Short Communication

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Dystocia Due to Hydrocephalic Teratology through Mutational Operations in a Cross-Bred Jersey Cow

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ABSTRACT

The present case depicts a case of primiparous cross-bred Jersey cow affected with hydrocephalic teratology leading to dystocia which was successfully managed by mutational procedures.

Keywords: Intermittent Contractions, Hydrocephalus, Anterior longitudinal, Retropulsion.

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INTRODUCTION

Hydrocephalus is a dropsical condition with the accumulation of fluid in cranium as a result of an imbalance between the formation and drainage of cerebrospinal fluid (CSF) either in the ventricular system or subarachnoid space characterized by marked enlargement of the cranium (Noakes *et al.,* 2009). The condition may be caused by environmental influences during fetal development or genetic predisposition of simple autosomal recessive gene with incomplete penetrance (Jackson, 2004) or acquired by in-utero viral infections such as Bovine viral diarrhoea (Agerholm *et al.*, 2015). Other predisposing factors for this malformation may include deficiency of vitamin-A or any other brain lesions that may cause a disturbance in the cerebrospinal fluid dynamics (Ferris *et al.*, 2011). Jubb and Kennedy (1970) stated that congenital hydrocephalus is known to be inherited in cattle and exacerbated its manifestation by a coexisting hypovitaminosis-A. Death of the fetus is due to pressure on vital centres of the brain. The frontal, temporal and parietal bones are usually involved becoming deformed, separated and thin resulting in improper sculpturing of cranial vault

(Purohit *et al.*, 2012). The condition does not affect foetal development but may result in death of the foetus at birth or soon after birth. The present case reports successful per-vaginal delivery of hydrocephalus foetus after relieving fluid from enlarged head through mutational operations.

CASE HISTORY AND OBSERVATIONS

A five-year-old full term primiparous cross-bred Jersey cow with a history of unable to deliver the fetus in spite of being in labor past 12 hours presented to the LAC-OP-OG a unit of Veterinary Clinical Complex, Veterinary College and Research Institute, Tirunelveli. Owner reported that

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the animal was experiencing restlessness, intermittent contractions for 12 hours and also there was rupture of the water bag after 4 hours of onset of labor but there was no fetal expulsion indicating that the animal was in the second stage of labor. Further, the owner also stated that there were unsuccessful attempts to deliver the fetus. Cow was dull, anxious and exhausted. Clinical examination revealed reduced respiration rate (12 breaths / min), elevated pulse rate (44 / min) with normothermia (38.8°C). Gynaeco-clinical examination revealed oedematous vulva with mucoid cloudy copious vaginal discharge. Per rectal palpation signified the presence of non-viable foetus. Per vaginal examination revealed a fully dilated cervix with anterior dorsal-sacral non-viable foetus with both foetal forelimbs extended tightly engaging the birth canal. Further, palpation of the fetal head revealed abnormally large dome shaped fluid accumulation on poll. These findings provided a positive indication of the case as Hydrocephalus - dropsical condition of foetus. Further examination of fetal limbs revealed ankylosis. Obstetrical manoeuvres were attempted after washing the perineum with diluted potassium permanganate solution (1:1000), epidural anaesthesia was achieved using 2 ml of 2% Lignocaine hydrochloride and 2 litres of Carboxy methylcellulose solution was introduced into birth canal followed by retropulsion of extended forelimbs under the body of foetus to generate space in the birth canal. Further, an incision was made on the soft portion of the distended cranium and the fluid was evacuated using William's long obstetrical hook thus the fetal size was reduced. The foetus was delivered by the application of eye hooks in the medial canthus of the eye along with the hand guided judicious traction. Hence assisted delivery yielded dead female hydrocephalic foetus followed by expulsion of placenta.

Post delivery dam was administered with intrauterine Nitrofurazone – Metronidazole – Urea bolus was placed and provided with supportive therapy consisting of a intravenous fluid (Normal saline solution 3 litres IV for 3 days), antibiotics (Ceftriaxone plus tazobactam 4.5 g IV for 3 days), calcium boro gluconate (450 ml, IV on the day of dystocia), chlorpheniramine maleate (10 ml IM for 3 days) Oxytocin (20 IU IM immediately after foetal expulsion), Non-steroidal anti-inflammatory drug (NSAID; flunixin meglumine 1.1 mg/kg for 3 days) and Oral ecbolics (Liquid. Uterovet 100 ml bid PO for 3 days). Post mortem examination of foetus revealed external hydrocephalus, malformed facial bones, arthrogryposis of fetal limbs, cystic changes in kidney.

Fig. 1: Hydrocephalic calf

Fig. 2: Cystic Kidneys of fetus

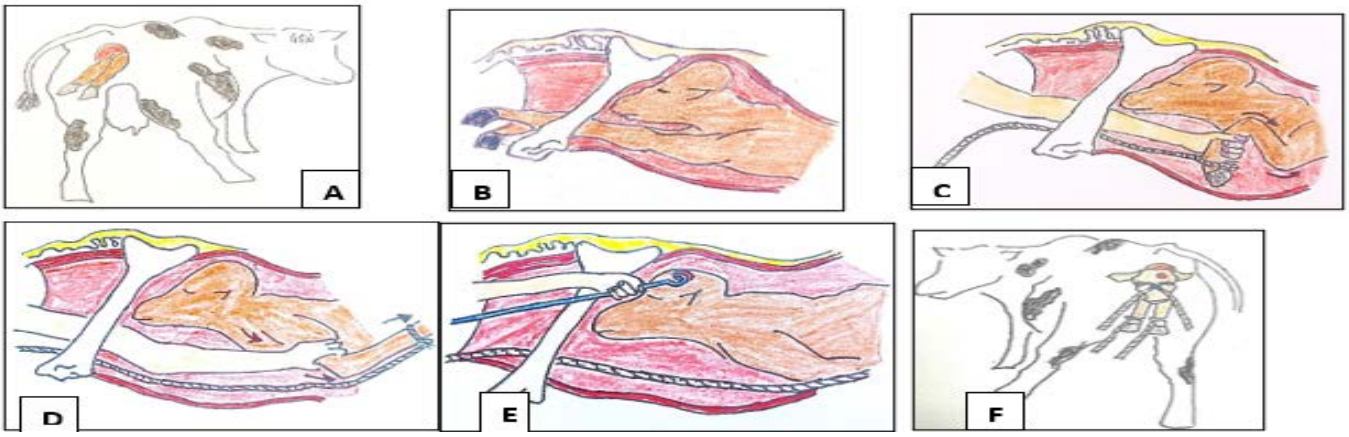

Fig. 3: A. Cattle with both foetal forelimbs presented at vulva. B. Per vaginal examination revealed dome shaped soft swelling on poll. C. Retropulsion of fetal forelimbs. D. Complete repulsion of forelimbs beneath the foetal body. E. Puncturing the dome shaped mass on the pole. F. Forced traction to extract the foetus

TREATMENT AND DISCUSSION

The term hydrocephalus indicates an increase in the cerebrospinal fluid volume within the ventricular system (internal hydrocephalus) or in the subarachnoid space (external hydrocephalus), due to an imbalance between the production, flow and absorption of CSF (Purohit *et al.*, 2006). There are two main causes of hydrocephalus: a loss of cerebral parenchyma (compensatory normotensive hydrocephalus) or an intraventricular obstruction of the CSF flow (obstructive hypertensive hydrocephalus). Compensatory hydrocephalus is generally due to a severe cerebral hypoplasia/ hydranencephaly frequently caused by in utero viral infections. In this form of hydrocephalus, the CSF volume increases to compensate the loss of the parenchyma and the intracranial pressure is normal. Obstructive hydrocephalus is due to an intraventricular obstruction to the CSF flow, preventing communication between the ventricular system and the subarachnoid space thus the CSF pressure increases expanding the ventricular system especially lateral ventricles (Lahunta and Glass, 2009). The hydrocephalus of our case study had accumulation in subdural space hence falls into the compensatory normotensive category.

The pathogenesis of the hydrocephalus is mainly contributed by glymphatic dysfunction in foetal brain caused by congenital or acquired damage to the NKCC1, Aquaporin-1 and Aquaporin- 4 protein transport channels resulting in vasogenic oedema, intracerebral osmotic imbalance and altered CSF production (Bramal *et al.*, 2022).

In congenital hydrocephalus, the increase in the intracranial volume occurs before the sutures of the calvaria have closed, allowing the enlargement of the cranial cavity. Affected animals often show prominent dome-shaped skulls, open sutures and persistent fontanelles. Moreover, there is a high incidence of dystocia due to the enlarged head (Miller 1993; Smith and George 2009). These findings were in concurrence with our case study and was the prime cause of dystocia in the present case.

According to Agerholm *et al.* (2015) teratogenic in-utero viral infections resulting in fetal cranial dropsy is usually accompanied by musculoskeletal lesions which is in concurrence with our findings hence viral origin might be a probable predisposing factor in our case. Most death of a fetus occurs due to the pressure on vital centres of the brain (Purohit et. al., 2012) which was in agreement with our findings.

Purohit *et al.* (2006) reported that dystocia in cows caused by hydrocephalic foetuses can be delivered per vaginum by excision or aspiration of accumulated fluid in the fetal head followed by traction. In this case, craniotomy with William's long Obstetrical hook followed by evacuation of accumulated fluid and then by forced traction, fetus was delivered per vaginally. As per Wodarczyk *et al.* (2009) Polycystin-1 (PC-1), the product of the PKD1 gene, mutated in the majority of cases of Autosomal Dominant Polycystic Kidney Disease results in Ependymal dysfunction and altered choroid plexus ciliogenesis thus predispose to hydrocephalus. This finding correlates with the post mortem findings of this case.

CONCLUSION

This case study concludes that dystocia due to dropsical fetus was relieved successfully by incising the soft portion of hydrocephalic head thereby reducing the fetal size and combining the obstetrical procedures of mutation (retropulsion) and forced traction.

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CONFLICT OF INTEREST

None

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