

## Evaluation on Blood Chemistry in a Thoroughbred Neonate (1-day old) with Congenital Torticollis

### 선천성 사경을 동반한 더러브렛 신생망아지의 혈액화학 평가

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#### Abstract

There have been no reports on the laboratory screening of congenital torticollis in equine medicine. The purpose of this study was to evaluate the blood chemistry of a newborn female foal with congenital torticollis. The filly was born after a normal parturition period, and the clinical pathology results were as follows: biochemistry of the plasma showed significantly higher levels of CK, LDH, and Na than those in a normal foal. However, Cl level was remarkably lower than that of the control. In conclusion, the cells in the neck muscles influenced the clinical pathological value in the neonate. A disorder of the muscles as well as the bone problem, such as the cervical vertebrae, may have caused congenital torticollis. To our knowledge, this is the first report detailing the clinical pathology results in a newborn foal with torticollis. These laboratory results can be used as a reference for interpretation of pathology results in foals.

**Key Words** : Biochemistry, Creatine kinase, Lactate dehydrogenase, Thoroughbred neonate, Torticollis

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## I. Introduction

Congenital malformations of the spine of the horse are rarely observed to occur on their own and usually are combined with limb contracture. The cause of these deformities are unknown, and the severity determines the prognosis because there are no treatments described (Rooney, 1966).

Contracted foal syndrome describes a variety of curvatures and contractions of the limbs (arthrogryposis), vertebral column (scoliosis, kyphosis, wry neck), and asymmetric malformation of the nose (wry nose) (Green, 1995). Scoliosis is a lateral deviation of parts of the spinal column. Some foals may be born with apparently severe deformities of scoliosis, lordosis or kyphosis but may recover spontaneously. In some cases, however, the defects are severe and unresolvable. The cause is uncertain but malpositioning in utero has been blamed (Knottenbelt *et al.*, 2004).

Torticollis is a wryneck. Early recognition of abnormalities is of utmost importance for the successful management of neonates. Congenital torticollis is seen occasionally resulting from malformation of more caudal cervical vertebrae (Wilkins, 2010). There is no specific treatment (Green, 1995).

Many of the problems of the newborn foal have their genesis in utero (Wilkins, 2010). Congenital defects or abnormalities in foals are poorly described from an epidemiological perspective. Genetic and congenital diseases are a very important consideration for the equine industry, particularly in breeds where the genetic pool may be narrow and where genetic etiologies have been postulated (Cruz, 2011). With congenital abnormalities genetic influences are often suspected, but difficult to substantiate. Effects of nutrition, toxins, infections can have profound effects on genetic expression (Madigan, 1987).

Clinical pathological findings of neonates or foals have been reported previously (Cowell, 2004; Yang *et al.*, 2011). However, to our knowledge, there has been no report detailing the clinical pathological data of a neonate with torticollis. Therefore, the purpose of this study was to determine the blood chemistry values of a newborn foal with torticollis.

## II. Case

A Thoroughbred filly was born after a normal parturition period of 341 days. The patient's dam had no history of drug administration or any abnormal occurrences during pregnancy. However, direct clinical examination results showed that the newborn filly had congenital torticollis.

Blood was collected from the jugular vein of the neonate using a plain tube (BD Vacutainer™, BD Vacutainer Systems, UK). Biochemical tests were conducted using a blood chemistry analyzer (Dri-CHEM 3500i, Fujifilm, Japan).

The clinical pathological results were as follows: biochemistry of the plasma showed higher levels of CK, LDH, and Na than those in a normal foal. However, Cl level was remarkably lower than that of the control.

Affected animals have a twisted neck due to involuntary contraction of the cervical muscle. In the congenital form the condition is due to an autosomal recessive, and thus both parents are carriers and should not be rebred (Hayes, 2002).

In this study, the CK value was significantly high at 1291 U/L in the neonate. The reference interval of CK was 65–380 U/L (manufacturer's reference interval). The CK level is included in the biochemical profiles, because it has high specificity for the damaged muscle. The major tissue sources of serum CK are skeletal, cardiac, and smooth muscle fibers (Stockham, 1995), and the brain (MacLeay, 2010). CK released from cells may be a result of necrosis or reversible cell damage (Stockham, 1995). When skeletal muscle is damaged or destroyed, CK leaks out of the cells and produces an elevated blood CK level (Sirois, 2007). Genetic conditions are perhaps one of important problems affecting equine health today (Cruz, 2011). The plasma half-life of CK released following muscle injury is approximately 9 hours (Volfinger *et al.*, 1994). Thus, following muscle damage, the plasma enzyme activity increases quickly, peaking at 4 to 6 hours but will return normal within 48 to 72 hours provided there is no further damage (Lindsay, 1989).

In this study, the LDH value was remarkably high at 750 U/L in the neonate. The reference interval of LDH was  $487 \pm 100$  U/L (Schott *et al.*, 1993). LDH is widely distributed in all tissues (muscle, liver, and intestine) and an increase in the circulating concentration is therefore of little specific diagnostic value, unless interpreted simultaneously with other liver and muscle enzyme results (Taylor *et al.*, 2010). In dogs, cats, and horses, CK, AST, and LDH levels have been used to evaluate muscle disorders (Cowell, 2004). In this study, however, the values of CK and LDH increased, but the value of AST remained unchanged.

Hypernatremia occurs primarily when there is a decrease in the water intake or increased loss of hypotonic fluids (Schott *et al.*, 1993). This has been observed in horses within 30 min of strenuous exercise (Cohen *et al.*, 1993). Hypernatremia is observed much less frequently than hyponatremia. A clinical diagnosis of hypernatremia may be made when the serum  $\text{Na}^+$  concentration exceeds 146 mEq/L (Johnson, 1995). In this study, the Na value was 250 mEq/L. The reference interval of Na is  $139.7 \pm 6.0$  mEq/l (Madigan, 1987).



Fig. 1. Torticollis in 1-day old Thoroughbred filly.

Table 1. Results of biochemistry in the neonate

	Reference interval	The patient
CREA(mg/dl)	2.3±0.6	2.2
TP(g/dl)	5.1-7.6**	3.8
BUN(mg/dl)	18.9±4.3	21
TBIL(mg/dl)	4.3±2.2	2.5
TCHO(mg/dl)	111-432**	279
ALB(g/dl)	2.5-2.8*	2.4
Na(mEq/l)	139.7±6.0	250
Cl(mEq/l)	102.5±3.0	5
AST(U/l)	154±55	89
ALT(U/l)	<47**	22
GLU(mg/dl)	136.0±40	147
GGT(U/l)	29.6±15	14
LDH(U/l)	487±100	750
Ca(mg/dl)	11.7±1.1	12.4
ALP(U/l)	2,282±1,100	3,000
IP(mg/dl)	5.0±0.85	5.8
CK(U/l)	65-380**	1,291
Mg(mg/dl)	2.2±0.35	1.8

Madigan 1987, \*Knottenbelt *et al.* 2004, \*\*Manufacturer's reference value

The Cl<sup>-</sup> ion is quantitatively the most significant an ion in the extracellular compartment (Carlson, 1989). The Cl level significantly decreased and was found to be 5 mEq/L in the neonate. The reference interval of Cl was 102.5 ± 3.0 mEq/L (Madigan, 1987). Hypochloremia is associated with hyponatremia, increased bicarbonate concentrations, or an increased anion gap. Accordingly, the cause of abnormal chloride concentrations usually can be found by pursuing the cause of the abnormal concentrations of Na, bicarbonate, or unmeasured anions (Schott *et al.*, 1993).

In conclusion, the neck muscle cells influenced the clinical pathological results in the neonatal foal. A disorder of muscle or bone, such as the cervical vertebrae, is suspected to be causes of congenital torticollis.

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## 요약

말의학에서 선천성 사경에 관한 임상병리학적 보고는 없었다. 본 연구의 목적은 선천성 사경을 동반한 신생망아지의 혈액화학을 평가하기 위함이다. Thoroughbred 망아지(암)가 341일의 정상 임신기간을 거쳐서 태어났다. 평가 결과는 다음과 같다. 혈액화학검사에서 CK (creatine kinase), LDH (lactate dehydrogenase) 그리고 Na은 상당히 증가하였으나 Cl는 심각하게 감소하였다. 결론적으로, 목근육의 세포는 망아지의 임상병리학적 수치에 영향을 미쳤다. 이는 선천성 사경의 원인이 경추와 같은 뼈의 문제뿐만 아니라 근육장애일 수 있다. 저자들이 알기로는 본 논문은 신생망아지의 사경증에 관한 최초의 임상병리 보고서이다. 이 자료는 선천성 결손을 가진 망아지의 임상병리 해석에 참고가 될 것으로 기대된다.

**주요어** : 사경, 혈액화학, creatine kinase, lactate dehydrogenase, Thoroughbred 신생망아지

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