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Hydrallantois in a non-descript doe (*Capra hircus*) – A case report

Ramsingh L, M Rajashri, and Vishal P

Abstract
The present case reports Hydrometra in a Non-Descript Doe and its diagnosis by trans-abdominal ultrasonography which was terminated by Prostaglandin therapy and relieved a live fetus.

Keywords: Hydrallantois, Goat, fluid accumulation, placental disorder

Introduction
Hydrops of fetal membranes is the large amount of fluid accumulation within the gravid uterus (Feliciano *et al*., 2013) [2]. Hydrallantois (hydrops of the allantois), is characterized by abnormal accumulation of allantoic fluid during a 5- to 20-day period in the last trimester of pregnancy (Milton *et al*., 1989) [5]. Hydrallantois is rarely diagnosed and usually affects cows and small ruminants (Loi *et al*., 2006) [6]. The physiopathology of hydrallantois is related to the reduction of placental vascularization, leading to metabolic changes in the placental tissue and fetal membranes and accumulation of fetal fluids. Such findings were observed in ovine clones (Loi *et al*., 2006) [6]. Other causes are fetal malformation, fetal hepatic or renal disorders (e.g., hydronephrosis) and umbilical cord torsion (Jackson, 2006) [3].

Case History
A full term pregnant 4 year old pluriparous goat in her third parity was presented to teaching veterinary clinical complex, Rajendranagar, Hyderabad with complaint of anorexia, respiratory distress, restlessness, cessation of rumination, and enormous ventro-bilateral abdominal distension (Fig. 1) with progressive distension of the abdomen drastically since 20 days. Clinical examination revealed normal rectal temperature and tachypnoea. Abdominal ballotment indicated the presence of large volume of fluid but fetus could not be palpated. Per vagina examination revealed closed cervix. Trans-abdominal ultrasonography (5MHz) revealed fluid filled uterine horns along with fully developed viable fetus and few placentomes. The fetus was presented with normal cardiac frequency without any abormalities.

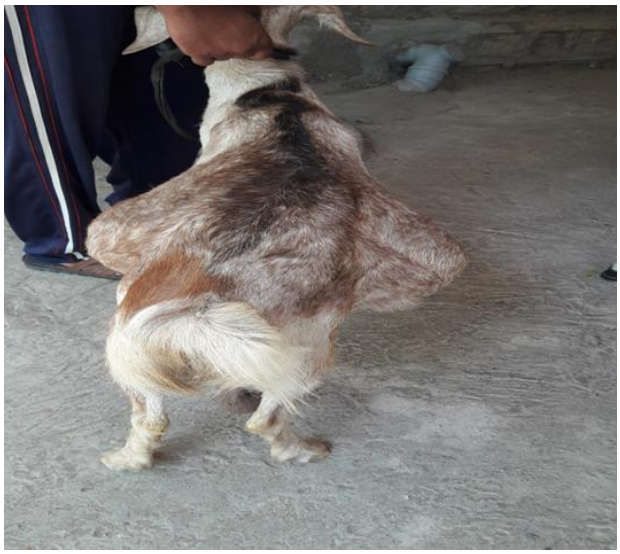

Fig 1: showing abnormal ventro-bilateral abdominal distension in Non-descript Doe

Treatment

Pregnancy was terminated by administration of Prostaglandin (Inj. Pragma 250 mcg, I/M) and Dexamethasone (Inj. Dexona 15mg I/M). Within 2 hours, cervix was dilated exposing the allantoic sac. Allantoic sac was punctured manually and around 10 litres of allantoic fluid was drained. Then amniotic sac was ruptured and one viable fetus was delivered by applying traction over the forelegs of the fetus. Fluid therapy (500ml of Ringer's lactate) was administered intravenously. Doe was injected with Oxytocin (15 IU I/M) and Calcium Borogluconate (40 ml, I/V). Parentral antibiotic therapy (Inj. Enrofloxacin 10 mg/Kg B.wt) was given for 5 consecutive days. Herbal uterine ecbolic (Susp. Exapar 20ml P/O) was given for 3 days

Discussion

Physical examination and transabdominal ultrasonography is recommended for confirming the hydroallantois in ewes and does (Bhattacharyya *et al.,* 2012) [1]. In the present case, trans-abdominal ultrasonography (5 MHz) was employed for arriving at a definite diagnosis of hydroallantois. Ventral herniation or rupture of the prepubic tendon (Stich and Blanchard, 2003) [8], rectal and vaginal prolapse, rupture of the uterus, dystocia associated with uterine atony, retained placenta, metritis and agalactia (Toniollo *et al.,* 2003) [9] are the possible sequelaeof hydroallantois. In present case study, after induction of parturition, dystocia was observed due to uterine atony. Hence the fetus was relieved by gentle traction. Moreover, placenta was not expelled even after 24 hrs. Hence, herbal uterine ecbolics was administered for consecutive 3 days for expulsion of placental membranes and for early uterine involution. If the Induction of parturition fails, caesarean section can be opted when the fetus survivability is low due to the pulmonary immaturity. In the present study, there were no fetal abnormalities indicating the probability of placental disorder in the form of either enhanced permeability of the chorioallantoic membrane or decrease in the active transport of sodium across the chorioallantoic membrane (Peiro *et al.,* 2007) [7]. Other factors like hormonal imbalances, fetal renal disease (Morin *et al.,* 1994) [6], multiple fetuses in the uterus, fetal liver disease, uterine torsion and/or twisting of the umbilical cord, deficiency of vitamin A which compromises the number of caruncles, malnutrition conditions and heart or renal diseases (Toniollo and Vicente, 1993) [9] may contribute to this condition (Peiro *et al.,* 2007) [7].

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