

CLINICO-THERAPEUTIC MANAGEMENT OF NERVOUS KETOSIS IN JAFFARABADI BUFFALO (*BUBALUS BUBALIS*)

Bhavika R. Patel* and Jayesh S. Patel

ABSTRACT

This short communication describes the clinical signs, diagnosis and successful therapeutic management of nervous ketosis in a Jaffarabadi buffalo.

Keywords: Jaffarabadi buffalo, hypoglycemia, Rothera's test, nervous ketosis, 25% dextrose

INTRODUCTION

Ketosis is condition characterized by abnormally elevated concentrations of ketone bodies acetoacetate, acetone and 3-hydroxybutyrate in the body tissue and fluids (Taylor, 1994). Ketosis becomes a disease condition only when the absorption and production of ketone bodies exceeds their use by the ruminant as an energy source, resulting in elevated blood ketone, free or non-sterified fatty acid and decreased blood glucose (Smith, 2002). In dairy animals, ketosis is a metabolic disorder which may appear as a primary disease or in association with other pathological conditions (secondary ketosis) and occurs mainly in animals during winter and spring months. Negative energy to hypoglycemia and ketonemia

are primary cause of the disease as maintenance of adequate concentration of glucose in the blood is critical in high yielder during first few weeks of parturition for the regulation of energy metabolism. Nervous signs are seen in about 10% cases of primary ketosis (Foster, 1988). Major economic losses are considerable due to decrease milk yield, failure of the animals to return to the normal potential even after recovery (Lean *et al.*, 1994), cost of treatment, decreased market value of animal (due to sever wasting and decreased milk yield) and occasionally from death and disposal of animal. In India ketosis mostly occurs in secondary form due to unhygienic farm conditions, as a result the cases of pure primary ketosis, that to nervous ketosis either go unnoticed or not been diagnosed or differentiated properly. Hence the present case report aims to provide information about the successful diagnosis and management of a case of nervous ketosis in a she buffalo.

HISTORY AND CLINICAL OBSERVATION

An eight year old she Jaffarabadi buffalo in her third lactation with the complaint of grain refusal, collapse and disorientation was examined. Four weeks back, the buffalo had

calved uneventfully. As per the history, the milk yield reduced drastically from eight liters to one liter along with wasting. The buffalo pass scanty, hard mucous coated faeces and had a tendency to sleep while standing with stretched head and neck posture. However, since last 3 days, the buffalo exhibited nervous signs lasting for 1 to 2 h with the frequency interval of 10 to 12 h. This nervous episode included pica, head pressing, apparent blindness, excessive licking of body and isolated muscle twitching. Physical examination included normal temperature (102°F), mildly elevated respiration rate (62/minute) and pulse rate (100/minute), lethargy, slightly dehydrated, hypomotile rumen, ketotic breath odour and an abnormal attitude. Upon biochemical investigation the blood glucose was found to be reduced to 26 mg/dl and the urine was found to be positive for ketone bodies by Rothera's test. After differentiating it from listeriosis, rabies and plumbism, the case was diagnosed as nervous ketosis.

TREATMENT AND DISCUSSION

Treatment of this buffalo include intravenous administration of 3000 ml of 25% dextrose and 15 ml neuroxin intravenous, 5 ml of triamcinolone acetonide intramuscularly and 200 ml gluca-boost once a day for three days. No further veterinary care was required after initial therapy. However the owner was advised to feed two kg of jaggary daily for at least five days. On second day, the owner reported marked improvement and after five days complete recovery was reported. The case was tentatively diagnosed as nervous ketosis especially on the basis of clinical signs: anorexia, hypoglycemia, decreased milk production and nervous signs. The nervous signs are thought to

be caused by the production of isopropyl alcohol a breakdown product of acetoacetic acid in the rumen, although the requirement of nervous tissue for glucose to maintain normal function may also be a factor in these cases (Radostits *et al.*, 2007).

Diagnosis was confirmed by blood glucose level 26 mg/dl and Rothera's test. Although at first instant the disease can easily get confused with rabies, listeriosis and plumbism (lead poisoning) due to similarity in one or more clinical signs. However, following tactful anamnesis and through clinical and laboratory investigations it can easily be differentiated. As in later, there is history of dog bite, ascending paralysis, always fatal termination (Rabies), fever, circling, abortion in last trimester (listeriosis) and plumbism occurs in all age group mostly young calves and tremors, rapid death unless treated, source of lead in the diet and no response to glucose therapy are principal manifestations (Radostits *et al.*, 2007). Primary ketosis is due to primary energy deficiency in high milk producers. The heavy demand of energy in early lactation can cause persistent hypoglycemia with resultant decrease in insulin production and subsequent lipid mobilization, the resultant hepatic lipidosis leads to increase in hepatic ketogenesis, hypoglycemia and hyperketonemia (Wotton, 1992). The low concentration of blood glucose could be attributed to the negative balance reflecting greater demands for glucose in the mammary gland (Anantwar and Singh, 1993). Hypoglycemia, ketonemia and ketoneuria are rather constant and a characteristic feature in ketosis (McSherry *et al.*, 1960). The severity of clinical ketosis in cattle follows rather closely the degree of hypoglycemia (Boddie, 1935; Roepke, 1942). In some cases of ketosis, however, normal or even high blood sugar has been reported (Boddie, 1935). The hypoglycemia associated with ketosis and curative effect of

parental glucose, is due to faulty carbohydrate metabolism responsible for ketosis (Hupka, 1928). Earlier workers, recognizing the need of ketotic cows for available carbohydrate, advocated the oral use of sugar and molasses to supply this need (Fincher, 1936; Roepke, 1942; Shaw and Ross, 1941). It has been shown, however, that as these products are first converted to fatty acids, some of which are ketogenic, this approach appears to be contraindicated (Adler and Dye, 1955; Holmes, 1952; Johnson, 1951a; Roberts, 1954). Johnson (1951b) and others (Roberts, 1954; Schultz, 1952) advocated the use of oral carbohydrate precursor other than sugar in treating and preventing ketosis such substances are propylene glycol, sodium propionate and glycerol.

REFERENCES

- Adler, J.H. and J.A. Dye. 1955. On the mechanism of ketogenesis in ruminants. *Cornell Vet.*, **6**: 58-63.
- Anantwar, L.G. and B. Singh. 1993. Epidemiology, clinic-pathology and treatment of clinical ketosis in buffaloes. *Indian Vet. J.*, **70**: 152-156.
- Boddie, J.F. 1935. Some aspect of ketosis in dairy cows. *Vet. Rec.*, **15**: 1539-1546.
- Fincher, M.G. 1936. Clinical observation in acetonemia in cattle. *Cornell Vet.*, **26**: 142-155.
- Foster, L.A. 1988. Clinical ketosis. *Vet. Clin. N. Am.-Food A.*, **4**: 253-265.
- Holmes, J.R. 1952. Carbohydrate metabolism in bovines III. The effect of administration of glucose by mouth to normal cattle of various ages. *J. Comp. Pathol.*, **62**: 41-53.
- Hupka, E. 1928. Die azetonemia der rinder. *Deut. Tieraztle Wochscher*, **36**: 98p. In McSherry, B.J., D.C. Maplesden and H.D. Branion. 1960. Ketosis in cattle-A review. *Can. Vet. J.*, **1**: 208-213.
- Jhonson, R.B. 1951a. The relative rates of absorption of the volatile acids from the rumen and their relationship to ketosis. *Cornell Vet.*, **41**: 115-121.
- Jhonson, R.B. 1951b. New method of treating ketosis a preliminary report. *North Am. Vet.*, **32**: 327-332.
- Lean, I.J., M.L. Bruss, H.F. Troutt, J.C. Galland, T.B. Farver, J. Rostami, C.A. Holmberg and L.D. Weaver. 1994. Bovine ketosis and somatotropin: Risk factors for ketosis and effects of ketosis on health and production. *Res. Vet. Sci.*, **57**: 200-209.
- McSherry, B.J., D.C. Maplesden and H.D. Branion. 1960. Ketosis in cattle-A review. *Can. Vet. J.*, **1**: 208-213.
- Radostits, O.M., C.C. Gay, K. Hinchcliff and P.D. Constable. 2007. *Veterinary Medicine. A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats*, 10th ed. Saunders Elsevier, Philadelphia, USA.
- Roberts, S.J. 1954. Ketosis-parturient paresis complex. *J. Am. Vet. Med. Assoc.*, **124**: 368-372.
- Roepke, M.H. 1942. Physiologic and metabolic aspect of acetonemia in cattle. *J. Am. Vet. Med. Assoc.*, **100**: 411-415.
- Schultz, N.H. 1952. Treatment of ketosis in dairy cattle with sodium propionate. *Cornell Vet.*, **42**: 148-155.
- Shaw, J.C. and C.P. Ross. 1941. Glucose therapy in ketosis in cattle. *J. Dairy Sci.*, **24**: 503.
- Udall, D.H. 1954. *The Practice of Veterinary Medicine*, 5th ed. Menasha, Wisconsin, George Banta Publishing Company.

Smith, B.P. 2002. *Large Animal Internal Medicine*,
10th ed. Mosby. Inc. St. Louis Missouri,
USA.

Wotton, P. 1992. Nervous ketosis. *Can. Vet. J.*,
33(3): 194-194.

Taylor, E.J. 1994. *Dorland's Illustrated Medical
Dictionary*, 28th ed. W.B. Saunders,
Philadelphia, USA.