

Salt Toxicity in a Buffalo Heifer

Kanwarpal Singh Dhillon^{1*} and Bikramjit Singh²

Krishi Vigyan Kendra, Amritsar, Punjab Agricultural University, Ludhiana-141 004, Punjab, India.

¹Assistant Professor

²Associate Director

Abstract

Sodium chloride is an essential dietary component for cattle, but excessive intake or water restriction can cause salt poisoning and hypernatremia, leading to severe neurological dysfunction. This report describes a case of salt toxicosis in one year old buffalo heifer that developed acute neurological signs after ingesting 1.5 kg of common salt without access to drinking water. Diagnosis was based on history, clinical presentation, and elevated serum sodium and chloride levels. The heifer was successfully treated through gradual rehydration, isotonic fluid therapy, and supportive management.

Keywords: buffalo, salt toxicity

Salt (sodium chloride) plays a critical role in bovine nutrition by maintaining osmotic balance, blood viscosity, acid-base equilibrium, and gastric hydrochloric acid secretion (Sastry and Rama Rao, 2001). Cattle diets generally contain 0.5-1% salt. However, excessive intake, particularly under conditions of restricted water availability, can lead to salt toxicosis. Errors in feed formulation or inadequate mixing can also contribute to excessive dietary salt (Aiello, 2016). Although salt poisoning is well documented in pigs and poultry, it is less frequently reported in cattle and buffaloes (Radostits *et al.*, 2007; Smith, 2020). This article describes successful management of salt toxicosis in a buffalo heifer

A year old buffalo heifer from Daddiyān village, Amritsar of Punjab, India was presented with acute neurological signs, including lateral recumbency, profuse salivation and paddling of limbs (Fig. 1). According to the owner, the heifer had been drenched with 1.5 kg of common salt mixed in a small quantity of water in the morning. The animal remained confined in the shed without access to water until evening, when nervous signs became evident. This practice of salt drenching, locally believed to stimulate appetite and improve body condition, is common among farmers in Punjab. Clinical examination revealed dullness, depression, congested mucous membranes, severe dehydration, reduced reflexes, anuria, absence of defecation, intermittent opisthotonus and profuse salivation. Vital parameters recorded were rectal temperature 103.2 °F, heart rate 110/min, and respiration rate 44/min. The laboratory findings revealed elevated packed cell volume (52%),

serum sodium (168 mEq/L) and serum chloride (124 mEq/L). Based on history, clinical signs, and laboratory confirmation of hypernatremia, a diagnosis of salt toxicosis was established.



Fig.4. Buffalo heifer in lateral recumbency exhibiting opisthotonus

Intravenous isotonic fluids were administered along with dexamethasone sodium phosphate (5 ml), mannitol (100 ml) and vitamin B complex (5 ml), as described by Radostits *et al.* (2007) and Aiello (2016). The heifer was managed with frequent provision of small amounts of fresh, clean water at short intervals. Immediate administration of large volumes of water was avoided, as rapid correction of hypernatremia can cause cerebral oedema. Hence, a gradual hydration was practiced in the present case. The animal responded favourably and recovered uneventfully after five days of treatment and monitoring. Salt toxicosis in ruminants arises from excessive sodium chloride intake, water

*Corresponding author : kanwardhillon55@pau.edu

restriction, or a combination of both (Sharma *et al.*, 1993; Radostits *et al.*, 2007; Smith, 2020). While cattle may tolerate up to 900 g of salt per day if unlimited water is available (Smith *et al.*, 1972), water restriction precipitates intoxication. In the heifer, observed signs i.e. salivation, opisthotonus, and limb paddling were consistent with earlier report (Aiello, 2016).

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