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Review Article

Cranium bifidum with meningocele in ruminants: a clinical insight

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ABSTRACT

Cranium bifidum is an incomplete closure of the skull at the suture lines, leaving a space through which the intracranial tissues can protrude. Meningocele is the herniation of the meninges containing cerebrospinal fluid through the cranium bifidum. This congenital defect is attributed to the intake of certain poisonous plants, genetic abnormalities, hereditary factors, malnutrition, bovine viral diarrhea, and others. The obvious clinical sign is a floating saccular protrusion of varied size and volume and is typically associated with cranium bifidum in the frontal or parietal region. Surgical management involves exposing the effective cranial opening and suturing the dura mater in a simple continuous pattern. Prognosis depends on the presence or absence of neurological signs; neurological signs are associated with morphological changes in brain tissue with poor prognosis.

Keywords: Cranium bifidum, Meningocele, Neural tube, Skull, Cerebrospinal fluid, Neurological sign

INTRODUCTION

Congenital malformations develop during embryogenesis in all species of animals and are characterized by morphological and functional impairment in tissues or organ systems.[1] Cranium bifidum is a deformity that occurs during the development of an embryo and results in incomplete closure of the skull at the suture lines, leaving a space through which the intracranial tissue can protrude.[2,3] Meningocele is the herniation of the meninges containing cerebrospinal fluid through the cranial defect and together with brain tissue known as encephalomeningocele.[3,4] Meningocele clinical sign in cattle is a floating saccular protrusion of varied size and volume and is associated with cranium bifidum in the frontal or parietal region.[5,6]

Diagnosis of cranium bifidum with meningocele can be achieved through physical examination and imaging methods like radiography, ultrasonography, computed tomography, and magnetic resonance imaging.[7] In some cases, the prognosis for surgical reduction is favorable.[5] This paper highlights the aetiology, morphogenesis, overview of the anatomy of the skull vault, clinical findings, radiographic and sonographic findings, mass fluid sample analysis, surgical management of the defect in ruminants, and the prognosis of surgical reduction.

AETIOLOGY

Cranium bifidum with meningocele is caused by varying aetiological factors and they include the intake of certain poisonous plants such as *Mimosa tenuiflora*, and *Poincianella pyramidalis*, as well as genetic abnormalities, such as autosomal recessive genes and hereditary factors.[1,2,4,8] Other aetiologic factors include malnutrition, bovine viral diarrhea, excessive pressure during rectal examination of a pregnant dam, and griseofulvin administration during pregnancy.[2,9]

MORPHOGENESIS OF CRANIUM BIFIDUM

The morphogenesis of cranium bifidum with meningocele or encephalomeningocele begins with a focal failure in the closure of the neural tube during fetal development.[10] This defect then precipitates failure in the development of skeletal encasement around the frontal, parietal, and occipital bones.[2,11] Thus, creating an opening for the protrusion of meninges (meningocele) alone or together with brain tissue (encephalomeningocele).

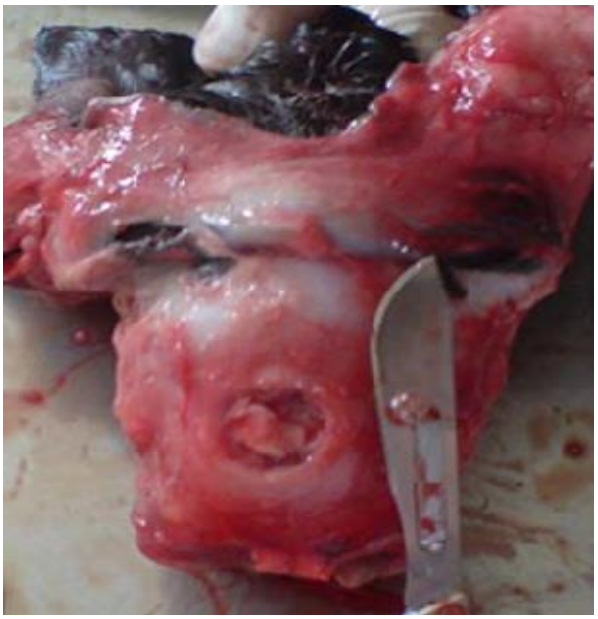

Figure 1: A cranial elliptical defect on the median parietal line.[19]

Figure 2: A fluid-filled meningeal sac that protruded through a cranial defect.[19]

OVERVIEW OF THE ANATOMY OF SKULL VAULT

The mammalian skull vault is an assembly of frontal, parietal, and a contribution from the squamous part of the occipital bone. These bones develop from different ossification centers derived from tissues of different embryonic origins.[12]

The occipital bone is made up of the basilar, squamous, and lateral sections, and all together create the foramen magnum, a ring that surrounds the spinal cord. The frontal bone is paired and is situated between the cranium and the face. The parietal bone is paired and forms most of the dorsolateral part of the cranial wall, it is bordered by the occipital bone caudally and the frontal bone rostrally.[13]

CLINICAL FINDINGS

Clinical findings in a crossbred jersey calf showed a turgid, fluid-filled swelling in the cranium extending from the central part of the forehead to a few centimeters above the ear. Aseptic puncture with a needle enabled drainage of colorless fluid with deep palpation after drainage revealing an elliptical median opening on the skull.[14]

A case report in a newborn calf showed an extensive swelling at the frontal region extending from the supraorbital curvature of the frontal bone to the end of the nasal bones. In addition, there were opisthotonos, spastic paresis in the thoracic limbs, and flaccid paresis in the pelvic limbs.[15] A case report in a lamb showed the animal in lateral recumbency, unable to lift its head and stand with a fluid-filled sac-like structure about 7 cm in diameter at the occipital region.[16]

RADIOGRAPHIC AND SONOGRAPHIC FINDINGS

Radiography in a newborn calf showed a round and well-define mass with radio-opacity typical of soft tissue, positioned externally and in close contact with the skull at the frontal bone. Sonography showed hypoechoic tissue with a lack of frontal bone integrity.[15]

Radiography in a lamb illustrated the caudal region of the skull with decreased bone opacity due to a defect on the cortex of the interparietal bone. Sonography revealed a sizable anechoic mass that resembles a duct and is linked to the cranium.[16]

MANAGEMENT OF CRANIUM BIFIDUM WITH MENINGOCELE

Premedication and anesthesia

Premedication is commonly achieved with xylazine at a dosage of 0.01 mg/kg body weight.[14] Anaesthetic induction is commonly achieved with midazolam at 0.4 mg/kg, propofol at 4 mg/kg, and isoflurane to maintain the anesthesia.[15]

Table 1: Values reported in the analysis of a mass fluid sample compared to normal values of a CSF sample.[4,14]

Components of mass fluid	Obtained value of the mass fluid analysis	Normal value range of a CSF sample
Specific gravity (mg/dl)	2.052	8-70
Glucose (mg/dl)	49	48-109
Total protein (mg/l)	751	18-28,084
Total cells (l)	38.0 × 10	11.9 ×10^6-12,310.0× 10^6
Neutrophils (%)	4.0	0.0-97.0
Monocytes (%)	50.0	1.0-96.0
Lymphocytes (%)	22.0	1.0-73.0

Surgical technique

Where surgery is indicated such as when there is no brain tissue herniation or evidence of neurological sign, the surgical technique starts with an elliptical skin incision on the base of the projecting sac with continuous dissection through the interior to reach the meningeal membranes and expose the cranial defective opening.[14] The cranial defect on the frontal bone measures 4.5 cm in diameter.[15] The protruding extra meningeal membranes are removed.[14]

The defect is closed by suturing the dura mater using size 0 catgut in a simple continuous pattern, the subcutaneous tissue is closed in a continuous mattress pattern, and the skin is closed using nylon size 1[15] or braided silk in a simple interrupted pattern.[14]

Postsurgical management

Treatment of choice post-surgically include, Streptopenicillin and Meloxicam for 5 days.[14] And ceftiofur, flunixin meglumine, dexamethasone, furosemide, and mannitol for 3 days.[15]

Prognosis

Sutures are removed 10 days postoperatively and no recurrence after 6 months in the crossbred jersey calf.[14] In the newborn calf with neurological signs, euthanasia was conducted on the third postoperative day and necropsy revealed atrophy of the diencephalon and corpus callosum and the absence of third and fourth ventricles.[15]

DISCUSSION

Other successful surgical management of cranium bifidum with meningocele are reported in a calf and a lamb with no reoccurrence. Meningocele's clinical symptoms are typically seen in newborn calves, and pathology is not documented in aborted animals.[15-18] The condition can be managed successfully depending on the presence or absence of neurological signs. The presence of neurological signs is associated with morphological changes in brain tissue with poor prognosis.[18] The use of an ear cartilage graft to surgically reduce a calf's 7-cm cranium bifidum resulted in a poor prognosis nine days after the procedure. Encephalitis and purulent intracranial material were discovered during necropsy.[6] Successful surgical reduction without the use of grafts was achieved by closing a 7-cm circular gap in the frontal bone using a simple continuous pattern of nylon suture, followed by dehorning.[5]

CONCLUSION

Cranium bifidum and meningocele have been widely studied in recent times, mostly as case reports. The etiology varies from the consumption of certain plants to genetic and hereditary factors and others. Neural tube defect is essential to the morphogenesis of cranium bifidum and meningocele. The outcome of surgical intervention depends largely on whether the affected animals exhibit neurological signs or not. The use of homologous material such as ear cartilage in the reduction of the defect is controversial.

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