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Equine Motor Neuron Disease in a Jeju Pony

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Abstract : Equine motor neuron disease (EMND) is a spontaneous neurologic disorder of horses, which results from the degeneration of motor neurons in the spinal cord and brain stem. An 8-year-old Jeju pony gelding presented with weight loss, muscle tremors, frequent recumbence, low head carriage, sweating, and standing with four limbs close together. The gelding has been on the same stable and limited access to pasture for several years. The gelding has been fed with dried hays and commercial concentrated feeds. Laboratory test revealed very low serum vitamin E level (0.14 μg/mL; reference range > 1.5 μg/mL), mildly elevated creatine kinase (402 IU/L; reference range 119-287 IU/L) and aspartate aminotransferase (876 IU/L; reference range 226-336 IU/L). Oral glucose absorption test showed decreased glucose absorption. Histopathologic examination using a biopsy specimen from *sacrocaudalis dorsalis medialis* muscle revealed atrophic and hypertrophic muscle fiber, centralization of nucleus, degenerating and necrotic muscle fibers. Taken together, the gelding was diagnosed as EMND. After oral vitamin E administrated for 5 weeks, the gelding showed normally improved stance, decreased periods of recumbency, improved head carriage and weight gain except consistent tremors. This is the first report that successfully treated EMND case occurred in a Jeju pony in Korea.

Key words **:** equine motor neuron disease, vitamin E deficiency, Jeju pony.

Introduction

Equine motor neuron disease (EMND) is an acquired neurodegenerative disorder related with prolonged vitamin E deficiency in the adult horse (2,7). The risk factor of EMND is that lacking access to pasture and fed forages and concentrates with low concentrations of vitamin E for prolonged periods (2). Vitamin E deficiency incurs systemic oxidative damage associated with the developing of EMND (5).

EMND was firstly reported in the U.S. in 1990 since then have been reported in Canada, U.K., Switzerland, Belgium, Brazil, Netherland and Japan (9). EMND affects various breeds including quarter horses, appaloosas, standardbreds, and thoroughbreds (1). EMND cases have been decreased in U.S. recently since the preventive management was committed for horses (7).

However, considering that a large number of riding horses in Korea have been likely in restricted stables with limited access to pasture for a prolonged time, there would be horses susceptible to EMND in Korea. In this case, we describe a case of EMND in a Jeju pony. To the authors' knowledge, this is the first case of EMND in Korea.

Case

An eight-year-old Jeju pony gelding was presented to Busan Korea Racing Authority (KRA) equine hospital with weight loss, muscle wasting, continuous muscle trembling, frequent recumbence, sweating, and standing with four limbs close together (Fig 1). Upon admittance, the gelding showed muscle wasting in the triceps, quadriceps, and gluteal muscles was obvious despite a ravenous appetite to dried hays and commercial concentrated feeds. In recent a few months, approximately 60 to 70 kg of body weight loss and generalized weakness were noticed. Consistent tremors in the atrophic triceps and gluteal muscles, shifting weight, low head carriage below wither, tail head lifting, and gathering of feet under the body were also observed. Body condition score was estimated as 2

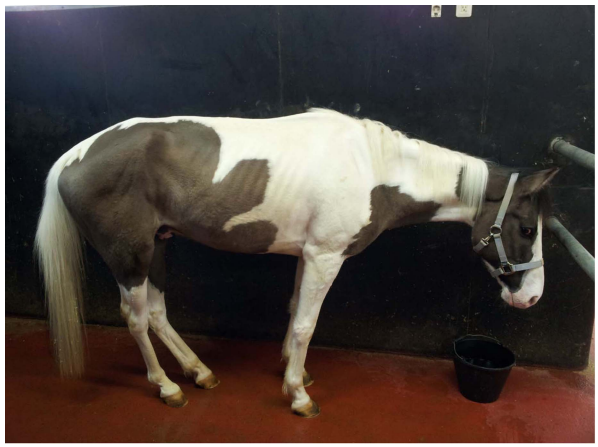

Fig 1. The pony stand with the legs gathered beneath the body ("Horse on a ball"). Head carriage is low and tailhead is lifted.

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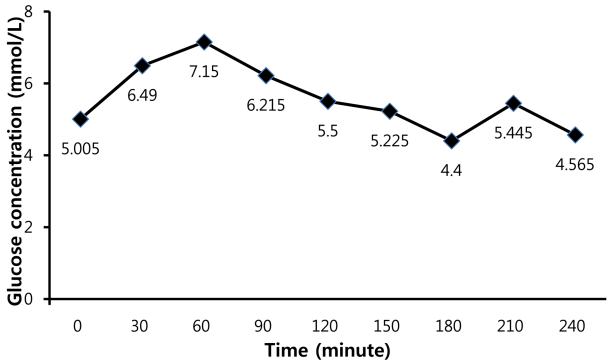

Fig 2. Result of oral glucose absorption test. The peak is 43% above the resting level.

out of 9 by two clinicians. The vital signs (heart rate, respiratory rate and body temperature) were within the normal range.

Serum biochemistry revealed elevated creatine kinase (402 IU/L; reference range 119-287 IU/L), aspartate aminotransferase (876 IU/L; reference range 226-336 IU/L), ALP (366, reference range 86-285 IU/L), AST (876, reference range 138-409 IU/L), and LDH (802, reference range 162-142 IU/L) respectively. Additional test for serum vitamin E showed decreased concentration (0.14 μg/mL; reference range > 1.5 μg/mL). Complete blood cell count results were within the normal ranges.

Oral glucose absorption test (OGAT) was performed (10). Briefly, 1 g/kg of 20% D-glucose solution was passed to the fasted gelding as a bolus through stomach tube. Blood samples were from 0 to 240 minute in a timely manner for glucose estimation. By OGAT, the peak was 43% greater than the resting glucose level. The glucose concentrations curve indicated partial malabsorption (Fig 2).

Biopsy of the *sacrocaudalis dorsalis medialis* (SCDM) muscle was performed (11). Briefly, the gelding was sedated by detomidine (2.2 mg/kg, IV) and infiltrated local anesthesia (2% lidocaine, SC) and into the SCDM muscle. A skin incision was made and undermined, and the muscle was resected. The obtained sample was placed on a tongue depressor and reserved in 10% formalin solution. The histopathologic examination revealed atrophic and hypertrophic muscle fiber, centralization of nucleus, degenerating and necrotic muscle fibers (Figs 3 and 4).

Based on the clinical findings, serum chemistry, and histopathology, this case was diagnosed as EMND. The gelding was treated with daily oral vitamin E 5000 IU for 5 weeks. After the treatment, the gelding showed normally improved stance, decreased frequent of recumbence, regained weight up to 340 kg, elevated head carriage, and reduced tail head lifting, except mild consistent muscle tremors. Body condition score was restored to 4 out of 9.

Discussion

EMND is a spontaneous and progressive neurodegenerative

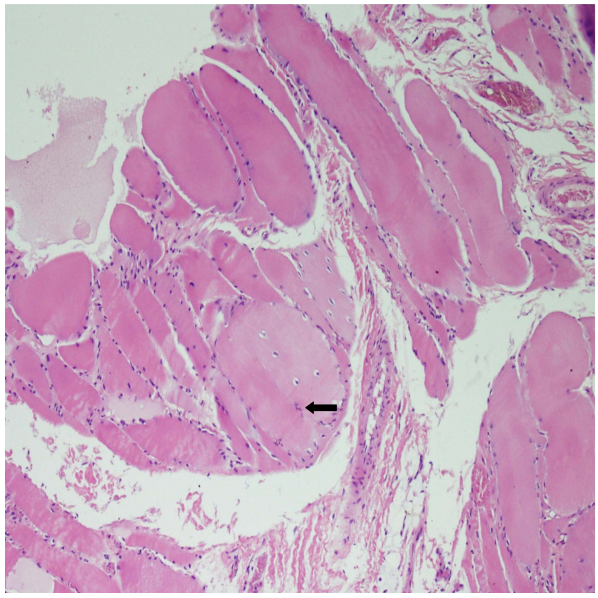

Fig 3. Longitudinal section of *sacrocaudalis dorsalis medialis* muscle showing atrophic and hypertrophic muscle fiber with centralization of the nuclei (arrow). Hematoxylin and eosin stain, × 100.

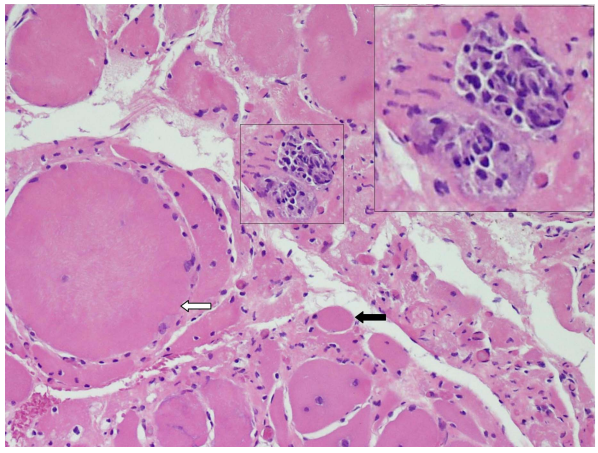

Fig 4. Horizontal section of *sacrocaudalis dorsalis medialis* muscle showing degenerativeor necrotic muscle fibers. Hypertrophic muscle fiber (white arrow) and atrophic muscle fiber (black arrow) are presented. Endomysial mononuclear cells infiltration is observed. Right upper: Mononuclear cell infiltration was noticed. Hematoxylin and eosin stain, × 100.

disease closely linked to prolonged deficiency of vitamin E affecting the lower motor neuron (5,6). This disease is similar to human motor neuron disease known as amylotrophic lateral sclerosis (ALS) or Lou Gehrig disease (7). The difference of both diseases is that EMND affects the lower motor neurons resulting in neuromuscular weakness and muscle atropy (7). However, ALS affects both the upper motor neurons and the lower motor neurons resulting in progressive muscular weakness, atrophy, muscle spacity and dead (3). Characteristic clinical signs of EMND include weight loss,

muscle wasting, trembling, excessive recumbency, shifting weight, elevated tail head, low head, and abnormal fundus in preponderant order (2). Most clinical signs in EMND are consequences of dysfunction of somatic motor neurons (2). Muscle tremors occur when over 30% of motor axons destroyed (7,9). Funduscopic change is a result of lipopigment deposition in the vasculature of retina by light generated oxidative damage (7,12).

The owner observed weight loss mainly followed by muscle tremors in this case. Weight loss preceded muscle tremors in several EMND cases (2). The absence of pasture was the most significant environmental risk factor of EMND (2). Green forages are main source of vitamin E for horses (2). Most EMND affected horses had no or minimal access to pasture for more than 18 months (2,9,12) Tissue stores abundant vitamin E in most horses. Over 18 months of vitamin E deficiency occurred EMND in an experiment (2). Therefore, EMND likely occur in 2 years or older horses which have been with prolonged vitamin E deficiency.

The 8-year-old Jeju pony gelding had limited access to pasture for over 2 years according to the owner in this case. Long term vitamin E deficiency leads to oxidative stress and damaging motor neurons and myopathy (7). Motor neurons supplying the *type I* fibers have a greater oxidative activity than *type II* fibers and thus preferentially affected by oxidative injury (7,12). Muscles with abundant of *type I* fibers are severely atrophied (2). Approximately 70% of EMND affected horses presented elevated tail (2). *Type I* fiber is predominant in SCDM muscle (12). Elevated tail in EMND horses is explained by severe denervation atrophy and fibrotic contraction of SCDM which is abundant of *type I* fibers (2). SCDM muscle biopsy is a relatively easy method as well as practical and accurate (sensitivity > 90%) diagnosis for EMND (2,12). Skeletal muscle changes with EMND include nonspecific myopathic changes such as excessive fiber size variation, internal nuclei, and cytoarchitectural alterations (7). Those histopathologic findings were revealed in our case.

Elevated serum enzyme activities of CK and AST, normal CBC findings, and very low vitamin E value were reported in most EMND (2,7). These findings support that prolonged vitamin E deficiency leads to chronic oxidative damage and result in dysfunction of motor neurons and myopathy in EMND cases.

OGAT curve has been frequently found to be abnormal in EMND cases (2). Abnormal OGAT results in several EMND cases are not fully understood (2). It was hypothesized that increased glucose metabolism occurred abnormal OGAT results (5,8).

This case was diagnosed as EMND, based on the clinical signs, laboratory tests, and histopathologic findings. Although the clinical signs have been improved after oral vitamin E treatment, consistent muscle tremors were reduced but still presented. It is thought be by permanent loss of some motor neurons (2). Treatment with vitamin E supplement for 4 to 6 weeks (5000 to 7000 IU/horse/day) may be worthy in EMND cases (7). Natural vitamin E in horses has greater bioavailability than synthetic one (4). Thus it might be beneficial for treatment with natural vitamin E in EMND cases. Approximately 40% of EMND cases continued to deteriorate, and necessitate euthanasia regardless of treatment; Nearly 40% of horses following vitamin E treatment as above restored to normal body condition, and nearly 20% lasted marked muscle atrophy (1,2,7).

Considering that a large number of riding horses in South Korea have been likely in restricted stables with limited access to pasture for a prolonged time, should urge equine veterinarians in South Korea to include EMND in the differential diagnosis of muscle wasting and tremors. Hence, it is advised to turn horses out to pasture or supply horses with vitamin E to prevent EMND in South Korea.

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제주 조랑말에서의 말운동신경세포질환 일례

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요 약 : 말운동신경세포질환은 척수와 뇌간에 존재하는 운동신경의 변성에 의해 자발적으로 발병하는 말의 신경성 질환이다. 8년령의 제주 조랑말 거세마 1두가 체중감소, 근육진전, 잦은 횡와, 낮은 두위, 발한, 사지를 모아 서있는 증상으로 내원하였다. 환마는 장기간 동일한 마방 내에서 초지 방목 없이 건초와 농후사료로 사육되었다. 혈청 생화학검사 결과 혈청 비타민 E(0.14 μg/mL; 정상 > 1.5 μg/mL)의 감소, CK(402 IU/L; 정상 119-287 IU/L) 및 AST(876 IU/L; 정상 226-336 IU/L)의 증가가 관찰되었다. 포도당 경구흡수시험에서는 부분적 흡수장애를 나타내었다. 미근부 근육생검 후 조직검사 결과 근섬유 위축과 비대, 핵 중심화, 변성 및 괴사된 근섬유가 관찰되었다. 상기의 내용을 종합하여 환마는 말운동신경세포질환으로 진단되었다. 경구 비타민 E 투여 5주 후, 환마는 정상적으로 개선된 기립자세와 두위, 횡와 빈도의 감소 및 체중증가를 보였다. 본 증례는 한국의 제주 조랑말에서 발생한 말운동신경세포질환으로서 비타민 E 치료에 성공한 최초의 보고이다.

주요어 : 말운동신경세포질환, 비타민 E 결핍, 제주 조랑말