liver disorders:-

The nutritional and biochemical effect of hepatic disease affect protein, carbohydrate and fat metabolism. In health, protein regulatory events including amino acid storage, deamination, transamination and aminoacid synthesis all occur in liver and thus it has an important role in regulating plasma concentration of metabolically important aminoacids. The alanine is important for gluconeogenic process and glutamine is obligatory fuel for intestine and other rapidly dividing cells. Biosynthesis of fatty acids, triglycerides, metabolism of fat, storage of A, D, E, K, B_{12} and C vitamins, preliminary activation of D precursors, storage of Fe, Cu, Zn, Mn and Mg all occur in the liver.

Diet therapy must aim in providing adequate protein required for hepatic regeneration and repair, reduced and nitrogenous waste product. Fat should be restricted and highly digestible carbohydrate and low fibre diet should be provided. Metronidazole and lactulose can be given to counteract hepatic encephalopathy. Ascorbic acid supplementation at levels upto 25 mg/day to compensate for decreased synthesis. Vitamin E supplementation is necessary to reduce lipid peroxide injuries, copper and Iron toxicity. Copper chelators like trientine and zinc reduces copper toxicity. Ascites could be managed by restricting sodium intake.

Renal diseases

Dietary management of chronic renal failure should focus on minimizing clinical signs and slowing down the progression of renal failure.

In chronic renal failure the patients would have history of polyurea, polydypsia, small irregular kidneys and non-regenerative anaemia. They often have poor body condition and weight loss. The dietary recommendation of 20-30 per cent of protein per ME is ideal. High protein and protein malnutrition causes generation of uremic toxins. Restriction of phosphorus is more important in preventing renal secondary hyperparathyroidism. Phosphorus binders like calcium carbonate, acetate, lactate and gluconate could be supplemented to reduce pH in blood. Sodium intake should be restricted gradually. Potassium should be supplemented in polyuric renal failure patients. Urinary acidifiers should not be given to patients having chronic renal failure. Plant oils which contains w-6 fatty acids hasten the decline of renal function and hence should be avoided.





Congestive heart failure

Patients with CHF will have fluid retention and cardiac output, which may lead to hypoperfusion of kidneys leading to azotonemia & renal failure. Whereas in- patients with right sided CHF, there will be hepatic congestion and portal hypertension. Left-sided CHF interfere with normal gas exchange, which leads to respiratory acidosis. Diuretics may cause loss of potassium, magnesium, calcium and B vitamins which contribute to digitoxin induced arrhythmia. The therapy includes diuretics, digitoxin, vasodilators, angiotensin converting enzyme inhibitors in addition to cage rest, oxygen and dietary sodium restriction. Nutritional management of CHF must also take into consideration obesity / cachexia of patient. Specific nutrients of particular concern include potassium, magnesium, which are excessively lost due to therapy along with taurine in case of cats. Carnitine, which plays an important role in transporting long chain fatty acids across mitochondrial membranes, is considered as coronary essential nutrient. The requirement can be up to 200 mg/kg orally, thrice daily. Patients with CHF have reluctance to eat due to lung congestion. High quality diets would be of some use.

Conclusion

Clinical management of disease is equally important as therapy. Management of critically ill patient should focus on restoration of water and electrolyte balance. Management of liver disorders include providing a diet with low fat, high carbohydrate and good quality protein for repair and regeneration of hepatic cells. It should also be supplemented with vitamins, zinc and branched chain fatty acids. Dietary management of chronic renal failure include, restriction of sodium and phosphorus counteracting hypokalemia, and supplementation of optimum protein along with carbonates and Co-3 fatty acids to reduce tissue damage. Congestive heart failure patients could be given a diet with balanced energy and protein according to the condition of the patient. Supplementation of Potassium, Magnesium, Taurine and Carnitine would be beneficial.

PHILIPPINES MAY OPT FOR INDIAN BEEF

The Philippines may buy more beef from India, Australia, New Zealand and other sources next year following a ban on the import of European meat due to the mad cow scare. Besides Europe, other major suppliers of beef are India, New Zealand and Australia.

-The Hindu Business Line

NO IMPORT OF GENETICALLY MODIFIED FOOD OCCURRED

The Union Health Ministry has reportedly decided to ask the customs department to ensure that no food items likely to have any adverse impact on human and animal health, are allowed to be imported in to the country. The Ministry seeks to propose a suitable amendment to the existing prevention of Food Adulteration (PFA) Act to ensure more food safety.

- Indian Dairyman

JIVA 38