Turning sickness in a cross bred cow naturally infected with *Theileria annulata*

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Abstract Bovine tropical theileriosis (BTT) has been since long recognized as a persistent major constraint on genetic improvement programme of native cattle breeds under “Operation Flood”. The disease accounts for high mortality in dairy cattle especially calves. A cross bred cow (Rathi × Holstein), aged 3 years and weighing ~300 kg, owned by a person from weaker sections of the society, initially suffered from ruminal hypotonocyt, severe dyspnoea, frothy discharge, lacrymation, cessation of defaecation, anorexia besides, icterus, mild form of anaemia and moderately elevated temperature etc., for the past 48 h. The cow suddenly developed pre mortal fall in temperature and neurological signs suggestive of posterior paresis. Ante mortem laboratory investigations revealed Koch Blue Bodies in the lymphocytes and *Theileria annulata* meronts in the erythrocytes. The cerebral form of BTT in the instant case was differentiated and discussed with other diseases causing analogous neurological signs.

Keywords Turning sickness · *Theileria annulata* · Cerebral theileriosis · Crossbred *Taurus* cattle

Introduction

In the Indian subcontinent, the prevailing epizootiological determinants offer the optimum environment for faster propagation of the acarine intermediate host (*Hyalomma anatolicum anatolicum*) and in situ development of the *Theileria annulata* causing the bovine tropical theileriosis (BTT). The disease accounts for Global losses to the tune of US \$ 800 million per annum and nearly 70 % bovine mortality (Brown 1997). In India, the BTT assumed considerable importance with the implementation of the genetic improvement programme of indigenous *taurus* cattle breeds, using exotic germ plasm. The total loss due to BTT in India has been estimated to US\$384.3 million per annum (Minjauw and McLeod 2003). Round the year prevalence of infective ticks in the surrounding and trans-placental transmission of *T. annulata* from dam to foetus, causing per acute and fatal disease in the neonates further enhanced the significance of the disease especially in the semi arid region of India (Godara et al. 2009). The api-complexan parasite has been restricting cross border movement of cattle and import of high yielding breeds from the advanced countries. Accordingly, BTT has been a recognized serious constraint in cross breeding programme aiming at upgrading indigenous cattle population under ‘Operation Flood’ (Ruprah 1985; Bhatia and Shah 2001). The authors have come across an yet another interesting but unusual observation, that *T. annulata* infection associated with cerebral form of the disease involving central nervous system (CNS), popularly known as “Turning Sickness”, in a cross bred cattle.

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Materials and methods

A cross bred cow (Rathi × Holstein), aged 3 years and weighing ~300 kg was presented on 18th April, 2011, before the clinicians at the Teaching Veterinary Clinical Complex, Apollo College of Veterinary Medicine, Jaipur. The cow was reported suffering from ruminal hypotonicity, sudden cessation of defecation and anorexia for past 48 h. Further enquiry revealed a sudden fall in milk yield, rapid loss of general condition and labored breathing for the past 1 week. The cattle shed management, housing, sanitation, etc. were not in absolute conformity with scientific standards. These were reported to be of typical traditional type, suited for small animal holdings of three to five animals. A closer look to the animal on next day revealed, severe dyspnoea, frothy nasal discharge, lachrymation, abducting hind limbs and was standing in a typical posture with arched and extended head in forward direction. The owner confirmed that these signs appeared a short while ago while the animal was being brought to the complex for therapeutic intervention. Peripheral blood (5 ml aliquot in EDTA) was aseptically collected and the body hair coat was carefully searched for the presence of acarine parasites. Rectal coprological samples were also collected. The samples were brought to the laboratory for identification of the pathogen(s) and its vectors using standard keys/techniques (Bowman et al. 2003; Hendrix and Ed 2006; Taylor et al. 2007).

Overall clinical assessment of the patient was suggestive of grave prognosis. The owner was suitably informed. In order to overcome serious neurological and digestive plight, the animal was forthwith managed and was administered symptomatic and supportive therapy comprising of single intramuscular injection of buparvaquinone (“Zubion” Intas Pharmaceuticals) @ 2.5 mg/kg, streptopenicillin 2.5 g intramuscular (“Dicrysticin” Sarabhai Zydus), 10 ml of meloxicam intramuscular (“Melonex” Intas Pharmaceuticals), 10 ml of Vitamin B complex intramuscular (“Tribivet” Intas Pharmaceuticals), 10 ml of dexamethasone intravascular (“Merideca” Wockhardt Ltd.), Rumentas (Intas Pharmaceuticals) 2 bolus per os as electuary.

However, the animal could not with stand the severity of the disease and massive infection in circulation. Unfortunately, she succumbed to the disease in the evening of 19th April, 2011. She could not be autopsied due to theological/religious limitations.

Results and discussion

Clinical examination of the cow revealed lusterless dull hair coat infested with the developmental instars, including

adult *H. anatolicum anatolicum* over dewlap, axilla, ventral abdomen, udder and around peri anal region. The animal was weak and emaciated. The conjunctival mucous membrane was pale and icteric. The muzzle was dry with frothy nasal discharge from both the nostrils. There was excessive drooling salivation, besides, accelerated pulse (76/min) and almost dry and hard rectal faeces. The pre scapular lymph nodes were moderately swollen and enlarged. The rectal temperature was 101.2 °F (38.5 °C). Signs of respiratory distress were quite evident. Auscultation sounds of the lungs revealed pulmonary oedema of diaphragmatic lobes and presence of exudates in the pleural cavity. The animal was seen repeatedly pressing head against the hard objects (walls of hospital) and showed occasional circling movements.

The condition of the animal deteriorated fast, while she was under clinical examination on 19th April, 2011. She suddenly developed harsh and forceful breathing, extended her head and neck in the forward direction with abducted hind limbs and producing characteristic audible breathing sounds. Finally, she fell into right lateral recumbancy. She was observed making repeated attempts to stand but hind limbs could not take her weight and ultimately she suffered from posterior paresis. In conclusion, the cow suffered from clinical signs suggestive of icterus, synchronous milder form of anaemia, respiratory distress, pre mortal moderate pyrexia and terminal involvement of central nervous system, developing neurological signs prior to her death.

Laboratory investigation of the peripheral blood revealed suppressed haematological indices [haemoglobin-9.0 g/dl, PCV-26 %, TLC- $8.5 \times 10^3/\mu\text{L}$] and altered differential cell counts [neutrophils 23 %, lymphocytes 75 %, eosinophils 2 %] and confirmed that the animal suffered from milder form of anaemia, severe leucopenia, lymphocytosis and moderate eosinophilia at the terminal stage of the disease. Microscopic examination of the Leishman stained thin smear revealed Koch blue bodies (KBB) in the cytoplasm of the lymphocytes (Fig. 1) whereas, the Giemsa stained smears evidenced characteristic intra erythrocytic piroplasms identified as *T. annulata* (Fig. 2). The parasitemia was 30 %. Whereas, copro samples could not demonstrate parasitic ova and/or cysts.

On the Indian subcontinent, in the enzootic semi arid desert, the BTT, is mainly caused by *T. annulata* (Godara et al. 2009; Sudan et al. 2012). Out of the 15 *Hyalomma* species of veterinary significance, the three host tick *H. anatolicum anatolicum* ranks high and designated as the word’s most damaging ticks, transstadially transmitting the BTT in the susceptible exotic and/or their cross bred progeny with the native *taurus* cattle (Levine 1973; Gill et al. 1978). The nymph and adult instars of the tick are widely distributed in the semi desert environment,

Fig. 1 Koch blue body in the lymphocytes

Fig. 2 *Theileria annulata* piroplasms in the RBC's

prevailing in a majority of the middle East and South Asian countries, including India (Aiello and Mays 1998; Radostits et al. 1994; Bhatia and Shah 2001). In the study area, *H. anatolicum anatolicum* has been active year around.

Epizootologically, BTT in dairy animals is regulated by four component systems, where in susceptible host, optimum environment, the parasite and its vector play an integral role and accounts for the high occurrence of the disease especially during summer months (May to June) in the semi desert ecosystem at Jaipur. Extremely high temperature, high milk yield and heat stress during summer months are recognized major predisposing factors.

“Turning sickness” is a sporadically occurring specific disease of cattle, aged 6 months to 2 years, terminating fatally in the Eastern Equatorial regions of the Africa. The earliest reports from South Africa on the occurrence of the

fatal disease in Elk was attributed to aberrant *T. taurotragic* infection and *T. parva* in the African cattle breeds (Radostits et al. 1994; Taylor et al. 2007; Urquhart et al. 2003). Elsewhere in Africa, infection with *T. parva* and *T. mutans* have been incriminated as causal agent of the BTT—the cerebral syndrome, characterized by pre mortal moderate pyrexia, icterus, sudden development of peculiar neurological signs, especially occasional circling and/or turning movements and abduction of hind limbs and paresis (Tustin and van Heerden 1979). Pathogenesis of the syndrome has been claimed to be unclear. Possibilities of crossing the CNS barriers by the parasite and causing auto immune disorders, characterized by intra vascular agglutination of the parasitized blood cells in the cerebral capillaries with consequential embolism/thrombosis could not be ruled out (Mettam and Carmichael 1936; Soulsby 2005). Of late, crossing the placental membranes by *T. annulata* and infecting the foetus of cow has been documented elsewhere (Godara et al. 2009). The occurrence of the lethal turning sickness in the deceased cross bred cow during summer months, reinforce to above findings on records. The diary animals, especially exotic and/or their cross bred progenies, have been documented to be more susceptible to the disease than the native breeds of cows in the enzootic areas (Gill et al. 1978; Godara et al. 2009; Taylor et al. 2007).

The characteristic clinical signs reported here in, were consistent with the terminal stage of the lethal BTT (marked fall in general condition and lactation, caesation of rumination and digestive disturbances, pre mortal moderately elevated body temperature (38.5 °C), accelerated pulse and respiratory rate, drooling saliva, frothy nasal discharge and respiratory distress), coupled with sudden development of pathognomonic neurological signs (occasional circling, head pressing and persistent abduction of hind limbs, animal falling in lateral recumbancy and finally suffering from transient posterior paresis) and demonstration of the pathogen in circulation were suggestive of sub acute lethal BTT with grave prognosis. Further, depressed haematological indices confirming moderate anaemia, icterus, leucopenia, lymphocytosis and eosiniphilia and above all, pre mortal poor response to therapeutic intervention made authors to strongly speculate and believe that the diseased cow suffered from cerebral form of lethal BTT (“turning sickness”) and her death was ascribed to anaemic anoxia, incidental to blood stasis caused by clogging of brain capillaries by accumulation and/or agglutination of parasitized erythrocytes there in, as elucidated elsewhere in the cerebral form of babesiosis/theileriosis, respectively caused by *Babesia bovis* and *T. parva* in bovine (Aiello and Mays 1998; Taylor et al. 2007; Lefevre et al. 2010). The diseased cow, while undergoing lethal phase of the disease and high parasitemia, might have adversely affected the in

situ physiology of pituitary and adrenal glands resulting in poor feed intake, nutrient utilization and characteristic marked fall in body temperature and overall fast deterioration of the general health of the animal (Forsynth et al. 1999; Lefevre et al. 2010).

In absence of observations on necropsy lesions, the differential diagnosis of the cerebral form of the lethal BTT vis-à-vis other disease conditions (Listeriosis, Rabies, Polioencephalomalacia), exhibiting almost analogous neurological signs viz head deviation, circling, loss of herding instinct, paresis, muscular tremors, stiffness of hind legs and in coordinated gait seems logical and imperative. In Listeric encephalitis, the cattle suffers from unilateral facial nerve paralysis causing drooping of eyelids, ears, dilated nostrils besides, drools saliva, incidental to pharyngeal nerve partial paralysis. The duration of the disease is invariably longer (2–6 weeks). The lesions are mainly localized in the brain stem and the clinical signs indicate dysfunction of the third to seventh cranial nerve (Radostits et al. 1994; Lefevre et al. 2010). Whereas rabies affected cow, makes unprovocative attacks and is unable to swallow and/or drink. It is characterized by early paralysis of the throat and masseter muscles. The animal often exhibits changed behavior, stop eating and drinking, frequently pass urine, seek solitude, produce characteristic loud bellowing sounds and there is dropping of lower jaw (Radostits et al. 1994; Lefevre et al. 2010). The Polioencephalomalacia (PEM) is primarily a non infectious neurological disease of the young and intensively reared cattle. It is incidental to the depressed activity of the tissue thiamine and related enzymes. The disease is characterized by amaurosis “glass eye” and strabismus followed by neurological signs and recumbancy. The disease is not prevalent on the Indian sub continent (Radostits et al. 1994; Lefevre et al. 2010).

Nevertheless, considering the *T. annulata* induced cerebral form of theileriosis “turning sickness”, a significant emerging field problem, the findings reported herein have undoubtedly opened newer frontier of research. It would be interesting to precisely investigate through well planned experimental studies to elucidate (a) effective integrated therapy aiming at complete elimination of the pathogen from the cerebral circulation and overcoming the sudden death of the host; (b) prevalence of the disease, epizootiological predisposing factors and reasons for its underreporting by the field veterinarians in the enzootic areas; (c) pathophysiological impact of the disease on endocrine glands especially pituitary and adrenal glands, adversely affecting the pre mortal thermoregulation, feed intake and nutrient utilization, spontaneous marked fall in lactation and deterioration of the general health of the affected animal besides, onset of shock; (d) mechanism of in vivo migration and access of the pathogen to cerebral capillaries and/or tissues and multiplication of the pathogen

there in; and (e) immunological tissue response of the host prior to sudden onset of pre mortal sequential events leading to onset of neurological signs including recumbancy and death.

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References

- Aiello SE, Mays A (1998) The Merck Veterinary Manual, 8th edn. Merck & Co Inc, New Jersey, pp 31–33, 678–679
- Bhatia BB, Shah HL (2001) Protozoa and protozoan diseases of domestic animals. ICAR Publication, New Delhi, pp 148–158
- Bowman DD, Lynn RC, Eberhard ML, Alcaraz A (2003) Georgi's parasitology for veterinarians, 8th edn. Saunders-An imprint of Elsevier, St. Louis, pp 1–422
- Brown CGD (1997) Dynamics and impact of tick borne diseases of cattle. *Trop Anim Health Prod* 29:15–35
- Forsynth LMG, Minns FC, Kirvar E (1999) Tissue damage in cattle infected with *Theileria annulata* accompanied by metastasis of cytokine producing, schizont infected mononuclear phagocytes. *J Comp Pathol* 120:39–57
- Gill BS, Bhayyacharyulu Y, Singh A (1978) Chemoprophylaxis with oxytetracycline drug in the immunization of cattle against *Theileria annulata*. *Int J Parasitol* 8:467–469
- Godara R, Sharma RL, Sharma CS (2009) Bovine Tropical Theileriosis in a neonate calf. *Trop Anim Health Prod* 42(4):551–553
- Hendrix CM, Robinson Ed (2006) Diagnostic parasitology for veterinary technicians, 3rd edn. Mosby IMC (Elsevier), St Louis, pp 245–254
- Lefevre PS, Blancou J, Chermette R, Uilenberg G (2010) Infectious and parasitic diseases of livestock, Lavoisier Tec & Doc, 343–352(985–989):1839–1866
- Levine ND (1973) Protozoan parasites of domestic animals and of man, 2nd edn. Burgess, Minneapolis, p 406
- Mettam RWM, Carmichael J (1936) Turning Sickness—a protozoan encephalitis of cattle in Uganda, its relationship with East Coast fever. *Parasitol* 28:254–283
- Minjauw B, McLeod A (2003) Tick-borne diseases and poverty. The impact of ticks and tick-borne diseases on the livelihood and marginal livestock owners in India and Eastern and Southern Africa. Research report, DFID animal health programme, centre of tropical veterinary medicine, University of Edinburgh
- Radostits OM, Blood DC, Gay CC (1994) Veterinary medicine—a textbook of diseases of cattle, sheep, pigs, goats and horses, 8th edn. Bailliere Tindall, London, pp 1206–1212
- Ruprah NS (1985) A textbook of clinical protozoology. Oxonian press Pvt. Ltd., New Delhi, pp 286–355
- Soulsby EJJ (2005) Helminths, arthropods and protozoa of domesticated animals, ELBS-7. Bailliere Tindall, London, pp 728–737
- Sudan V, Sharma RL, Borah MK, Mishra R (2012) Acute bilateral proptosis in a cross bred calf naturally infected with *Theileria annulata*. *J Parasit Dis*. doi:10.1007/s12639-012-0111-6
- Taylor MA, Coop RL, Wall RL (2007) Veterinary parasitology 3rd edn. Blackwell Publishing Ltd., Oxford, pp 109–113
- Tustin RC, van Heerden J (1979) Bovine cerebral theileriosis (turning sickness) with spinal cord involvement. *J S Afr Vet Assoc* 50(1):49–51
- Urquhart GM, Armour J, Duncan JL, Dunn AM, Jennings FW (2003) Veterinary parasitology. Blackwell Science Ltd., Oxford, pp 246–249