

HAEMOGALACTIA (BLOOD IN MILK) IN A BUFFALO

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History

On October 14, 1996, a seven-year old Nili-Ravi buffalo was brought to the outdoor clinics of the Department of Clinical Medicine and Surgery, University of Agriculture, Faisalabad for the treatment of reddish discolouration of milk. The condition had started three days prior to seeking our professional assistance. The animal was in 3rd lactation and had calved three months ago. The milk yield dropped drastically with the onset of the condition although feeding and water consumption had remained unaffected. The feed of the animal consisted of concentrate + fresh berseem and wheat chaff. The animal was housed on brick-floor without any bedding. There was no history of any trauma to the udder or teat.

Clinical and Laboratory examination

The vital parameters were within their normal ranges (temperature: 102.4°F; pulse rate: 48 per minute; respiration rate: 18 per minute) for buffalo (Cockrill, 1974). Urine colour was normal but faeces had a foul odour and were slightly brownish in colour. Microscopic examination of the faeces revealed the presence of a few *Bunostomum* eggs. On palpation of the udder and teats, no abnormality (heat, pain, firmness, squelching, etc.) could be detected. The colour of the milk was dark pinkish from all four quarters. Quarter fore-milk samples were examined for mastitis by Surf field mastitis test (Muhammad *et al.*, 1995). To this end, quarter fore-milk samples were mixed with an equal quantity of 3 per cent solution of household detergent, Surf (Lever Brothers, Pakistan) and examined for gel formation on a pattern similar to California mastitis test (Schalm *et al.*, 1971). Milk samples from all the quarters were negative for mastitis as there was no gel formation. Microscopic examination of wet milk film revealed the presence of innumerable intact erythrocytes. Centrifugation of the milk samples sedimented these erythrocytes in the form of a bead at the bottom of the conical centrifugation tube.

Diagnosis

On the basis of a positive sedimentation test and the presence of erythrocytes in the wet milk film (*supra vide*), a diagnosis of haemogalactia (blood in milk) was reached. The absence of any systemic reaction ruled out

the possibility of leptospirosis in this case. The feed of the animal did not contain any moldy sweet clover and this negated the implication of dicoumarin intoxication as one of the causes of reddish discolouration of milk.

Treatment

The following treatment was instituted daily for two consecutive days:

i. Inj. Calcium borogluconate with magnesium and phosphorus (Milfone-C; Star Labs. Pakistan) 300 mL IV, ii. Inj. Adrenochrome mono semicarbazone (Anaroxyl, Organon, Pakistan) 15 mL IM.

iii. Ice cold water irrigation of the udder for 15 minutes.

No tangible improvement in the colour of the milk was noticed till 72 hours after initiation of therapy. And it took another seven days (thus the total course of the condition was 12 days) for the complete disappearance of the discolouration. The client recommenced consumption of the milk from this buffalo 12 days after the onset of the malady.

DISCUSSION

A reddish milk discolouration of varying severity may result from such causes as rupture of a blood vessel in the gland by direct trauma, a capillary bleeding in the congested udder soon after calving, leptospirosis or dicoumarin intoxication associated with moldy sweet clover poisoning (Radostits *et al.*, 1994). Of these, the traumatic rupture of some varicose blood vessel within the lactiferous sinus of the mammary gland is the most frequent cause of reddish discolouration of milk (Hungerford, 1990). In leptospirosis, the milk may be stained red but since the aetiopathogenesis of this disease involves the elaboration of a haemolysin by leptospirae, the milk is not expected to contain intact erythrocyte. Wet milk film on microscopic examination was found teeming with intact erythrocytes. As such a diagnosis of haemogalactia associated with blood vessel rupture was concluded in the case being reported herein.

Some salient features of the haemogalactia in the case described in the present report were:

- a) The condition was not associated with calving which had occurred three months ago. Blood in milk is typically a malady associated with

- congested udder at time of parturition.
- b) The condition was non-responsive to the administration of calcium borogluconate + parenteral coagulant (Anaroxyl) and cold water therapy. Radostits *et al.* (1994) have stated that although calcium borogluconate is the standard therapy for blood in milk, better results can be expected by parenteral coagulants.
- c) The condition had a protracted course; secretion from all four quarters remaining blood-tinged (although of dwindling intensity) for a period of 12 days since its onset. Radostits *et al.* (1994) stated that in exceptional cases, haemogalactia may persist for 7 to 8 days in cattle. Although no attempt was made to locate the bleeding point, we theorize that in the present case the leakage might have occurred from some major blood vessel supplying or draining the udder. This assumption may explain the protracted nature of the haemogalactia from all four udder quarters as well as refractoriness of this condition to parenteral

administration of calcium borogluconate and coagulant since these agents are effective only in capillary haemorrhage.

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