ARTERIAL THROMBOSIS AND FIBRINOID NECROSIS-POSSIBLE ETIOLOGY BEHIND HYDROALLANTOIS IN *Bubalus bubalis* (WATER BUFFALO)

Ankit Kumar Ahuja[1,*], Deepak Sharma[1], Arundeep Singh[1], Nitin Dev Singh[2] and Prahlad Singh[1]

Received: 6 July 2020
Accepted: 25 March 2022

ABSTRACT

A pluriparous buffalo in 7th month of gestation was presented with history of anorexia since last 15 days and abdominal distention past one month. Per vaginum examination revealed the presence of fluid thrills with no palpation of fetus, fetal parts and cotyledons. Ultrasonography of the abdomen shows mild to moderate pleural effusions in thoracic cavity, reticulum being pushed to 6th inter-coastal space, intestinal loops pushed cranially in the abdomen and anoechoic fluid seen in abdomen suggesting of hydroallantois. Our treatment regimen followed by regular per vaginum examination generated positive outcome with dam recovered uneventfully. Histopathological examination suggested uterine epithelium hyperplasia and arterial thrombosis causing obstruction in the normal blood supply. Fibrinoid necrosis was also observed in few smaller blood vessels which are mostly encountered in antigen-antibody complex reaction. Apart from successful management following treatment regimen and frequent examination, we also hypothesize a new etiology behind hydroallantois in this case.

Keywords: *Bubalus bubalis*, buffaloes, hydroallantois, *Arterial thrombosis*, *Fibrinoid necrosis*

INTRODUCTION

Hydroallantois is one of the rarest gestational disorders characterized by rapid fluid accumulation in allantoic cavity due to placental pathology leading to bilateral enlargement of abdomen (Roberts, 1971). In hydroallantois, speedy and anomalous distension of abdomen occurs (Drivers and Peek, 2008) due to rapid accumulation of watery, amber color fluid inside the allantoic cavity over a period of 5 to 20 days in late gestation and is always giving suspicion for twin/triplet pregnancy (Morrow, 1986). Hydroallantois perpetually results in fetal gestational accident occurring due to placental dysfunction and fetal renal failure (Arthur *et al*., 1989; Purohit *et al*., 2012).

[1]Department of Veterinary Clinical Complex, Dr GC Negi College of Veterinary and Animal Sciences, Chaudhary Sarwan Kumar Himachal Pradesh Krishi Vishvidyalaya, Himachal Pradesh, India, *E-mail: ankit.ahuja1947@gmail.com

[2]Department of Veterinary Pathology, College of Veterinary Science, Guru Angad Dev Veterinary and Animal Sciences University, Punjab, India

However, some school of thoughts claimed it as maternal aberration associated with pathologically diseased uterus characterized by compromised caruncles in one horn and compensatory hypertrophy along with disease of remaining (Roberts, 1971). Proliferation and hyperplasia of cytotrophoblastic cells were detected in uterine torsion and hydroallantois in an attempt to bring more oxygen to compensate for the deficient oxygen (Zaher *et al*., 2017).

It is usually treated by terminating the pregnancy using prostaglandin F2α and corticosteroids (Manokaran *et al*., 2011) but the sudden removal of allantoic fluid leads to hypovolemic shock and animal may collapse (Peiro *et al*., 2007). Slow and gradual removal of allantoic fluid may be an alternative method to avoid hypovolemic shock to the dam (Noakes *et al*., 2009). Trans-cervical allantocentesis is another option to drain out the allantoic fluid from the uterus along with bilateral jugular fluid administration to prevent hypovolemic shock in dam (Singhal *et al*., 2018).

CASE HISTORY AND OBSERVATION

A 7 years old pluriparous buffalo was presented in Teaching Veterinary Clinical Complex, GADVASU, Ludhiana with the history scanty fecal output and abdominal distension since one month. Animal was 7 months pregnant and otherwise active. The animal was anorectic since last 15 days and dyspnoic on presentation. On presentation, the buffalo was quiet, alert, and responsive, but within a few hours became progressively obtunded. The rectal temperature was 37ºC, heart rate was 92 beats per minute (tachycardia) and respiratory rate was 20 breaths per minute (tachypnea). Body condition score was three out of five. Heart sounds were intensified and radiated widely over the thorax and abdomen. Mucus membrane was congested with no evidence of petechiation. Blood serum biochemistry revealed a low BUN-creatinine ratio (Table 1).

Per vaginum examination revealed the presence of fluid thrills with no palpation of fetus, fetal parts and cotyledons. However, the uterine blood flow was adequate. Ultrasonography of the abdomen shows mild to moderate pleural effusions in thoracic cavity, reticulum being pushed to 6th inter-coastal space showing mild peri-reticular reaction, intestinal loops pushed cranially in the abdomen and anoechoic fluid seen in abdomen corroborating with our per vaginum findings suggestive of hydroallantois.

TREATMENT

Animal was induced for parturition after inspecting the condition of cervix. Summary of regular per vaginum examination and treatment schedule followed is presented in Table 2. Post operative therapeutics was performed and animal was kept on fluid therapy and antibiotics for 5 days.

Histopathology

The uterine and placental tissue fragments were immediately taken in 10% neutral buffered formalin (NBF) for fixation to obtain sections for further tissue alteration study. Tissues were therefore processed through graded alcohols and subjected to a treatment with xylene and final casting with paraffin wax as tissue blocks (Luna, 1968). Fine tissue sections of 3 to 4 µ thickness were generated following microtomy was placed on clean glass slide and subsequently undertaken

Table 1. Results of haematology and blood biochemistry.

Parameter	Unit	Result		Reference (Abd Ellah *et al*., 2013)
		At presentation (0 h)	After calving (29 h)	
BUN	(mg/dL)	08	09	15.13-59.50
Creatinine	(mg/dL)	2.8	2.5	1.02-2.17
ALT	(IU/L)	64	51	9.84-51.11
AST	(IU/L)	199	137	23.19-89.17
Total protein	(g/dL)	8.7	6.6	6.0-9.8
Albumin	(g/dL)	3.3	2.4	2.2- 4.4
Calcium	(mg/dL)	10.0	8.3	8.20-14.87
Phosphorus	(mg/dL)	3.4	2.3	5.27-8.81
Sodium	(mmol/l)	133	136	121.73-169.28
Potassium	(mmol/l)	3.5	3.1	3.12-6.79
HB	(g/dL)	11	12.5	9.2-15.9
TLC	(/µL)	10090	13240	5000-15000
TEC	(/µL)	6.47×10^6	7.84×10^6	$5.33\text{-}10.72\times10^6$
PCV	(%)	31	37	24-46
Platelets	(/µL)	315×10^3	556×10^3	$100\text{-}800\times10^3$

Table 2. Treatment regimen followed.

Time (h)	Cervical texture	External-Os	Internal-Os	Treatment Given
0	Soft pliable	3 fingers open	Closed	Inj. Vetmate-2 ml I/M Inj. Dexona-10 ml I/M Inj. Diethylstilbesterol-2 ml I/M
21	Soft pliable	Full hand open	2 fingers open (Cervical seal liquefied)	Inj. Vetmate-2 ml I/M Inj. Dexona-10 ml I/M
29	Relaxed and pliable	Full hand open	Full hand open (Dead male calf delivered)	Inj. Normal Saline- 6 L I/V Inj. RL- 6 L I/V Inj. Metrogyl-1.5 L I/V Inj. Enrofloxacin- 20 ml I/M Inj. Belamyl-10 ml I/M Inj. Tolfine-15 ml I/M

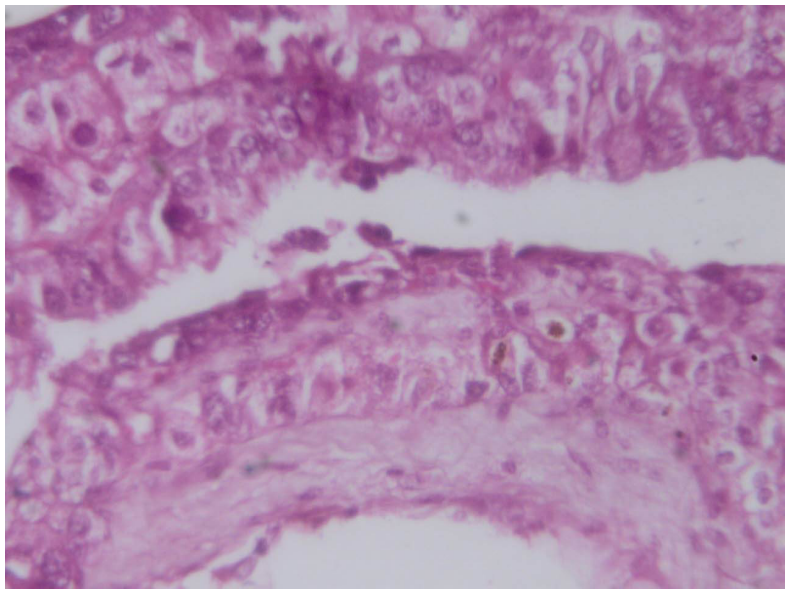

Figure 1. Uterus: Showing epithelial cell hyperplasia. H & Ex 40X.

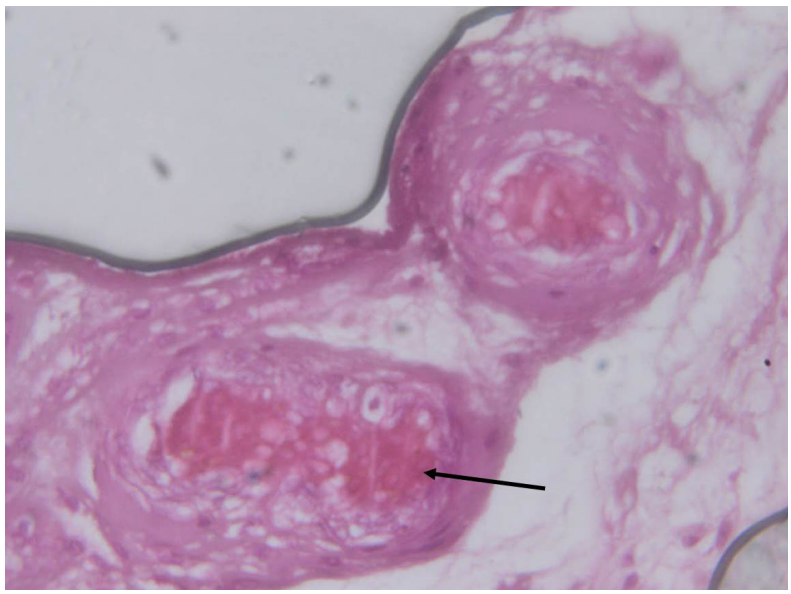

Figure 2. Uterus: Showing arterial thrombus. Note the thrombus is attached to the endothelium (Arrow). H & Ex 40X.

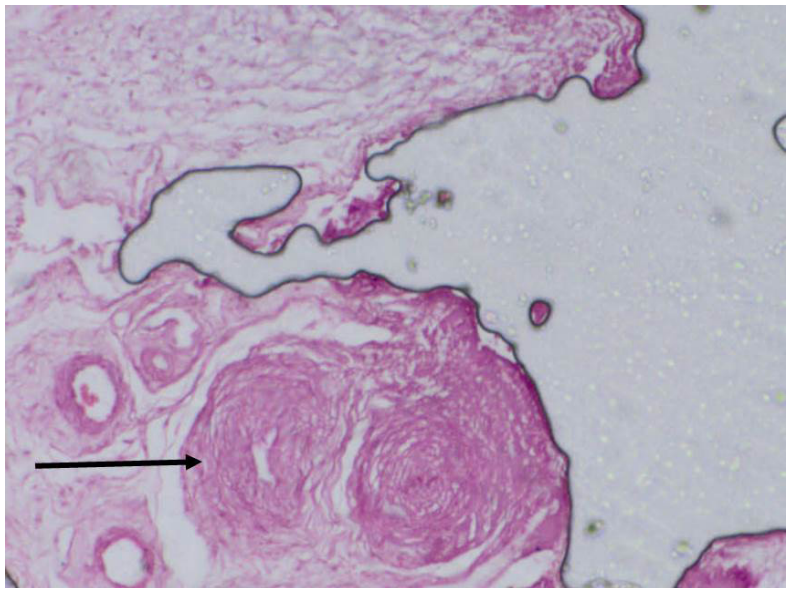

Figure 3. Uterus: Showing fibrinoid necrosis of the blood vessels (Arrow). H & E x 20X.

hematoxylin and eosin (H&E) staining.

On histopathological examination there was hyperplasia of uterine epithelium (Figure 1), which is quite normal during the gestation period. Arterial thrombosis (Figure 2) was observed in one of the blood vessels of the uterus which might have led to blockage of the proper blood supply. In addition to the above findings fibrinoid necrosis was also observed in few smaller blood vessels of the uterus (Figure 3). Fibrinoid necrosis is mostly encountered in antigen-antibody complex reaction (Bajema and Bruijn, 2000) which might be one of the causes of this complication as the blood supply was hindered.

DISCUSSION

Handling of hydroallantois case varies with the duration and severity of the condition. Barter (1986) reported abnormal parturition in hydroallantois due to incomplete cervical dilataion with primary uterine inertia and lack of strong abdominal contractions. The condition is more common during the last phase of third trimester in dairy cattle and the occurrence is less in buffaloes and heifers (Srinivas and Sreenu, 2006). Two major suggestive etiologic pathologies that contribute towards hydroallantois are increased production of fluid or decreased trans-placental absorption (Zaher *et al*., 2017). Corroborating to the findings of Zaher *et al.* (2017), our histopathological findings suggests that impaired transplacental fluid absorption due to chorionic arterial thrombosis and fibrinoid necrosis of blood vessel as the main etiology in the present case. Although some evidence of caruncular hyperplasia is also there. Our findings are in concordance with Jones and Fox (1980), who mentioned that necrosis results in impaired blood supply to placenta leading to

placental insufficiency is the most appreciable histologic feature. Our results were also corroborates to Bane *et al*. (2003) who cited that massive perivillous fibrinoid was associated with intrauterine death, intrauterine growth restriction, and preterm delivery. Blood biochemical picture suggestive of low BUN-creatinine ration indicates a sever muscle injury which is evident in this case.

The treatment regimen we followed during this case gives us the best promising results. Present case reports the successful management of hydrollantois in a buffalo along with its possible etiology. Perhaps more studies are needed to ascertain the etiology hypothesized.

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