

## HISTOPATHOLOGICAL CHANGES IN DIFFERENT VITAL ORGANS OF BUFFALO CALVES IN EXPERIMENTAL HYPOMAGNESEMIA

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### ABSTRACT

Experimental hypomagnesemia was successfully induced in buffalo calves by administering potassium chloride and citric acid @ 1.3 and 1.1 g/kg body weight, respectively as 15 per cent solution given intraruminally daily until the development of clinical illness. Histopathological examination of different organs in buffalo calves died of experimental hypomagnesemia revealed severe congestion, and petechiae and ecchymotic hemorrhages. Dilated sinusoids with degenerative changes in hepatocytes were observed in liver. Histopathological alterations in lungs included emphysematous changes with congestion and interalveolar tissue as well as alveoli were filled with edematous fluid. Noticeable changes in lymph nodes were edema, depletion of lymphocytes, presence of immature cells and haemorrhages in the cortical region. Hyperplasia of glomerular tufts with cloudy swelling of tubular epithelium were recorded in kidneys.

**Key words:** Hypomagnesemia, buffalo calves, histopathology

Hypomagnesemia is a magnesium ions deficiency of blood and cerebrospinal fluid that can occur in acute and chronic or sub-clinical forms. Hypomagnesemia is a disease of economic importance (Harris, *et al.*, 1983) because of high fatality rates in all classes of ruminants and its highest incidence in lactating animals affecting the production. Although, the conditions under which disease can arise are fairly known but pathological alterations in different body tissues caused by hypomagnesemia are still not certain.

### MATERIALS AND METHODS

The present study was conducted on 8 healthy buffalo calves, which were 6-12 months old. Deworming of calves was carried out with 1 per cent solution of Ivermectin @ 1 ml/kg body weight subcutaneously one week before experiment. These calves were examined clinically and blood, faeces and urine samples were analyzed to rule out parasitic or other conditions. These calves were divided in two groups of 4 animals each. Calves of group-I served as control and experimental

hypomagnesemia was induced in buffalo calves of group-2 by administering potassium chloride and citric acid @ 1.3 and 1.1 g/kg body weight, respectively as 15 per cent solution given intraruminally daily until the development of clinical signs of hypomagnesemia. Blood magnesium level of these calves decreased from initial level of  $2.75 \pm 0.39$  mg/dl to  $1.26 \pm 0.19$  mg/dl before death. Gross lesions in various visceral organs of buffalo calves died of hypomagnesemia were recorded and representative pieces of different organs were collected in 10 per cent formal saline for histopathology. The tissues were processed for paraffin embedding using ascending grades of alcohol as the dehydrating agent and cedar wood oil as the clearing agent. Sections of different tissues were cut at 4-5  $\mu$  thickness and stained with routine hematoxylin and eosin stain (Luna, 1968).

### RESULTS AND DISCUSSION

All the calves of group-2 died within 7 to 9 days after the start of experiment. These calves also showed different signs of clinical hypomagnesemia like depression, erect ears,

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shaking of head, hypersensitivity to external stimuli, backward carriage of ears, opisthotonus and occasionally muscle fasciculations of hind legs and muzzle prior to death. At necropsy, the calves died of hypomagnesemia revealed congestion, petechial to ecchymotic haemorrhages in liver, heart, lung and mesenteric lymph nodes.

The sections of heart revealed severe congestion and haemorrhages in myocardium (Fig. 1) however, no histopathological changes were noticed in endocardium and pericardium. Dilated and congested sinusoids with degenerative changes in hepatocytes were observed in liver (Fig. 2). Histopathological changes in lungs included emphysematous changes, congestion, and interalveolar tissue as well as alveoli were filled with edematous fluid (Fig. 3). Noticeable changes in lymph nodes were edema, depletion of lymphocytes, presence of immature cells and haemorrhages in cortical region (Fig. 4). Hyperplasia of glomerular tufts with cloudy swelling of tubular epithelium were recorded

in kidneys. Different histopathological findings observed on development of hypomagnesemia were also noticed by Bohman *et al.* (1969), Oshima *et al.* (1973), May *et al.* (1976), Haggard *et al.* (1978) and Sarode *et al.* (1991). Tissue calcification in clinical and experimental cases of hypomagnesemia observed by Oshima *et al.* (1973) and Haggard *et al.* (1978) respectively, could not be observed in the present study which may be due to short duration of experimental model. The rarefication of lymphocytes, presence of immature cells in lymph nodes and cloudy swelling of tubular epithelial cells in kidneys were indicative of early degenerative changes.

The occurrence of hemorrhages in hypomagnesemia has already been pointed out by earlier workers (Blaxter *et al.*, 1954, Rook, 1963) in experimentally and naturally affected animals which might be induced by the vascular contractions, the decline in magnesium content of tissue fluid makes the blood vessels contract excessively (Oshima *et al.*, 1973).

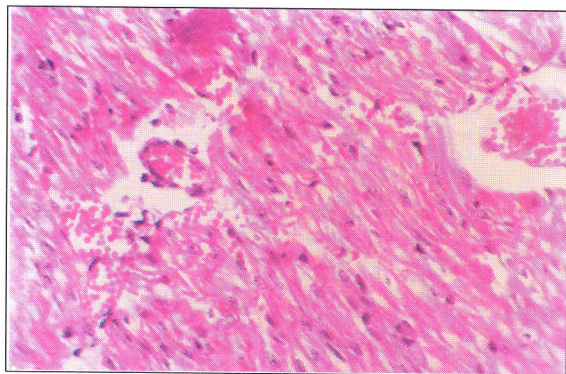


Fig.1. Congestion and hemorrhages in myocardium.  
(H. & E. x 132)

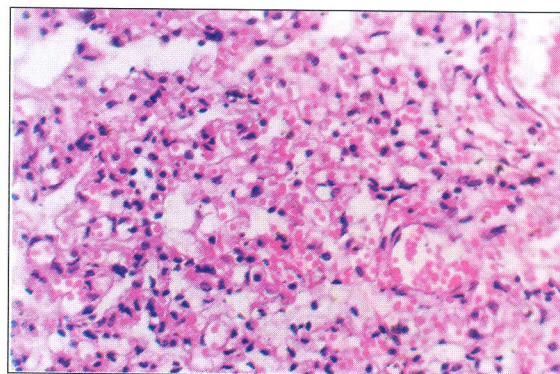


Fig.3. Congestion and edematous fluid in alveoli and interalveolar tissue.  
(H. & E. x 132)

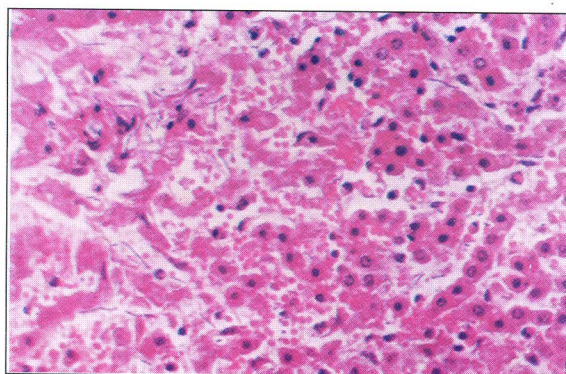


Fig.2. Congested sinusoids with degenerative changes in hepatocytes in liver.  
(H. & E. x 132)

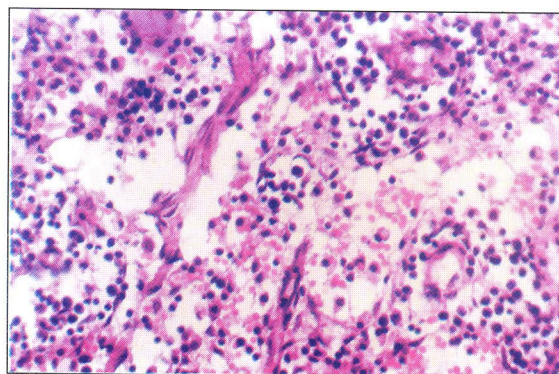


Fig.4. Depletion of lymphocytes and hemorrhages in cortical region of lymph node.  
(H. & E. x 132)

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