# Spina bifida in a dead stillbirth mongrel dog

# Espinha bífida em um cão natimorto sem raça definida

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### **Abstract**

Spina bifida is a congenital spinal deformity that is characterized by an incomplete closure of the dorsal portion of the vertebrate. This report describes the radiological and pathological findings of this abnormality in a dead still birth mongrel. The dog demonstrated a cleft at the dorsal region of the thoracic vertebrate column, which resulted in exposition and protrusion of the spinal cord and the meninges. Plain radiographs of the ventral-dorsal of the vertebrate column revealed a defect in the fusion of the dorsal spinous processes of the distal thoracic vertebrates (T6-T13); while the right-lateral view revealed xyphosis of the entire lumbar region of the spine.

Key words: Spina bifida, radiology, pathology, dog

### Resumo

A espinha bífida é uma deformidade congênita caracterizada pelo incompleto fechamento da porção dorsal de uma ou mais vértebras. O presente caso descreve as alterações radiológicas e patológicas de um cão natimorto sem raça definida. O animal apresentava uma fissura nas vértebras torácicas, resultando em exposição e protusão da medula espinhal e meninges. O exame radiográfico simples em posição ventro dorsal das vértebras revelou defeito na fusão do processo espinhoso das vértebras torácicas (T6-T13), enquanto na radiografía lateral foi observada cifose em toda região lombar.

Palavras-chave: Espinha bífida, radiologia, patologia, cão

#### Introduction

Spina bifida refers to a congenital abnormality of the spinal column that is characterized by an incomplete closure of the dorsal portion of one or more vertebrates (CLAYTON; BOYD, 1983; JUBB; HUXTABLE, 1993), that is often accompanied by protrusion of the spinal cord or its membranes (BRAUND, 1994). Although the

embryonic pathogenesis of this disorder has not been totally elucidated (BRAUND, 1994), hyperplasia of the cells from the dorsal neural tube that affects fusion of neural tube and vertebral arches and/or a vascular defect that restricts or hinders blood flow to the dorsal region of the vertebrate column have been suggested as possible causes (CLAYTON; BOYD, 1983; BRAUND, 1994).

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This dysraphic condition has been described in the dog, cat, cow (JUBB; HUXTABLE, 1993), and even in lions (GALLOWAY et al., 2002). However, a relatively high incidence of spina bifida has been associated with young English bulldogs and in Manx cats (WALKER, 2002), while the disorder is considered congenital in Beagles (BRAUND, 1994). Brachycephalic dogs are more frequently affected by this defect relative to their dolicephalic counterparts (JUBB; HUXTABLE, 1993).

Clinically, the disorder is recognized when affected animals ambulate; these may demonstrate pelvic limb ataxia and paresis, fecal and urinary incontinence, perineal analgesia, and flaccid anal sphincter (BRAUND, 1994). Diagnosis is based on the macroscopic presentation associated with plain radiographs, myelographs, and computerized tomography (BRAUND, 1994; WALKER, 2002).

Reports of this disorder in the dog have not been recently published. Therefore, this article is opportune as it describes the gross and radiographic findings of spina bifida in a full-term stillbirth female mongrel dog.

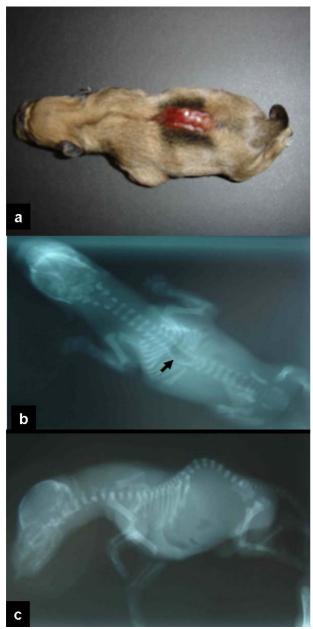
## Case history

A 6 year-old mongrel bitch was attended at the Veterinary Teaching Hospital, Faculdade Integrado de Campo Mourão, Campo Mourão, Paraná, Brazil. The owner indicated that the bitch, with almost 62 days of gestation, had difficulty during parturition and was in delayed labor for almost 12 hours. Clinically the animal was prostrated, moderately dehydrated, with abdominal sensibility, and presented a greenish foul-smelling vaginal secretion. Abdominal palpation revealed a fetus. An emergency caesarian section was realized; a female dead stillbirth fetus with a bone abnormality at the thoracic region was seen and subsequently removed. The dog has since recovered, has been discharged, and is doing well.

The dead female stillbirth mongrel dog weighed 250 g. At necropsy, a cleft (1 x 5 cm) was observed at the dorsal region of the thoracic vertebrate column, which resulted in exposition and protrusion of the spinal cord and the meninges (Figure 1a); all other organs and systems were normal. Ventral-dorsal plain radiographs of the of the vertebrate column revealed a defect in the fusion of the dorsal spinous processes of the distal thoracic vertebrates (T6 - T13); while the right-lateral view revealed xyphosis of the entire lumbar region of the vertebrate column (Figure 1b and c). Radiographs of the head, cervical vertebrates, and of the fore and hind limbs revealed normal bone development and opacity. Tissues were processed for routine histopathology; however histological evaluation of the spinal cord and other tissues were distorted by initial post-mortem autolysis.

#### **Discussion**

In this case, a diagnosis of spina bifida, which occurred through vertebrates T6 to T13, was based on gross presentation of the cleft, formed by incomplete fusion of the dorsal spinous processes, and confirmed by radiographic findings that are consistent with this abnormality (BRAUND, 1994; WALKER, 2002). Although myelographs and computerized tomography (BRAUND, 1994; WALKER, 2002) in addition to plain radiograph have been recommended for a diagnosis of this defect, the former two methods were not needed in this case. since the abnormality was identified by gross observation and confirmed by radiographic evaluation. This report also contributes to the incidence of spina bifida in mongrels; cases in mongrels have been previously described (GEIB; BISTNER, 1967); while incidence of this abnormality is elevated in young English bulldogs (BRAUND 1994), and the Rotweiller breed of dogs may have a genetic predisposition for spinal abnormalities (JURINA; GREVEL, 2004).



**Figure 1.** Spina bifida, stillbirth mongrel dog. Note cleft with exposition of spinal cord at thoracic vertebrates (a). There is absence of several dorsal spinous processes (arrow) of the thoracic vertebrates (b), and xyphosis of lumbar region (c).

This bone defect can be also classified as spina bifida cystica associated with meningomyelocele. In some of these cases, the anteroposterior part of the cutaneous lesion may not correspond exactly to the bone deformity, due to differences in growth and development of the ventral and neural axes (JUBB;

HUXTABLE, 1993); however, this was not observed in this stillbirth dog. The bone abnormality herein described has been related to failure of dehiscence of the neural crest from surface ectoderm resulting in a central area without the corresponding epithelial protection (JUBB; HUXTABLE, 1993). The cleft may involve most of the vertebral arch or only the dorsal spinous process (BRAUND, 1994), can be focal or multifocal (SHAMIR; ROCHKIND; JOHNSTON, 2001), and could be observed in cervical or thoracic vertebrates (KEALY; MCALLISTER, 2000). Although cases are more frequently described in the lumbosacral region (JUBB; HUXTABLE, 1993; BRAUND, 1994), in this case the abnormality occurred within the thoracic region.

Based on the severity of the deformation, this abnormality can be divided into: a) *myeloschisis*, a divided spinal cord; b) *spina bifida occulta*, characterized by bone defect without visible protrusion of structures within the vertebral canal; and c) *spina bifida cystica*, in which there is a cystic protrusion that may contain skin, dura mater, and condensed meninges (meningocele) or meninges and spinal cord, known as meningomyelocele (JUBB; HUXTABLE, 1993; BRAUND, 1994).

There is no known effective treatment for spina bifida in veterinary medicine; prognosis may vary between restricted to poor (BRAUND, 1994). Nevertheless, a partially successful surgical intervention was realized in a dog with similar lesions (SHAMIR; ROCHKIND; JOHNSTON, 2001); the authors indicated that the dog recovered from the spinal disorder but fecal incontinence and urinary dribbling persisted. However, in human medicine spina bifida is a severe public health concern and is treated by prevention. The frequency of this neural tube defect in the USA has been reduced due to the daily intake of 400 µg of folic acid during or before pregnancy, and has resulted in lower levels of still births, fetal deaths, and elective terminations (MERSEREAU et al., 2004). Therefore, attempts should be made to evaluate the effects of the

administration of folic acid to gravid domestic animals when this abnormality is diagnosed during gestation thereby reducing the incidence of this malformation in dogs.

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