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Hydroallantois in Large Animals

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ABSTRACT

Hydroallantois, which is typically regarded as maternal abnormality, is characterized by a fast and abnormal distension(symmetrical abdominal expansion)of the abdomen that happens over a period of 5 to 20 days in late gestation(Bull frog appearance)(Selvaraju *et al*., 2012).

This fluid accumulation is watery and amber in color, and it always raises the possibility of twin or triple pregnancy (Drivers and Peek, 2008). According to (Kumar *et al,*.2019), it is frequently linked to fetal developmental abnormalities. The physiopathology of hydroallantois is associated with a decrease in placental vascularization(hydrallantoic placenta has poor angiogenesis), which leads to metabolic alterations in the placental tissue, fetal membranes and the accumulation of fetal fluids (Kapadia *et al*.,2018).Furthermore it leads to fetal abnormalities, hepatic or renal diseases in foetus (hydronephrosis). Hydrops allantois is the common condition in animals but, simultaneous occurance of hydrops allantois and hydrops amnii is also found in same animal.

In cows, the disease can be identified by ultrasound and physical examination (vaginal/rectal) (Sutaria *et al*.,2020).The condition was confirmed and clinical findings were documented in compliance with earlier research by(Pandey *et al*.,2014) and Tripathi *et al.* (2015).

Keywords: Electrocardiography, Hydroallantois, Ultrasonography Termination, Angiogenesis.

INTRODUCTION

Hydroallantois is the uterine disease related to dysfunction of caruncle resulting in hypertrophy, edema and non-infectious degenerative condition of placentomes with adventitious placenta effects. Most gestational hydropsies (approximately 80–90%) are caused by hydroallantois (Youngquist *et al* 2007).Thus, it would seem that the abnormal conditions arised when fetal fluid was increasing, but the regulatory system was malfunctioning (Robert, 1971). Both the fetus and the fetal membrane were affected by hydroallantois (Napolean 2012).

In this configuration, the majority of the caruncles in one horn were non-functional and the remaining placentomes are significantly enlarged and might be diseased. The number of functional

cotyledons was discovered to be abnormally low, and the pregnant horn developed compensatory supplementary caruncles (Reddy *et al* .,2018) .

Because of placental anomalies and potential disruptions to sodium metabolism at the cellular level, allantoic fluid accumulates quickly during the hydroallantois disease.

Animal with a history of stomach pain, dyspnea, bloat, and significant belly enlargement . During the clinical examination, several observations were made, including sunken eyeballs, a dry snout, anxiety, and difficult breathing. The uterus was tight and the fetus was not seen during rectal examination.(C. Rohit Kumar*et al*.,2019).

During electrocardiography, arrhythmia and low voltage QRS complex amplitude were observed (Reddy *et al.,* 2015, Reddy and Sivajothi, 2016).

An ultrasound examination found a significant amount of fluid along with fewer and larger cotyledons.

The intrauterine fluid put pressure on the diaphragm in the current investigation, resulting in respiratory discomfort. Uterine inertiawhich results from overstretching of the abdominal and uterine musculature, might aggravate spontaneous miscarriage which wasone of the aftereffects of hydrallantois . Furthermore, hydrallantois can have fatal consequences if left untreated. These disorders include cardiovascular shock, abdominal wall hernias, ruptures of the prepubic tendon, abdominal wall ruptures, and ultimately death (Pouya Dini *et al*.,2020). Hypertrophied caruncles and cotyledons were typically linked to hydrallantois (Drost, 2007).

Prostaglandin (PGF2α) and corticosteroids are typically used to end the pregnancy (Manokaran *et al*,.2016). Removing allantoic fluid graduallyvia transcervical allantocentesis and bilateral jugular fluid administration (Singhal *et al*.,2018) could help to prevent hypovolemic shock to the dam (Noakes *et a*l.,2009 and Peiro *et al*.,2007))

CAUSES OF HYDROALLANTOIS

In certain species, the condition's genesis is not fully understood. A missense mutation in solute carrier family 12 member has been linked to the hydrallantoic disease in Japanese Black cattle (S.Sasaki.,*et al* 2016). Some breed predilection and genetic characteristics have been suggested, but not proven, to be linked to this problem in horses.Yet it has also been proposed that structural or functional alterations in the chorioallantois are linked to the pathophysiology of hydrallantois (M.D.de.Amorim*et al.*,2018).

On the chorionic surface, the macroscopic examination of the affected placentas revealed regions with small, sparse villi or avillous patches (A. Foote*et al*.,2012). In situations of hydroallantoic acid exposure, there have been reports of some degree of edema and cystic development without any indication of inflammation .

A decrease in the number of caruncles is frequently observed in sheep and cattle during adventitious placentation, which suggests functional insufficiency of the placenta (R.S. Youngquis*et al*.,2006).

Furthermore, in vitro generated embryos have been shown to have aberrant placental vascularization, which is followed by hydrallantois (L.C. Smith.*,et al* 2012).
It is interesting to note that pregnant sheep fed locoweed (Astragalus and Oxytropis species) containing indolizidine alkaloid swainsonine have disruptions in placental vascular development and hydroallantois. For the placenta to function properly, there must be sufficient vascular growth in the chorioallantois and the formation of a vast capillary network (L.Reynolds*et al*.,2005).

The process of vascular development is intricate and involves the proliferation, migration, and differentiation of endothelial cells within preexisting blood arteries . In order to create new tube-like structures, these processes cause endothelial cells to sprout out of capillaries. At the same time, a secondary vasodilatation occurs to improve circulation and nutrient absorption .
Dynamic gene expression is necessary for proper vascular development in any tissue, including the placenta (C.J. Favre*et al*.,2003).The most studied genes related to angiogenesis are endothelial nitric oxide synthase (eNOS [NOS3]), hypoxia-inducible factors (HIF1A), vascular endothelial growth factor (VEGF), vascular endothelial growth factor receptor-1 (VEGFR-1 [Flt-1]), vascular endothelial growth factor receptor-2 (VEGFR-2 [KDR or Flk-1]), angiopoietin 1 (ANGPT1), and angiopoietin 2 (ANGPT2). Vascular endothelial cells require VEGF and its receptors as vital growth factors, and these substances are strong angiogenic factors .Vascular endothelial cell migration, protease synthesis, and vascular permeability are all regulated by VEGFs and are essential elements of the angiogenic process. It has also been demonstrated that ANGPTs and eNOS are important angiogenic factors that control the growth and development of blood vessels (L.P. Reynolds*et al*.,2001).

Estrogen is thought to be one of the main regulators in primates of new vascular development in the placenta (T.Napso*et al.,*2018). During pregnancy, estrogen increases the fraction of stromal tissue occupied by blood vessels inside the villous placenta, promotes angiogenesis, and causes a progressive increase in blood vessel density (E.D. Albrecht*et al*.,2010).It has been proposed that NO, which can stimulate VEGF synthesis, this mediates estrogenic impact. Furthermore, it has also been proposed that abnormalities in the placental vasculature, which result in a buildup of fluid and edema in the chorioallantois, are caused by disruptions in estrogen concentrations (L.C. Smith*et al*,.2012).

Allantoic fluid resembles plasma in the early stages of fetal development, prior to the development of the mesonephros. Later on, while the kidneys of the metanephric stage are working, creatinine builds up in the allantoic fluid. In normal pregnant cows, electric potentials across the allantoic fluid and the maternal blood showed that the concentration of sodium in the maternal blood was higher than that of the allantoic fluid, indicating that the distribution of sodium was maintained against its electrochemical gradient. This points to the possibility of an active transport system from the allantoic fluid to the mother's blood. The chloride ion is passively transferred from the allantois to the maternal circulation, and the placental membranes seem to be very impermeable to the potassium ion.

Allantoic fluids grow quickly between forty and sixty-five days, and again between six and seven and a half months. The amnionic fluid is increase between three and a half to four months.The allantoic fluid surpassed the amnionic fluid during the firstand third trimesters of pregnancy. So chance of hydroallantoic is more during the second increase in allantoic fluid.Typically, 20 liters of allantoic fluid was found however, in the case of hydroallantois, that amount might increase to 150–260 liters (Aftab *et al*.,online).

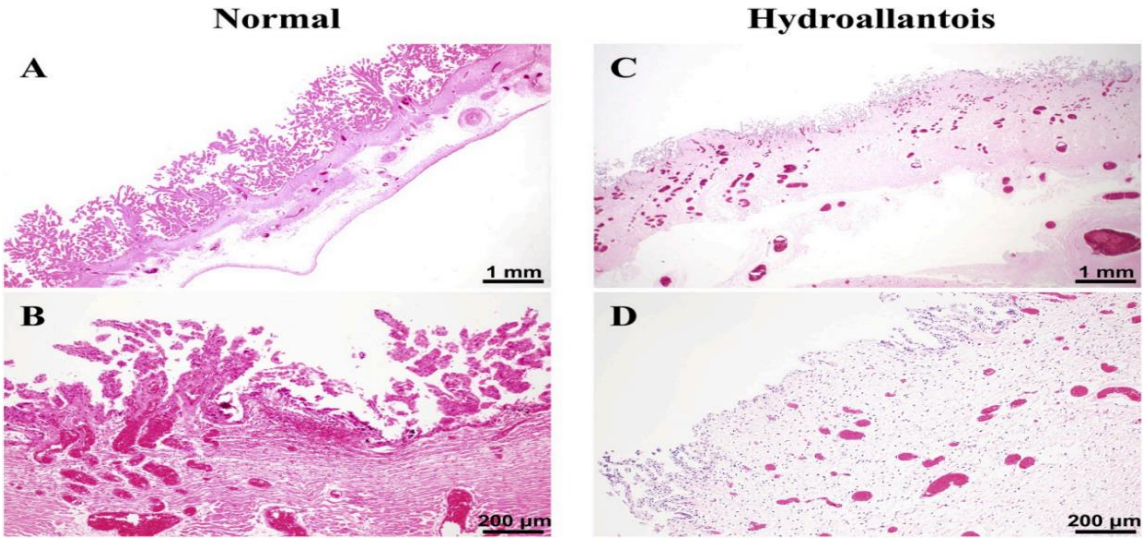

Figure 1: *Histologic alterations linked to hydrallantois in the mare are seen in* ***Fig.1****. A and B Chorioallantoic membrane in horses: normal. Many chorionic microcotyledons held up by a loose chorionic stroma define the chorionic surface. Age at gestation: nine months. 20X, 100X, and H&E. D and C. Chorioallantoic membrane of horses obtained from a hydrallantois case. The chorionic stroma is fibrotic and edematous, and the chorionic surface is distinguished by noticeably diminished chorionic microcotyledons. Age at gestation: ten months. 20X, 100X, and H&E.*

There are two main hypothesized etiologic diseases that lead to hydroallantois: either impaired trans-placental absorption or increased fluid generation . Furthermore, it is possible to consider fibrinoid necrosis of blood vessels and chorionic arterial thrombosis as the etiology of abnormal placental function (Ahuja *et a*l.,2020). The placenta in this instance was like wise dysfunctional and displayed compensatory caruncle enlargement. It was determined that, as long as fluid from the allantoic sac is evacuated gradually, cloprostenol and dexamethasone are useful for inducing parturition in animals with hydroallantois.

In the current investigation a correlation between the extracellular fluid buildup (edema) in the placenta of hydroallantoic patients and decreased capillary density. Hypoxia is known to result from a reduction in capillaries in the chorioallantois, which restricts gas exchange between the mother and fetus. The hypoxic event modifies the expression of placental genes (VEGF, VEGFR1, ANGPT1, and eNOS), increasing the permeability of the existing arteries in the placenta and causing edema. Abnormal vessel function (high permeability), abnormal vascular formation

(reduced columnar density), edema, and hypoxia may result in the placenta's decreased ability to exchange fluid between the fetus and the dam, which would cause an accumulation of extra fluid (hydrops formation; **Fig 2**). It is necessary to do additional research to clarify the primary cause and underlying mechanism of the aberrant angiogenesis in the placenta.

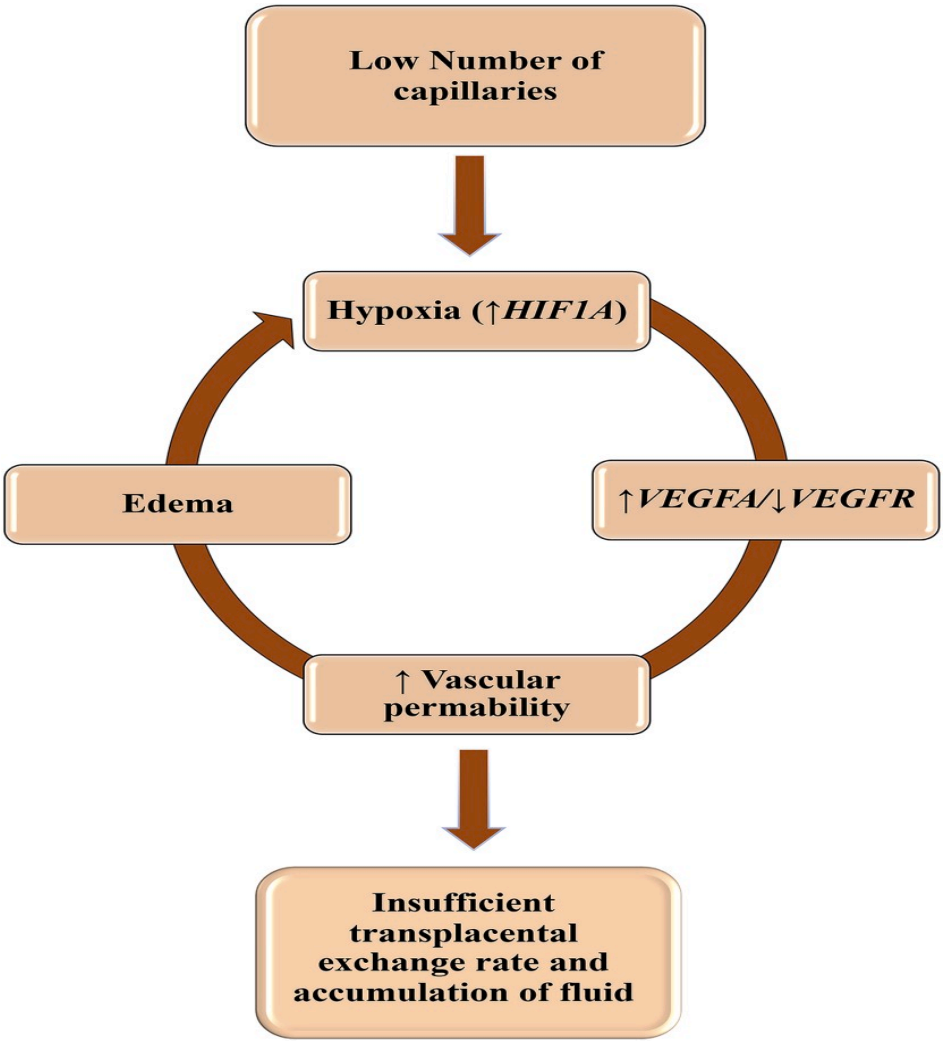

Figure 2: A schematic representation of the potential pathogenicmechanisms that may be involved in the development of hydrallantois.(P. Dini *et al*.,2018)

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