

FCE- Fibrocartilaginous Emboli

I can promise you that you will have more questions than answers after reading this material. I hope to help you understand what the disease is and separate the few facts that we know from the theories, guesses and outright rumors. Like all afflictions in our breed, we would all like it to be Black and White, a simple dominant gene with an easy solution. This is not that type of story. There are few facts and LOTS of theories.

FCE—Fibro cartilaginous Emboli was first diagnosed in man in 1961. Dogs in 1973....certainly it occurred before that time, but our diagnostic tools just were not good enough to help us know what it was. Humans, pigs, cattle, one cat and one horse have been diagnosed with this ailment, but dogs are the most common species affected.

In most dog breeds, it occurs in adults most commonly from 3-7 years of age. In Irish Wolfhounds specifically, it occurs more frequently as puppies; generally between 6 and 16 weeks of age. (Some other large breeds have had it reported as young as 16 weeks) Since it was associated in timeframe with the vaccination age, people often made the leap that vaccines were causing this paralysis. In my own opinion and that of any author of journal article I have read, the timeframe of vaccination is completely coincidental. I know of at least 5 youngsters with presumed FCE who had not been vaccinated at all.

WHAT IS IT?

Fibrocartilaginous Emboli or Spinal Cord Infarction ; also called Puppy Paralysis and Drag leg Syndrome This is an emboli made up of fibrous/cartilage type of tissue. Let's get the terms defined before we start throwing them all around.

Emboli is just plural for embolus. An embolism is:

OBSTRUCTION OF A VESSEL BY A SOLID OR GAS MATTER WHICH HAS BEEN TRANSPORTED THRU THE BLOODSTREAM.

And the embolus can be a blood clot or air or, in this case, a specific type of fibro-cartilaginous tissue that gets lodged in the bloodstream and blocks further blood flow. When that happens to a blood vessel, whatever cells it was serving no longer get the benefit of nutrient/oxygen exchange resulting in cell death. This is called:

Ischemia or the lack of blood flow to a part or organ.

The damage that occurs is often referred to as **infarction** or the tissue death that occurs when blood supply is lacking. You have probably heard the term myocardial infarction when this process happens to the heart muscle.

Therefore, some people call FCE a spinal cord infarction. Doing a journal search, this term will give you more connects than FCE. In FCE, the source of the embolus is from the disc in between the vertebrae in the spinal column. Let's take a look at where this occurs.

Looking at a diagram of the intervertebral disc, find the center of the disc. This central area is the location of the Nucleus Pulposus...a gel like material and this is what is found to be the source of the



emboli in FCE.

Now the \$64,000 question is HOW? How does a bit of that disc material get into the blood vessel to block the blood flow? There are several theories, but nothing proven.

1. Trauma to the vessel bed causes communication of the disc material
2. Persistence of embryonic arteries of the disc and herniation of the disc material into those arteries. (This might be a likely theory in the case of young puppies)
3. New arteries forming in the disc due to chronic inflammation (This theory might be more likely in the older animal.)
4. Herniation of the disc material into venous supply, then lodging in an artery.

None of these theories have been proven out consistently in autopsies on people or dogs.

FCE is a diagnosis of EXCLUSION. What that means is that the only way to positively identify this disease is by frozen microscopic sections of the nerve to find the emboli after euthanasia. So what if we would like to keep the dog alive??? !!! We diagnose FCE by elimination of other probable disease processes.

This makes it a very difficult disease for data collection and research. The diagnosis is actually “presumed” FCE in any live dog.

WHAT DOES FCE LOOK LIKE?

It mimics many other conditions—there is no outward symptom that will be unique to FCE.

A typical presentation of FCE follows:

Puppy, active and normal...perhaps with a history of trauma. Slipping, falling, dropped.

Sitting position, unable to rise.

When placed in a stand, cannot walk forward...will often collapse into a sit

Deep pain response intact (somewhat controversial statement, but that is my personal experience with Wolfhound puppies)

PAIN is not usually consistent with an FCE diagnosis. I have received several calls in the past year about pups that have a paralysis with an onset of severe pain...screaming in intense pain for 24 hrs or more with slowly improving condition and pain relief. My best guess is trauma...not FCE

Many of the human patients who succumbed to FCE reported a transient sudden pain on onset. Many owners of dogs who were positively identified with FCE reported yelps of pain and then no pain thereafter, just paralysis.

DIAGNOSIS

In my opinion, you need to get a neurological exam immediately. ALL SPINAL INJURIES, WHETHER FCE OR TRAUMA NEED IMMEDIATE CARE!!!! THE FASTER YOU GET VETERINARY TREATMENT, THE BETTER THE PROGNOSIS. REMEMBER THE GOLDEN HOUR. After 6 hours, steroid therapy will probably not make a difference. After 24 hours, there is probably no advantage of steroid therapy.

If you remember nothing else about this discussion: All dogs that present with a paralysis should be immediately transported to a veterinarian who is willing to do a neurological exam and treat the dog for spinal shock as appropriate. (Do not wait until morning!) The standard steroid therapy is Methylprednisilone 30 mg/kg IV followed by a tapering schedule over the next several days. The earlier, the better!!!!

Diagnostic procedures will help us with a diagnosis and prognosis. If you find evidence of trauma (swelling, redness, etc.) the treatment will essentially be the same as an FCE treatment. There are conditions of the spine and paralysis that can involve infection where steroids alone could make matters worse. If infection (discospondylitis, meningitis or neurospora/ toxoplasmosis in very young pups) is suspected, the neurologists that I have spoken with would concurrently treat with antibiotics. The other common rule out with an older animal is cancer of the spine. Sudden onset is the picture you get with FCE, not a gradual onset over days.

Lower Motor Neuron signs versus Upper Motor Neuron signs. This separation has to do with the pathways that these nerves serve. In LMN disruption, you will see flaccid muscle tone where with UMN, there can be a heightened tone to the muscle. LMN pathways do not recover as well as UMN. Paralysis from FCE is usually complete in 24 hours. If you see worsening signs after that timeframe, especially ascending paralysis...it is usually indicative of a softening of the spinal cord and is a very grave sign. If there is little or no improvement over 14 days after onset, the prognosis for recovery becomes very guarded. There is a better prognosis for lateral signs (one sided).

Myelograms in Wolfhounds: The myelogram is a radiography study where dye is injected into the spinal column to look for abnormalities, swellings, disc protrusions, etc. I have seen hundreds of these be successful with dogs, but I have lost a young bitch in a myelogram procedure and know several other breeders who have had dogs die during this procedure. Certainly that is not the case with all Wolfhounds. At this point, I would recommend an MRI as a less invasive procedure that may give better information for us to rule out other disease processes in FCE cases. Many authors look to improvements in MRI technology that would provide easier and better diagnostic results.

TREATMENT

Steroid administration in the first 24 hours (earlier the better!) Antibiotics as indicated. Treat any trauma.

NOTE: One Wolfhound owner treated two pups several years ago with intravenous DMSO (dimethylsulfoxide) in the first two days after onset. DMSO is a popular topical anti-inflammatory here in the USA, particularly in large animals. It has the unique characteristic of being a carrier mechanism for other substances.(i.e. other drugs could be added to DMSO and carried thru the skin into the tissues below.) These pups had a strong recovery. I have not been able to locate any supporting data on this treatment option. .

Supportive care:

Nutrition, rest, elimination, avoiding pressure sores. High level of care may be needed for first 10 days. Then, recovery to ambulatory state should be there if recovery chance is good/. Look at options of carts/slings (recovering animals need exercise...not limited motions)

Complementary recovery techniques

Swimming/Water therapy: Provides for use of muscles, limits atrophy, doesn't depend on gravity. Warm water therapy pools may have advantages.

Acupuncture: "Opening" of channels of energy along meridians. Eliminates stagnation and increase flow of "qi" in Chinese medicine terms

Chiropractic: Assists in correction of compensatory limitations. Most animals with FCE or other paralysis develop stiffness, soreness or muscle adaptations to compensate for the affected limb. (opposite front leg restriction example)

Massage: Tellington/Jones, standard, range of motion, fascia release techniques. All help to avoid compensatory damage

Bowen work This soft tissue treatment centers on releasing trigger points of muscles, tendons and ligaments to help the body stay in a healthy alignment.

Nutrition: High quality proteins/vegies pulped for repair and growth. Vitamin E/C/B complex. Trace minerals may be needed...alfalfa and kelp are good sources.

Herbs: Arnica, rhus tox, homeopathics for inflammation and injury are indicated early in the onset of FCE. Asafoetida is a Chinese herb that may assist in longer-term recovery.

RECOVERY After diagnosis, most dogs reach a stable condition within 24-36 hours. Dogs with no favorable progress in their condition within 14 days have a poor prognosis. Signs of ascending paralysis are very serious. There is a great article chronicling the recovery of a 16 week old Wolfhound in the Spring 1996 issue of the The Irish Wolfhound magazine from England. I would venture to say that the majority of FCE puppies recover to a level of activity and independence that they can live happy, painfree lives with a moderate amount of care. Few puppies or adults ever recover completely as the nerve damage leaves a lasting impairment. Unfortunately, multiple emboli or those located higher up in the spine have a grave prognosis.

SO, IS IT GENETIC????

Ninety nine percent of all the vet neurologists out there will tell you NO WAY. But a 1999 article from University of Utrecht reviews 8 IW pups with presumed FCE. Certainly our breed has the tendency to FCE at a young age. But is it a tendency like bone cancer afflicting giant breeds or is it a genetic fault that could be bred away from?

My personal data has revealed more than 9 lines involved worldwide with few common ancestors in each of those lines. In one example, one popular stud dog had been linebred several times and produced NO FCE pups. In 3 subsequent outcrosses, he produced at least one FCE pup in each litter. His sire had been bred both in outcross and linebred breedings with no FCE pups, but did produce one FCE pup in his last litter which was an outcross. Obviously, if there is an inherited component, the genetic expression is complex.

So we are left with the question, what do we do as breeders? As with most things, it will be a very personal decision. We have no proof that breeding an affected bitch increases the chance of FCE. (Yes, affected bitches have been bred without producing FCE.) But, without data collection on significant numbers of dogs, we will never be able to recognize a possible pattern.

Anne Janis, who leads the data collection for seizure disorders, rhinitis (primary ciliary dyskinesia) and liver shunt has offered to collect information on dogs with presumed FCE.

Difficulties in presumed diagnosis makes the data less reliable than other diseases. Differing levels of expertise and diagnostics may result in false positives. My hope in collecting anecdotal information in large enough numbers is that we will see a trend toward random occurrence or distinct patterns of inheritance. I would encourage all breeders who have had a presumed or confirmed FCE case to contact Anne with pedigree information. iwstudy@earthlink.net Anne will gladly explain her data collection process

The WITCH-HUNT PROBLEM—When improved diagnosis and testing allows us better identification of the dogs (and perhaps bloodlines) involved, will we use the information responsibly? Or will we treat those who have shared the information with us as somehow irresponsible and unworthy? It's my opinion that its human nature to gossip and bad news seems to travel faster than good news. I ask each of you to consider positively supporting ALL breeders who benefit our breed by sharing information about the health of our dogs.

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