Kafkas Univ Vet Fak Derg
16 (5): 887-892, 2010
DOI:10.9775/kvfd.2010.2206

REVIEW

Developmental Orthopaedic Diseases in Foals

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Makale Kodu (Article Code): KVFD-2010-2206

Summary

Developmental Orthopaedic Diseases (DOD) is seen frequently in horses which completed their maturity. Osteochondrosis, physitis, angular limb deformities, flexural deformities, juvenil arthritis, cervical vertebral anomalies, cuboidal bone abnormalities are problems investigated under Developmental Orthopaedic Diseases title. This diseases can develop single or some together in fast growing, heavy animals (especially Arabian and English Thoroughbreds). Multifactorial causes of this diseases etiopathogenesis can be listed as genetic predisposition, trauma, nutrition, vitamins/minerals and endocrine disorders. But the exact causes of these diseases are not known. In this review detailed information are given about the diseases mentioned above.

Keywords: *Developmental orthopaedic disease, Etiology, Foal, Radiography*

Taylarda Gelişimsel Ortopedik Hastalıklar

Özet

Gelişimini tamamlamamış atlarda, gelişimsel ortopedik hastalıklara (Developmental Orthopaedic Diseases-DOD) oldukça sık rastlanmaktadır. Osteokondrozis, fizitis, anguler ekstremite deformiteleri, fleksural deformiteler, jüvenile artritisler, servikal vertebra anomalileri, küboidal kemik anormaliteleri gelişimsel ortopedik hastalıklar başlığı altında incelenen sorunlardır. Bu hastalıklar, hızlı büyüyen, ağır hayvanlarda (özellikle Safkan Arap ve İngiliz atlarında) tek başına veya birkaçı birlikte olacak şekilde gelişebilir. Hastalıkların etiyopatogenezi multifaktöriyeldir; travma, beslenme, vitaminler/mineraller, genetik yatkınlık, endokrinoloji nedenleri arasında sayılabilir. Fakat bu hastalıkların tam olarak nedenleri hala bilinmemektedir. Bu derlemede sözü edilen hastalıklarla ilgili detaylı bilgi verilmiştir.

Anahtar sözcükler: *Gelişimsel ortopedik hastalık, Etiyoloji, Tay, Radyografi*

INTRODUCTION

Developmental Orthopaedic Disease (DOD) was first defined in 1986 and is a general term used to describe all orthopaedic problems in foals during adolescence [1,2]. This term consists of general developmental disorders in horses and it is eventually nonspecific [1].

Developmental orthopaedic diseases are listed as follows;

1. Osteochondritis dissecans [2,3]
2. Subchondral cystic lesions [2]
3. Angular limb deformities [4]
4. Physitis (Physeal dysplasia) [5]
5. Flexural deformities [6,7]
6. Cuboidal bone abnormalities [2,8]
7. Juvenile osteoarthritis [9]
8. Cervical vertebral malformations (Wobbler's Syndrome) [10,11]

Developmental Orthopaedic Disease is the term used to define different developmental disorders in skeletal system of equine species, although the etiology of the diseases is not completely known, it is believed to be multifactorial [2,12]. These consist of over or imbalanced

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feeding [13], genetics, growth rate, hormonal disorders, and biomechanical traumas [14]. DOD rarely cause lameness in foals younger than 2-4 months. Nevertheless it is a common skeletal disease complex seen in weanling and yearling horses and usually thought to limit itself [7].

A perfect musculoskeletal system is very important for horses [15]. Skeletal growth and developmental disorders in horses may be consequent to excess or shortage of components which essentially need to be in the diet.

Overfeeding of Foals

Nutrition may play a role in the pathogenesis of developmental orthopaedic disease. Mineral deficits, excesses or imbalances may be the result of excessive energy or carbohydrate intake [14,16]. Young horses which are fed high energy diets and exercised have a low osteochondrosis incidence compared to horses which are fed the same diet but with no exercise. This finding implies that exercise can alter diet effects on induction of osteochondrosis. On the contrary, young horses which are fed low energy diets and exercised can have a more severe osteochondrosis compared to horses which are fed high energy diets and exercised or which are fed low energy diets and not exercised. Exercise may cause detrimental effects in presence of lesions [14].

Mineral Imbalance

Maintenance of mineral balance is another important aspect of DOD pathogenesis. As a result of excessive phosphorus in the diet of foals, even though clinical findings of associated secondary hyperparathyroidism will be absent (even if the animal is fed enough Ca), osteochondrosis lesions will persistently occur [14,17]. Excessive Ca in the diet is a reason for hypercalcitonin and a frontier for osteochondrosis formation, there is no evidence to prove this theory in foals, however, its presence has been reported in dead lambs whose mother was fed excessive Ca [14].

Copper deficiency is considered a reason for DOD in young horses. Approximate values (10 mg/kg) determined by NRC (National Research Council) in presence of recommended levels of energy and phosphorus, cannot be associated to the increase of osteochondrosis incidence in foals. Despite this fact, a 20-50 ppm increase in copper levels is reported to decrease DOD incidence (esp. physitis). Horses are resistant against chronic copper toxicities and they can handle until doses ≤ 800 mg/kg, therefore it is doubted that 25-50 mg/kg copper will have detrimental effects. Although the mare is fed high copper diet during pregnancy, very small amounts are excreted to milk. There is no evidence present on copper metabolism and molybdenum effecting each other. It is ambiguous that excess molybdenum in the diet will cause copper deficiency and end up with DOD and is still a hypothesis[14].

Trauma and Biomechanical Forces

According to the results of a study 224 race horses physical and radiological examinations findings lesions due to trauma causing lameness mostly affected the carpal region [18]. However the exact role of trauma and biomechanical forces on pathogenesis of osteochondrosis is not clearly understood. The usual belief is that biomechanical forces are responsible for conversion of an osteochondrosis lesion to split osteochondrosis dissecans (OCD), nonetheless very little data is available to support this argument. There is no mechanical model in induction of osteochondrosis. That's why it is a challenge to distinguish biomechanical roles in OCD pathogenesis [3,14].

Genetics

Rapid growth is believed to be an important genetic factor in DOD development. Osteochondrosis incidence is higher in warm-blooded and Standard breeds compared to other breeds. However, familial susceptibility is not enough to estimate the osteochondrosis formation [14].

Endocrinology

Cartilage cell proliferation and growth are enhanced by growth hormone, insulin and insulin growth factor. Cartilage maturation is controlled by thyroid hormone where bone mineralization is controlled by vitamin D, parathyroid hormone and calcitonin hormone. Among these hormones only thyroid hormone has a direct effect on DOD [3,19].

Rapid Growth and Body Size

Previous studies show that horses diagnosed with developmental orthopaedic disease have not always grown faster than their clinically normal equals. However, there are numerous cases on horse populations which are heavier with more rapid weight gain. Variability degree in growth may also be another factor causing DOD. Horses with rapid growth or sudden weight gain may be prone to DOD [16,20]. In other species, especially in pigs and dogs, when studies are evaluated, rapid growth rate and wide body size are believed to cause susceptibility to OCD [2]. But the relation between rapid growth rate and wide body size in horses on effecting DOD incidence is still controversial among researchers. If the rapid growth has been a significant factor, the vast majority of lesions would have to be expected in this interval. However this is not what's clinically observed.

Thus, there is no determinant to prove the effects of growth speed and body size in pathogenesis [8].

In a study of 102 race horses (67 English, 35 Arabic), encountering rate of extremity lesions causing lameness is high in front extremity, between 1-4 years age group, and in English racing horses and regarding this race, lesions accompanying early racing age (2 years) are dependant on nourishment and rider faults, improper terrain, inappropriate foot and hoof treatment and shoeing [21].

OSTEOCHONDRITIS DISSECANS-OCD

The term Osteochondritis Dissecans is referred to by König [22] in 1987 in order to describe complete or incomplete substance loss in joints of young individuals. This consists of three reasons; very severe trauma, less severe trauma and necrosis, or minimal trauma enough to enhance the underlying lesion. Although the name and content have caused confusions ever since they were first referred, they are still valid today [22]. Osteochondrosis, osteochondritis and osteochondritis dissecans terms are used as substitutes despite the fact that they are not fully distinguished yet, osteochondrosis refers to the disease, osteochondritis to inflammatory response to disease and OCD to express the condition where the flap is recognized [23,24]. Two researchers Olsson and Reilan [25], had explained in late 1970's with a monograph they wrote the occurrence of osteochondrosis in pig, cattle, horse, turkey and chicken, the clinical and pathological findings in detail. Osteochondrosis is a developmental orthopaedic disease characterized with cartilage flaps, osteochondral fragments or defects or flaws in endochondral ossification which cause subchondral bone cysts. It happens as a result of isolated disruption in endochondral ossification process [24], may be observed in epiphyseal or metaphyseal growth plates. The first lesion most likely occurs prenatally or postnatal when the growth rate is highest. Although numerous studies have been performed, the occurrence is not fully explained [26]. The mechanism is not completely known, however, it is noticed that cartilage cells are not calcified and bone formation is stopped [27]. This condition results with a focal cartilage or plate protruding into the normal subchondral bone. The remaining cartilage may die, may become erode or cracked, besides, broken cartilage or bone pieces may appear in joint cavity.

Clinical symptoms of OCD are very variable [2]. OCD lesions are summarized in three categories: patients (1) with clinical and radiographic findings, (2) clinically determined but with no concomitant radiographic findings, (3) determined radiographically without clinical findings [28]. Foals with OCD findings spend most of their time lying or they have difficulties in standing up with the other horses in the paddock. Effected joints reveal swelling, stiffness or increased temperature, and sometimes lameness. In purebreds, stifle joint is the most common site for OCD, which is followed by tarsal, shoulder, fetlock joints and cervical vertebrae [29]. However, tarsal joint is the most common site for standard breeds. Involvement of multiple joints and usually two forelimbs or hind limbs is possible in OCD [2]. The veterinary surgeon must be careful defining lesions in the horses [30]. Diagnosing OCD in addition to the clinical and radiological examinations arthroscopy can be performed for definitive diagnosis and treatment [31]. Its reported that arthroscopy is a versatile and effective diagnostic method could be used form many joint examinations in veterinary medicine [32-34]. Arthroscopic surgery is a treatment option used often in order to regain athletic activity and prevent degenerative joint disease, but conservative treatment is reported to be successful in certain conditions [28]. Ultrasonographic examination may be beneficial in defining articular fractures and lesions of the subchondral bone which are subtle for radiography [35].

Osteochondrosis treatment depends on type of lesion, site and size, clinic effects, the object of breeding the horse and age factors [8].

PHYSITIS (EPIPHYSITIS)

Physitis occurs in three different forms in horses: infectious physitis, type V and type VI Salter-Harris growth plate injuries [5]. It is the inflammation of physis or metaphyseal growth plate. Despite the usual belief that is an aspect of OCD, some researchers think bio-mechanical traumas (over-exercise, excess body weight or weak conformation causes stress on immature bone) may cause the disease [5]. It mostly effects distal ends of radius, tibia and metacarpal bones and usually occurs in foals 4-10 months of age. The clinical symptoms of physitis are wide or nodular joints and especially increased temperature and pain. Effected knees reveal a bowl look, while fetlock joints are in shape of hour-glass. In radiographs, widening, glowing or irregularities and callus formation of the growth plate are observed [2,8]. Epiphysitis generally heals spontaneously as the foal grows old. If an early diagnosis is made, it is advisable to regulate the diet and restriction of exercise [8]. In case the foal is in pain, non-steroid anti-inflammatory drugs can be administered [37].

ANGULAR LIMB DEFORMITIES

Angular limb deformity is a result of deviation of the

extremity from normal vertical plane to axial direction [4,27]. Distal part of the extremity may deviate to lateral (valgus deformity) or to medial (varus deformity). Carpus valgus is the most common deformity [8,27] and it can effect fetlock joints [38] as well as tarsal joint [2]. Congenital angular limb deformities are observed with birth. These are generally believed to be the result of malpositions in the uterus, joint laxity and insufficient ossification of cuboidal bones of carpus or tarsus [2,4,8]. The latter two are usually seen in premature foals. Acquired angular limb deformities are apparent in the first weeks or months in lives of foals. These may be results of OCD formation in physitis and may cause asymmetrical growth in long bones or may be secondary to trauma or uneven diffusion of weight bearing on joints [2]. Vast majority of angular limb deformity cases are treated conservatively, however surgical approach may be considered in aggressive cases [38].

FLEXURAL DEFORMITIES

Flexural deformities or contracted tendons prohibit the leg to full extension or outstretching. The extremity seems folded or straight.

Congenital flexural deformities are observed at birth. Flexural deformities may be the result of toxins, infections which effect developing foal embryo, malposition in the uterus or genetic factors [6,7]. Fetlock and carpal joints are most commonly effected [28] and it is usually bilateral [2]. Tarsal area may be effected sporadically [6]. Flexural deformity of distal phalanx joint is characterized with high heels and clubfoot.

Acquired flexural deformities are usually secondary to physitis with pain, OCD or other injuries, they occur due to the difference between bone development and tendon development [2]. Treatment for congenital flexural deformity varies according to anatomical localization and severity. Acquired flexural deformity can be treated conservatively and is based on analgesic usage, limited exercise and regulating nutritional problems [7].

In a study made on flexor tendons of the horses it has been reported that possible tendon injuries could be defined ultrasonographically in the early period [38]. A variety of angular and flexural limb deformities can improve by podiatry and shoeing techniques, which can be performed with the cooperation of a veterinary surgeon and a farrier provided that a few basic rules are followed and it is not too late, so horses could have a chance of a future athletic career [40].

SUBCHONDRAL CYSTIC LESIONS

This condition is characterized with radiolucent cyst-like structures in subchondral bones. Lameness is the general clinic symptom which becomes evident either when the horse starts practicing or from a trauma to the lesion or after stress. Stifle joint is the most effected one, but lesion may occur in the shoulder, fetlock joints, carpus or tarsus. Subchondral cystic lesions may occur secondary to OCD. Cracks and fissures in the articular cartilage let the synovial fluid be repressed to the cavity during normal joint movement. These lesions may be the result of biomechanical traumas on weight-bearing surfaces of joints [2]. The treatment of choice is surgical evacuation where access to the cyst is feasible. Conservative therapy may be successful in some cases, but many will suffer from long term degenerative joint disease [3].

CERVICAL VERTEBRAL MALFORMATION (CVM OR WOBBLER'S SYNDROME)

This disease is characterized by narrowing in the cervical vertebral canal with resultant spinal cord compression10. The onset of disease in horses will vary between 6 months and 3 years of age [11]. Clinical symptoms of CVM are summarized as progressive ataxia and loss of coordination in hind limbs and tripping. Clinical symptoms are more severe when the horse is walked in small circles. The reason for CVM is still controversial. Various researchers believe in a subsequent development to OCD in cervical vertebrae [13]. Other anticipated reasons are; malposition in uterus causing elongated compression on the vertebrae, degenerative joint disease secondary to chronic joint instability and other biomechanical diseases. This may be the result of mineral deficiency or imbalance in foals [41]. Accurate diagnosis in a horse with CVM presenting with neurologic symptoms will require one finding showing narrowing of the vertebral canal and another finding for static or dynamic compression in the spinal cord, as well as myelography. If surgical treatment is decided, effected areas should be adequately determined. Ventral cervical vertebral fusion and partial dorsal laminectomy are the two defined surgical procedures [2].

CUBOIDAL BONE MALFORMATIONS

Cuboidal bones are the small bones which form carpus and tarsus joints. Cuboidal bone malformations are resultant to delayed endochondral ossification in these bones. This condition may be consequent to prematurity, hypothyroidism (abnormally low thyroid

hormone levels) or changes in normal ossification speed. This disease is usually seen during birth or right after birth with immature bone fracture after weight-bearing stress. In a few weeks premature foals, cuboidal bones short of complete development are present. Prognosis for survival and future athletic performances are very low in these cases. Collapse in the cuboidal bones in the carpus usually ends up with valgus, where collapse of tarsal bones will cause sickle appearance or lameness [2].

JUVENILE ARTHRITIS

This is a term used to define degenerative joint disease in horses with complete development. This condition basically occurs in proximal interphalangeal, intertarsal and tarsometatarsal joints, as well as other joints in the extremity. Recent findings show that juvenile arthritis is a complexion of DOD. It occurs in horses of the same population, horses with juvenile arthritis generally represent other symptoms of DOD. Clinical appearance is lameness and/or joint effusion and usually effect occurrence in multiple extremities. Diagnosis is made according to physical examination, regional and/or intra-articular anesthesia and radiographic findings. Unfortunately when degenerative cycle is determined, treatment is more palliative than curative. Similar to degenerative joint disease in adults, treatment is based on chondroprotection and control of synovitis. Additionally, in order to avoid development of further problems in juvenile animals, deficiencies or excessiveness of nutrition must be regulated, however the benefits on effected joints is not known [3].

CONCLUSION

As it has already been reported in several studies on developmental orthopaedic diseases, the reason for these diseases to have different incidences may be the result of different environmental conditions, genetics, nutrition or various criteria used in diagnosis. Clearly developmental orthopaedic diseases are significant problems in major horse breeding facilities worldwide.

It is important to provide at least 12 h of free exercise for foals up to 1 year and to avoid forced excessive exercise. Since there are numerous different and quality diets are available, it is hard to control the diet very often and foals tend to respond to different diets individually. The best suggestion is, following the analysis of feedstuff in the laboratory, consultation with a horse nutrition specialist. Similar attention must be paid to mares in lactation period and especially addition of excessive mineral/vitamin complexes to diets must be avoided at all times.

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