

# FIBROCARILAGINOUS EMBOLIC MYELOPATHY

## (SPINAL CORD DISORDER CAUSED BY BLOCKAGE OF A BLOOD VESSEL)

### BASICS

#### OVERVIEW

- “Fibrocartilaginous” refers to fibrocartilage; “fibrocartilage” is cartilage that contains bundles of collagen fibers; “cartilage” is a tough connective tissue found in joints and other body parts; “embolic” refers to an embolus or embolism; “embolus” is a material (such as a blood clot or foreign material) that blocks a blood vessel; “myelopathy” is a disorder of the spinal cord
- Fibrocartilaginous embolic myelopathy is a condition in which a piece of fibrocartilage breaks off the intervertebral disk and travels in the blood vessel until it blocks blood flow to the spinal cord
- Sudden (acute) death of spinal cord tissue due to lack of blood flow (known as “ischemic necrosis of the spinal cord”) caused by fibrocartilaginous emboli
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C<sub>1</sub>-C<sub>7</sub>; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T<sub>1</sub>-T<sub>13</sub>; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L<sub>1</sub>-L<sub>7</sub>; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae

#### SIGNALMENT/DESCRIPTION of ANIMAL

##### Species

- Dogs and cats

##### Breed Predispositions

- Giant- and large-breed dogs—highest number of cases
- Miniature schnauzers and Shetland sheepdogs—reported to have fibrocartilaginous embolic myelopathy at higher than anticipated rates; high levels of lipoprotein (compounds containing lipid [a group of compounds that contain fats or oils] and protein) in the blood (condition known as “hyperlipoproteinemia”) and resultant increased protein in the blood leading to sludging of the blood [known as “hyperviscosity”] are common in these breeds; hyperlipoproteinemia and hyperviscosity may contribute to sudden lack of blood supply that leads to death of tissues (known as “infarction”) of the spinal cord, without the presence of a piece of fibrocartilage material within the blood vessel (fibrocartilage embolus)

##### Mean Age and Range

- Most patients are 3 to 5 years of age
- Range, 16 weeks to 10 years

##### Predominant Sex

- Slight male predominance

#### SIGNS/OBSERVED CHANGES in the ANIMAL

- Mild trauma or vigorous exercise at the onset of signs is common
- Sudden (acute) onset
- Affected dog typically cries in pain; pain subsides in minutes to hours (at most)
- Signs of weakness or partial paralysis (known as “paresis”) or paralysis develop over a matter of seconds, minutes, or hours
- Condition stabilizes within 12 to 24 hours
- Nervous system deficits—usually involve one side primarily; unaffected side usually mildly affected or normal
- Pain—at onset of signs and then generally absent; usually subsides by the time the patient is being examined; may be felt for a few hours in severely affected patients
- Spinal cord injury—any level of the spinal cord can be affected, depending on the location of the piece of fibrocartilage material within the blood vessel (fibrocartilage embolus)
- Mild wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- Upper or lower motor neuron disease and related nervous system deficits; disease involving the nerve cells of the brain, brain stem and/or spinal cord that control the muscles is known as “upper motor neuron disease;” disease of the nerves that connect the spinal cord and muscles is known as “lower motor neuron disease”
- If signs progress beyond 24 hours, other diseases should be considered

#### CAUSES

- Unknown

#### RISK FACTORS

- Vigorous exercise may trigger the incident
- High levels of lipoprotein (compounds containing lipid [a group of compounds that contain fats or oils] and protein) in the blood (hyperlipoproteinemia)

## TREATMENT

### HEALTH CARE

- Inpatient—for immediate medical treatment and diagnostic procedures
- Keep recumbent patients on a padded surface; turn frequently to prevent pressure sores
- Assist and encourage patients to walk as soon as possible

### ACTIVITY

- Restrict, until diagnosis is made

### DIET

- Normal

## MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Methylprednisolone—may be beneficial, if given within the first 8 hours after onset of signs

## FOLLOW-UP CARE

### PATIENT MONITORING

- Sequential nervous system evaluations—during the first 12 to 24 hours after initial physical examination
- Nervous system status—2, 3, and 4 weeks after onset of clinical signs
- Lack of control of urination (known as “urinary incontinence”)—urinalysis and bacterial culture and sensitivity to detect urinary tract infection

### PREVENTIONS AND AVOIDANCE

- Recurrence highly unlikely
- No known method of prevention

### POSSIBLE COMPLICATIONS

- Inability to control bowel movements (known as “fecal incontinence”) and to control urine (urinary incontinence)
- Urinary tract infection
- Pressure sores and skin lesions that develop due to contact with urine, when the hair and skin remain damp (known as “urine scald”)
- Destruction of spinal cord tissue
- Euthanasia

### EXPECTED COURSE AND PROGNOSIS

- Pain perception and upper motor neuron (involving the nerve cells of the spinal cord that control the muscles) signs—prognosis for marked improvement generally good
- Loss of pain perception—prognosis poor
- Lack of reflexes (known as “areflexia”) of legs or sphincters—almost no chance of recovery
- Reduced purposeful movements and reflexes—functional recovery common; some degree of permanent deficit likely
- Progression of clinical signs from upper motor neuron (involving the nerve cells of the spinal cord that control the muscles) to lower motor neuron (involving the nerves that connect the spinal cord and muscles) and an enlarging area of sensory loss indicate development of a condition in which the motor neurons (nerve cells that control muscles) are destroyed, leading to progressive spinal cord disease that is not reversible (condition known as “myelomalacia”) and a hopeless prognosis; consider euthanasia
- Nervous system status—usually little change in the first 14 days after onset; most improvement occurs between days 21 and 42; replacement of myelin (a white material that covers certain nerve fibers; process known as “remyelination”) is complete in most patients within 6 to 12 weeks after onset; if no improvement after 21 to 30 days, recovery is highly unlikely

## KEY POINTS

- Recovery from weakness or partial paralysis (paresis) or paralysis is slow and gradual, when it occurs
- Most patients need considerable supportive care at home during recovery