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Case Report

# Fetal abdominoschisis and chondrodysplastic dwarfism in hydroallantoic buffaloes

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## Abstract

We describe 2 rare cases of hydroallantois in buffaloes, each associated with a distinct severely malformed fetus (abdominoschisis and chondrodysplastic dwarfism, respectively). Both buffaloes, in advanced pregnancy, were presented with progressive abdominal distension, anorexia, and reduced ruminal activity. Transrectal examination revealed excessive allantoic fluid accumulation with no palpable fetal parts. Pregnancy was terminated followed by controlled drainage of allantoic fluid and supportive therapy. Biochemical analysis indicated compromised fetal metabolism; however, buffaloes recovered fully without postpartum complications.

**Keywords:** Abdominoschisis, buffalo, chondrodysplastic, fetal monsters, hydorallantois

## Introduction

Hydroallantois is a pathological condition of pregnancy characterized by excessive and rapid accumulation of 40-160 litres allantoic fluid within the allantoic cavity,[1] typically occurring within 5-20 days prior to parturition.[2] This condition arises due to fetal membranes functional or structural abnormalities, particularly allantochorion, and leads to marked bilateral abdominal distension of the dam.[3] Hydroallantois is infrequently diagnosed and is observed across multiple species, with the highest incidence reported in cattle and buffaloes.[4] Occasional cases have also been documented in mares,[5,6] in an ewe,[7] and a dog.[8] In severe cases, failure to diagnose and treat the condition at an early stage may lead to recumbency with guarded prognosis. Although hydroallantois occurs infrequently in cattle, it is often associated with concurrent fetal and fetal membrane abnormalities.[9] Pathophysiology of hydroallantois is primarily due to reduced placental vascularization, leading to functional impairment of placentomes and fluid accumulation within the allantoic sac. It may also result from fetal anomalies, including hepatic or renal dysfunction and umbilical cord torsion.[10,11] Hydroallantois accounts for 85-90% of bovine dropsical conditions, often characterized by non-functional caruncles in one uterine horn and compensatory enlargement of remaining placentomes.[3] Fetal monstrosities, severely malformed fetuses are attributed to a combination of genetic mutations and environmental insults.[12] Although, the overall incidence is relatively low (~ 0.5% in domestic animals[13]) significantly higher prevalence of 7.9-12.8% has been documented in river buffaloes.[14] We describe the clinical presentation and successful management of 2 cases of hydroallantois in buffaloes, each associated with distinct severely malformed fetuses (abdominoschisis and chondrodysplastic dwarfism, respectively).

## Case presentation

Both referred buffaloes were in advanced pregnancy (> 240 days) and presented with a history of progressive to sudden bilateral abdominal distension (Figures 1 and 2), accompanied by anorexia, reduced water intake, and scanty fecal output. Animals had shallow, laboured respiration, and reduced ruminal motility. Both animals were artificially bred (semen from the same bull) and had a history of normal previous parturitions.

### Case 1

A 9-year, multiparous Murrah buffalo on day 260 of pregnancy was presented with a sudden onset of bilateral abdominal distension noticed over the last 10-12 days. Animal had signs of

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systemic compromise, including dullness, dry muzzle, congested mucous membranes, and shallow respiration with no signs of parturition. Transrectal palpation revealed shortened cervix (suggestive of early cervical changes pertaining to parturition) and a severely distended uterus that occupied most of the abdominal and pelvic cavity; fetal parts were not palpable. Vaginal examination revealed a closed cervix.

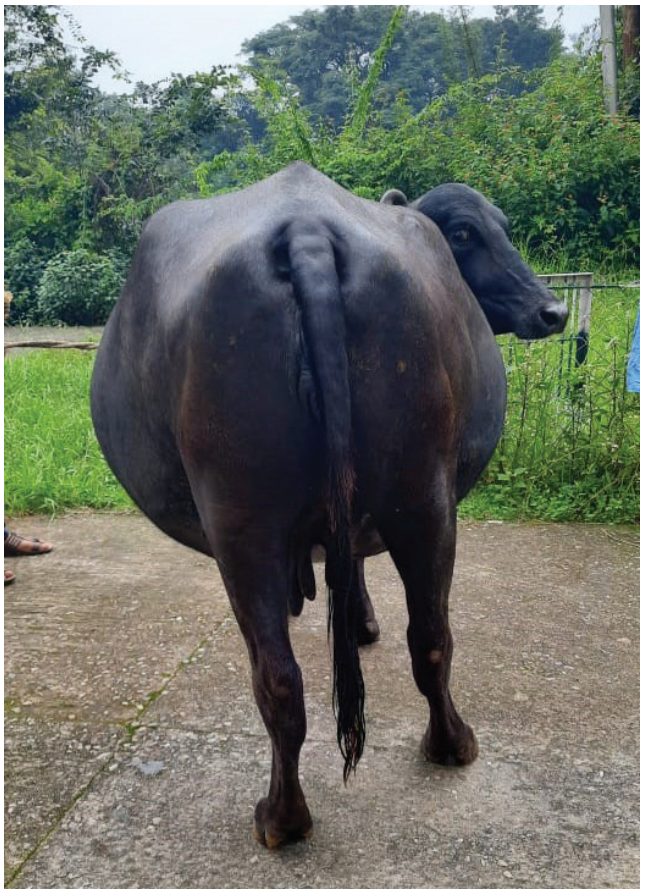

**Figure 1.** Bilateral abdominal distension in a buffalo; Note: symmetrical enlargement of the flanks (Case 1)

## Case 2

A 6-year, Murrah-crossbred buffalo on ~ day 255 of pregnancy was presented with a gradual abdominal distension progressing over 20-25 days. Owner reported reduced appetite but no acute signs of distress. Transrectal examination confirmed a grossly distended uterus filled with fluid, with no palpable fetal structures. Vaginal examination revealed a firm and closed cervix with no indication of impending parturition.

## Treatment

Hydroallantois was the presumptive diagnosis in both cases, based on consistent clinical presentations and diagnostic findings. Our goal was to preserve dams' survival. Parturition was induced with 32 mg of intramuscular dexamethasone sodium phosphate (Dexona®; Zenex Animal Health, Ahmedabad, India) and 25 mg of intramuscular dinoprost tromethamine (Lutalyse®; Zoetis, Mumbai, India). In both cases, cervical dilation commenced within 5-6 hours; allantois was punctured to gradually drain allantoic fluid with a 16 French foley catheter. Initially ~ 60-80 liters of fluid was drained. To prevent hypovolemic shock, buffaloes received intravenous fluid therapy consisting of 5 liters each of Ringer's lactate and normal saline. Supportive medication included 5 grams intramuscular streptopenicillin (Dicrysticin®; Zenex AH) and fluid therapy. Second stage of parturition (visible abdominal and uterine contractions) was observed (during fluid therapy) after ~ 15 and 17 hours of induction in Cases 1 and 2, respectively. Allanotoic fluid was collected for electrolytes estimation (Table). In Case 1, ~ 120 liters of allantoic fluid was expelled during an interval of 12-15 hours. A dead female fetus, exhibiting kyphosis at the second last thoracic vertebra and abdominoschisis with externalized viscera (Figure 3), was extracted manually. Fetal membranes were manually removed after 42 hours of induction of parturition with gentle teasing and avoiding uterine hemorrhage; had numerous small-sized cotyledons and hypoplasia. In Case 2, ~ 100 liters of allantoic fluid were drained in a controlled manner. A dead, malformed female fetus identified as chondrodysplastic dwarf (Figure 4) was removed manually; fetus had short limbs, broad thorax, flat face with mandibular prognathism, bulging eyes, and fluid-filled abdomen (Figure 5).

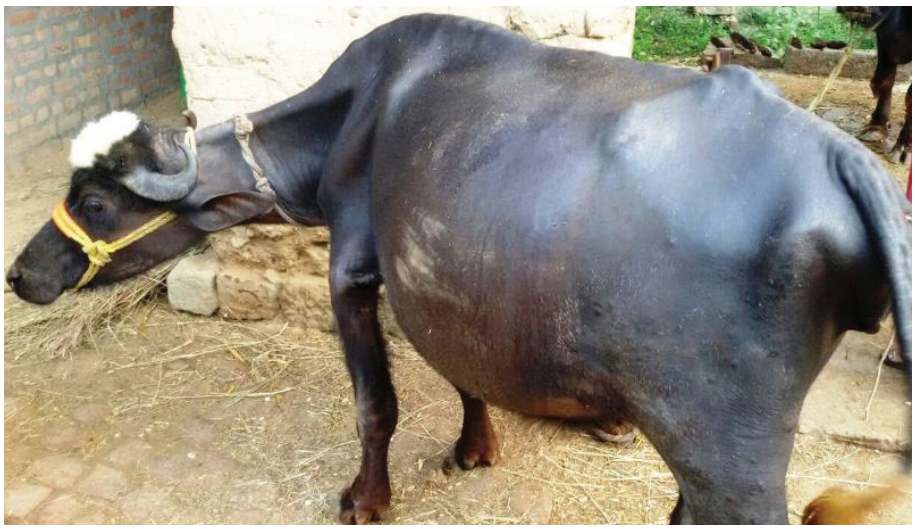

**Figure 2.** Marked abdominal distension observed from left lateral side (Case 2)


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**Table.** Summary of clinical, biochemical and therapeutic findings

| Parameter | Case 1 | Case 2 |
|---|---|---|
| **Age/parity** | 9 years/multiparous | 6 years/multiparous |
| **Stage of pregnancy at presentation** | ~ 260 days | ~ 255 days |
| **Onset and duration of abdominal distension** | Sudden; 10-12 days | Gradual; 20-25 days |
| **Vital signs** | Temperature: 100°F; heart rate: 114 bpm; respiratory rate: 32/minutes; dehydration: 3% | Temperature: 102.2°F; heart rate: 100 bpm; respiratory rate: 37/mins; dehydration: 2% |
| **Transrectal examination** | Markedly distended uterus; no palpable fetal parts | Markedly distended uterus; no palpable fetal parts |
| **Cervical findings** | Closed but shortened | Closed and firm |
| **Initial fluid drained** | ~ 60-80 liters | ~ 60-70 liters |
| **Total fluid drained** | ~ 120 liters | ~ 100 liters |
| **Electrolytes of allantoic fluid** | | |
| **Electrolytes (reference range)** | **Case 1** | **Case 2** |
| Sodium (142.85 ± 10.1 mmol/l) | 135.4 mmol/l | 140.2 mmol/l |
| Potassium (4.67 ± 1.21 mmol/l) | 4.77 mmol/l | 4.52 mmol/l |
| Chloride (92.99 ± 6.72 mmol/l) | 95.2 mmol/l | 93.1 mmol/l |
| **Biochemicals of dams' plasma** | | |
| **Biochemical (reference range)** | **Case 1** | **Case 2** |
| Total protein (normal: 5.5-8 grams %) | 0.11 grams | 1.7 grams |
| Bilirubin (1-2 mg%) | 0.66 mg | 1.1 mg |
| BUN (12-25 mg%) | 29.4 mg | 30.0 mg |
| Glucose (45-75 mg%) | 30.7 mg | 20 mg |
| Creatinine (1-2 mg%) | 1.05 mg | 0.8 mg |
| **Parameter** | **Case 1** | **Case 2** |
| **Fetal diagnosis** | Abdominoschisis with kyphosis; externalized viscera | Chondrodysplastic dwarf; short limb, flat face and prognathism |
| **Fetal membranes** | Hypoplastic with small cotyledons | Normal without appreciable gross abnormality |
| **Time to cervical dilation** | ~ 8 hours | ~ 12 hours |
| **Time to puncture allantois for drainage** | ~ 15 hours | ~ 25 hours |
| **Time of fetal delivery** | ~ 30 hours | ~ 38 hours |
| **Time of fetal membranes expulsion** | ~ 42 hours after parturition induction (with gentle teasing) | Immediately after calving; spontaneous |

Despite differences in progression and fetal pathology (Table), both animals responded favorably to parturition induction and supportive management.

Animals were given intravenous tranexamic acid (Texableed®; Vet Mankind, New Delhi, India) at 5 mg/kg BW, 300 ml of intravenous and 150 ml subcutaneous calcium-magnesium borogluconate (Mifex®; Elanco, Thane, India), and slow intravenous oxytocin (100 IU diluted in normal saline).

## Outcome

Complete recovery occurred after 5 days in both buffaloes, with no postpartum complications for 60 days as reported by the owner. Owners were advised for artificial insemination in the next breeding season.

## Discussion

Hydroallantois is a relatively uncommon pregnancy disorder, more frequently reported in cattle but also observed in buffaloes;[15–18] typically manifests during last trimester, particularly between the 8th and 9th months of pregnancy.[3] Pathophysiology of hydroallantois primarily involves rapid and excessive accumulation of allantoic fluid, leading to severe abdominal distension. In many cases, the condition is associated with abnormal placentation, particularly the formation of adventitious placental structures resulting from the absence or dysfunction of maternal caruncles.[3,19] Such changes are often secondary to uterine pathology, including fibrosis or inflammatory conditions that impair normal placentome development and function. Additionally, fetal renal dysfunction, altered membrane permeability, impaired active sodium transport across chorioallantoic membrane, and hormonal

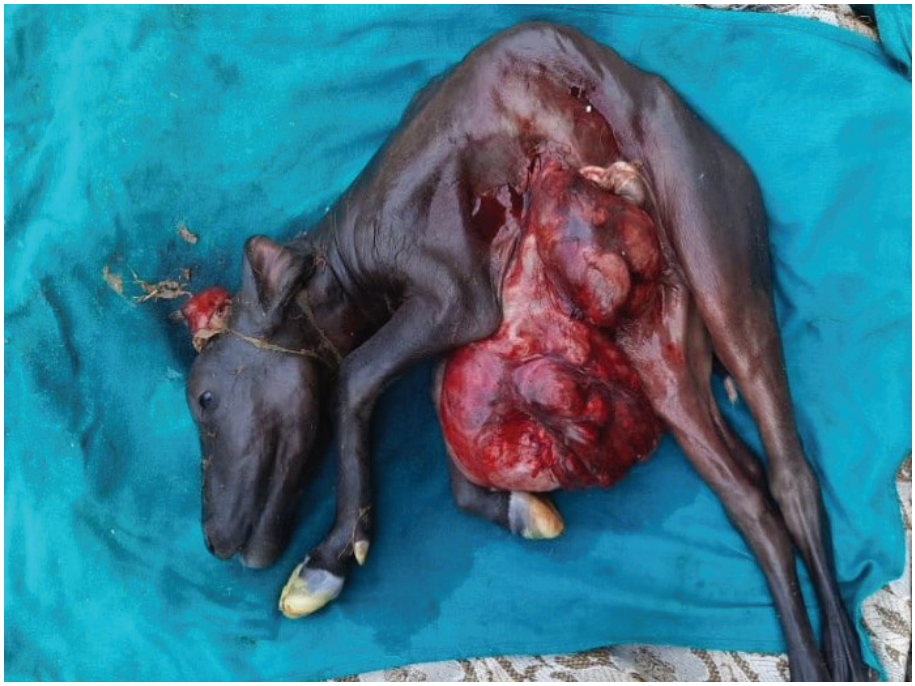

**Figure 3.** Manually extracted dead female fetus exhibiting kyphosis and abdominoschisis

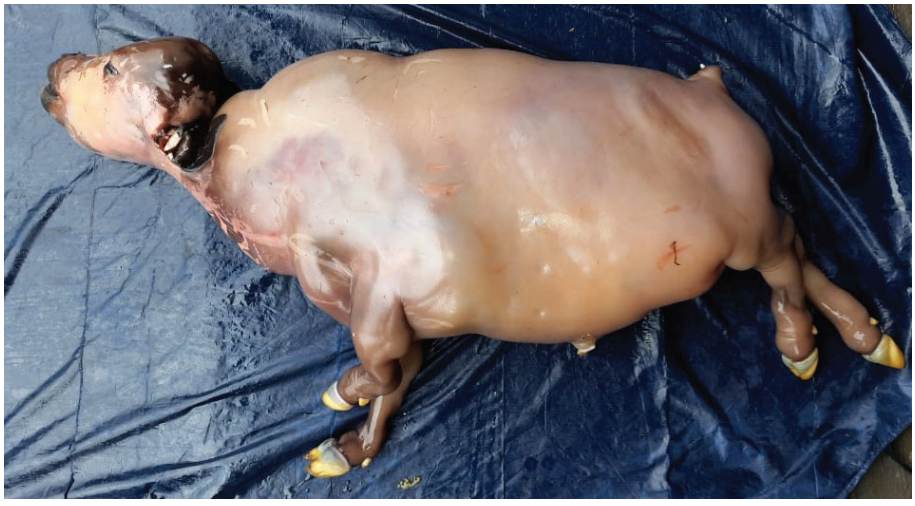

**Figure 4.** Lateral view of chondrodysplastic dwarf monster

imbalance have also been implicated in the etiopathogenesis of this condition.[18]

Clinically, hydroallantois is characterized by a rapid increase in abdominal size, loss of body condition, recumbency, and eventual death if untreated.[1] The condition often results in a compromised fetus, commonly underdeveloped, edematous, afflicted with congenital anomalies such as ascites, or in some cases, apparently normal but nonviable.[3,18,20] Chloride concentrations remain lower than plasma, whereas sodium and potassium exhibit inverse trends.[21] Management of hydroallantois depends on the severity and prognosis. In severe cases, termination of pregnancy is warranted and can be achieved pharmacologically using prostaglandin $F_{2\alpha}$ and corticosteroids.[21,22] However, caution must be exercised as rapid removal of the fluid may result in hypovolemic shock and collapse.[23]

In both cases, tranexamic acid was given empirically after fetal delivery, despite lack of overt uterine trauma or hemorrhage. This decision was based on the application of manual traction during assisted delivery that carries a potential risk of subclinical uterine injury and delayed bleeding, especially in cases involving severe uterine distension and manipulation. Although not routinely indicated for hydroallantois management, tranexamic acid has been used off-label in bovine obstetrics to minimize haemorrhagic risk in high-intervention scenarios. Its empirical use in such contexts has been described in veterinary literature and may be justified as a precautionary measure.[24]

In some hydroallantois cases, associated fetal anomalies such as abdominal wall defects may be present. Abdominoschisis and omphalocele are congenital conditions characterized by herniation of abdominal organs through a defect in the


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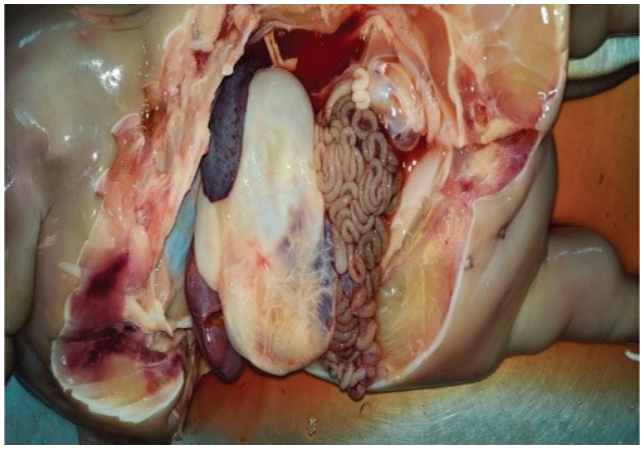

**Figure 5.** Postmortem image depicting fluid filled abdomen along with anasarca

abdominal wall. These defects are detectable via prenatal ultrasonography in humans, allowing for early diagnosis and safer maternal outcomes.[25] Additionally, chondrodysplastic anomalies, such as the bulldog calf phenotype, are often genetically inherited and have been well-documented in specific cattle breeds like Dexter. These conditions are typically attributed to disruptions in fetal pituitary development and function.[26] Antepartum ultrasonography was not performed to assess placental or fetal abnormalities due to referral hospital facility and histopathological examination of fetal membranes was not conducted. Lack of genetic testing, pedigree analysis, or follow-up on other offspring from the same sire limits interpretation of a heritable cause.

## Conclusion

The cases highlighted a rare association of hydroallantois with severely malformed fetuses (abdominoschisis and chondrodysplastic dwarfism) in buffaloes. Successful management required timely diagnosis, controlled fluid drainage, and supportive therapy. Same breeding bull used in both cases suggested a possible genetic association. Importance of vigilant monitoring in advanced pregnancy and the need to consider genetic factors in recurrent fetal anomalies and monsters is emphasized.

## Learning points

- Hydroallantois, although rare, can be associated with severe fetal monsters such as abdominoschisis and chondrodysplastic dwarfism in buffaloes, indicating a possible shared pathophysiological or genetic basis.
- Prompt identification of hydroallantois through clinical and transrectal examination, followed by pharmacological induction, gradual fluid drainage, and supportive care, can ensure successful outcomes and full recovery of the dam without postpartum complications.
- Genetic link from natural service with the same breeding bull is possible. Both buffaloes were bred by the same bull via natural service and therefore a heritable component cannot be excluded. However, no genetic testing was performed, and this association remains speculative.

## Conflict of interest

Authors declare no conflict of interest.

## Authors contribution

Diagnosis and treatment: PS, PK, and Akshay S; after calving management: DY and DT; all authors contributed to manuscript writing.

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