EQUINE OSTEOCHONDROSIS AND OSTEOCHONDRITIS DISSECANS

These common developmental orthopedic conditions can limit your horse’s athletic potential.

OCD? Or is it simply horses with fluid-filled navigate. Do all young waters through which to dissecans (OCD) provides joints of foals and young osteochondrosis (OC)?

Although in real life, the terms OCD and OC often become intermeshed and used interchangeably, ergo incorrectly. One recently published review article helped clarify by explaining. “OC represents the initial disease process, whereas OCD reflects secondary changes resulting in cartilage flap or osteochondral fragment formation.” In other words, osteochondrosis occurs after cartilage fails to properly turn into bone. Those OC lesions either (1) resolve on their own in young animals, or (2) the condition worsens to the point that fragments of cartilage can form, becoming OCD lesions.

HOW OC AND OCD DEVELOP

Bone forms from cartilage located at growth plates (the epiphyses) near the ends of long bones. This process is called “endochondral ossification.” Additionally, cartilage canals that serve as conduits for blood vessels must develop to nourish growing bone. The failure of cartilage cells to produce normal, healthy bone (or failure of endochondral ossification) results in abnormal joint surfaces. In healthy joints, articular cartilage lines the ends of bones inside joints, providing fluid, frictionless movement between bones during locomotion and cushioning the weight transfer. When endochondral ossification failure occurs, the articular cartilage layer can become irregular and sections of cartilage can detach from the underlying bone, causing joint surface defects. Pain, swelling, and inflammation ensue, commonly resulting in lameness.

WHO’S AT RISK?

While any horse can develop OC/OCD, some breeds are overrepresented. Standardbreds top the list with an estimated incidence of 10.5-35%, followed by Thoroughbreds and Warmbloods (20% each). Affected foals appear to develop lesions consistent with OC by about 7 months of age, but many aren’t diagnosed until later in life, usually when training commences.

Will your foal’s bone develop appropriately, or is he at risk for OC and potentially OCD? Unfortunately, despite more than 150 studies being conducted in the past 20-plus years, a firm answer has yet to be prof-

Current data suggest many factors contribute to OC and OCD, such as:
- Genetics;
- Nutrition/growth rate of the foal;
- Trauma or exercise;
- Endocrine/hormonal factors;
- Failure of vascularization of the bone during endochondral ossification;
- Environmental conditions; and
- Basic biomechanics (i.e., how the horse is “built”).

Here’s a summary of current theories about OC and OCD development:

1. Failure of cartilage canals. Lack of blood supply to the developing bone can lead to tissue death. Ultimately, this delays bone production. Several experimental studies support this theory, making it popular with experts.

2. Shearing of bone-forming tissues. Joints commonly affected with OC and OCD include the tarsus (hock) and femur, which are associated with high shearing forces and high impact. If the regions of developing bone experience trauma, then the damaged tissues can’t function appropriately.

3. Altered molecular signaling. A number of highly controlled signaling molecules orchestrate bone development from cartilage. Studies report that alterations in factors supporting healthy bone development can disrupt the pathways involved in endochondral ossification, leading to OC.

4. Genetics. While OC/OCD appears to have a genetic component, studies suggest the trait is polygenic (involving multiple genes) with a complex inheritance. Offspring from affected stallions tend to have a higher occurrence of disease, and feral horses and ponies are affected uncommonly.

5. Nutrition. Researchers have implicated diet, including caloric intake and mineral concentration, in OC development for years, although the jury is still out on its role. Originally, copper deficiency was proposed to cause OC; however, more recently, adequate copper has been shown to have a beneficial effect on lesion repair and no direct relationship to disease development. Scientists also have differing opinions on whether fast growth rate or high-calorie feeds lead to OC. Some studies show OCD increases with fast growth rate regardless of diet, whereas others have shown OCD to increase in foals fed high-energy diets.

PROGRESSION OR REGRESSION OF OC?

Some OC lesions can regress, being replaced with normal, healthy bone. In other cases, the damage progresses and OCD lesions form, characterized by loose or separated flaps or fragments of cartilage and bone at the ends of long bones that can break free from the underlying tissue. Because cartilage has the capacity for repair in young foals, lesions can regenerate spontaneously until about 8 months of age. By one year, however, lesions are unlikely to change, and the diagnosis becomes OCD rather than simply OC.

CLINICAL SIGNS AND DIAGNOSIS

OCD signs include joint swelling, lameness, and poor performance in young horses, especially those in training. Commonly affected joints are:
- Metatarsophalangeal and metacarpophalangeal ( fetlocks);
- Tarsocruial ( hock); and
- Femoropatelar ( stifle).

Lesions can also occur in the carpus (knee), elbow, shoulders, and neck. Many lesions also are bilateral (on both sides). Within each affected joint, OCD lesions have predilection sites. For example, femur OCD le-
EQUINE OC AND OCD

Lesions typically occur on the lateral trochlea (the large knoblike bony protuberance in the knee), as well as the medial trochlea, the sulcus intertrochlearis between the two trochlea, and the patella (kneecap).

Radiology typically suffices in locating OC and OCD lesions. In some cases, a vet won’t identify specific lesions until the horse is already diagnosed and undergoing surgery—usually arthroscopy (using a small camera and surgical instruments inserted into the joint through small incisions)—for previously identified OCD lesions.

Veterinarians can also use MRI and computed tomography to diagnose lesions. Recently, researchers explored ultrasonography as an alternative diagnostic.

After X raying and ultrasounding the lateral trochlear ridge of the stifle (LTRS) in 46 Thoroughbred foals between 27 and 166 days old, researchers found that “...ultrasonography provided a better overall subjective assessment of the osteochondrosis lesion topography (length, depth, and width) compared with radiography.”

TREATMENT

Foals younger than 6–8 months can be monitored and rested to determine if OCD lesions develop and require surgical management. Removing loose cartilage flaps in affected joint(s), typically via arthroscopy, is the current treatment. If the cartilage flap is large enough or still partially attached to surrounding tissues, surgeons can use an absorbable pin to fix the flap in place, essentially restoring the joint’s frictionless surface.

Outcomes depend on which joints are affected and response to management. Lesions of the LTRS—one of the most common OCD locations—generally have a good to excellent prognosis if the lesions aren’t too large (>4 mm long). As cited in one study, 65% of horses that underwent surgical debridement for lesions of the lateral trochlear ridge of the stifle performed at their intended level. Racehorses with stifle lesions that were surgically debrided had fewer starts and placed finishes and earned less money than unaffected individuals. Further, and not surprisingly, researchers associated larger OCD lesions with poorer prognoses postoperatively. Lesions of the distal intermediate ridge of the tibia in the hock, another common OCD location, also have an excellent prognosis if treated surgically early in the disease process. Overall, prognosis depends on the lesion’s location and size.

TAKE-HOME MESSAGE

With so many potential factors influencing whether your horse’s joints will develop normally, prevention might seem an impossible feat. Until more details regarding factors involved in OC and OCD development surface, early diagnosis, monitoring, and treatment remain best practices for healthy horse joints.

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Recommended Resources