THE GREAT PRETENDERS: THINGS THAT LOOK LIKE SEIZURES BUT AREN’T

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Overview
Seizures are one of the most common neurologic problems encountered in practice. Some patients have seizure-like episodes that can be difficult to distinguish from a true seizure. This lecture will review several neurologic conditions that may be misinterpreted as a seizure.

There are several characteristics of a seizure that will help differentiate a seizure from another neurologic disorder. When obtaining the history, the owner should be questioned about the following seizure characteristics to help determine if the episode is consistent with a seizure.

Characteristics of a Seizure
1. A loss or alteration of consciousness:
   Probably the most important factor when deciding if an animal had a seizure or a seizure-like episode is to determine if the animal was conscious during the episode. There is usually a loss or alteration of consciousness with a seizure. The owner should be asked if their pet was conscious or not during the episode. This may be difficult for an owner to determine. In these instances, ask the owner if their pet was responsive to voice command when standing behind them.

2. Motor and/or autonomic involvement:
   Most seizures have motor movements that may be generalized (tonic, clonic, tonic-clonic) or focal (twitching of one limb or one side of the face). Autonomic signs are a common feature of a seizure and include urinating, defecating, salivating, dilated pupils, and vomiting.

3. Automatisms (repetitive movements) or paroxysms (psychic disturbances):
   Examples of automatisms and paroxysms include licking, chewing, “fly biting,” aggression, fear anxiety, and seeking attention.

4. Three phases (preictal, ictal, and post-ictal phases):
   There are three distinct phases to a seizure - the preictal, ictal, and post-ictal.

Having the owner video-tape an episode is also helpful in determining if it was a seizure or not.
**Episodic Muscle Hypertonicity Syndrome**
This condition may be interpreted as a seizure because it is characterized by episodes of increased muscle tone (hypertonicity) that can vary in frequency and severity. Animals with this condition are not painful and maintain consciousness during an episode, which can help differentiate this condition from a seizure. Knowing the breeds that affected is also helpful.

**Cause**: The cause of this condition is not known but it is believed to be due to a deficiency of serotonin in the CNS because of the clinical manifestations. Clinical signs improve when an affected animal is given a drug that potentiates the effects of CNS serotonin (eg. Acepromazine), whereas drugs that decrease the effects of CNS serotonin will worsen or induce clinical signs (eg. Amphetamine). Scottie Cramp is believed to be an autosomal recessive hereditary disorder that is widely disseminated within the Scottish Terrier breed.

**Breeds**: Scottish Terrier is the most common breed affected and it has been termed “Scotty Cramp” in this breed. This condition has also been reported in Cavalier King Charles Spaniels, Dalmatians, and Norwich Terriers. The majority of affected breeds begin to show clinical signs within the first year of life, although some breeds may start showing signs later. Cavalier King Charles Spaniels may exhibit clinical signs at 4 years of age.

**Clinical Signs**: Episodes are most often triggered by exercise, stress, or excitation. The pelvic limbs are typically affected first. Affected animals are normal between episodes. Episodes of muscle cramping and hypertonicity can range from primarily pelvic limb involvement, to severe hyperflexion of all limbs, to collapse. Affected dogs can also exhibit exaggerated flexion action of the pelvic limbs (string-halt or goose-stepping gait), bunny hopping of the pelvic limbs, and an arched back. Some dogs may collapse due to the severe hypertonicity. The episodes typically last less than 10 minutes.

**Diagnosis**: There is no specific diagnostic test for this condition. A presumptive diagnosis is based on the signalment and characteristic clinical signs.

**Treatment**: There is no cure. Oral benzodiazepines may help control the severity of the episodes (diazepam or clonazepam - 0.5 mg/kg PO TID).

**Prognosis**: The disease is not life-threatening. Triggers should be avoided.

**Idiopathic Head Tremor Syndrome**
A movement disorder characterized by head and neck tremors has been recognized in several breeds and is often misinterpreted as a seizure. Affected animals are conscious and responsive during the episode which can help differentiate these episodes from a seizure.

**Cause**: The etiology of this syndrome is unknown.

**Breeds**: Most often seen in young English bulldogs, Doberman pinschers, Boxers. Can occur in any breed.

**Clinical Signs**: A coarse head tremor, “head bob”, characterized by a rapid, coarse tremor
of the head and neck in either an up-and-down motion (a “yes” movement) or a side-to-
side motion (a “no” movement). Episodes are random, last seconds to hours, and do not
appear to be progressive. There does not appear to be a trigger although some owners
will report that stress or excitement may cause an episode. Affected animals are
responsive to voice command and are able to walk around.

**Diagnosis**: Based on signalment, history, and description of the episode.

**Treatment**: There is no treatment or cure. The most effective way to stop the episode is
to get the patient to fix their attention on something (feeding them or fetching ball).

**Prognosis**: This is not a life-threatening problem and it appears to become less severe
with time.

**Exercise Induced Collapse in Labrador Retrievers (EIC)**
A syndrome of exercise intolerance and collapse has been observed in very active,
excitable Labrador retrievers. Affected dogs often have an episode in a field or hunt trial.

**Cause**: This is an autosomal recessive disease caused by a mutation in the dynamin 1
gene. Dynamin 1 is involved with neurotransmission and synaptic vesicle endocytosis.

**Breeds**: Young Labrador retrievers that are very excitable. Similar conditions have been
seen in Border Collies and Australian Shepherds.

**Clinical Signs**: Signs typically develop between 7 months and 2 years of age. After 5-15
minutes of heavy, intense exercise, dogs become weak, ataxic, and then collapse. Dogs
will also hyperventilate and their body temperature is often severely elevated. Most dogs
recover completely within 15-30 minutes. Dogs are not in pain during the collapse or
after they recover. A few affected dogs have died during exercise or while resting
immediately after an episode of EIC, so an affected dog’s exercise should always be
stopped at the first hint of incoordination or wobbliness.

**Diagnosis**: Identification of the DNM1 mutation done through University of Minnesota
(blood, semen, dew claw, or cheek swab sample).

**Treatment**: There is no specific treatment; avoid strenuous exercise and excitement.

**Prognosis**: Good as long as strenuous activity is avoided.

**Feline Hyperesthesia Syndrome**
This is an episodic, poorly understood pain disorder in cats that is often interpreted as a
focal seizure. There is no loss of consciousness which can help differentiate it from a
seizure.

**Cause**: Unknown. Possible myopathy

**Breeds**: Any age, breed, or sex cat. Abyssinians, Himalayans, Siamese, Burmese may be
predisposed.

**Clinical Signs**: Affected animals will lick, bite, chew, and attack their flank and tail. Cats
may have dilated pupils, vocalize, and run around the house. Other signs include rippling
of skin over the dorsum of their back, exaggerated tail movement, and erratic behaviors.
Diagnosis: Is a diagnosis of exclusion. A presumptive diagnosis is based on clinical signs and ruling out other disorders (musculoskeletal, dermatologic, neurologic, behavioral).

Treatment: Treatments are empirical and include anticonvulsants, behavior modifying medications, pain medications (gabapentin, tramadol), and supplements (coenzyme Q10, carnitine, omega-3 fatty acid). Drug trials and titrations are needed to determine the most effective drug or combination of drugs. The treatment goal is to reduce the frequency and severity of the clinical signs.

Prognosis: Guarded for controlling or eliminating episodes. Episodes tend to progress.

Acquired Myasthenia Gravis

Cause: Autoimmune disease in which antibodies are formed against the nicotinic acetylcholine (ACh) receptor resulting in decreased numbers of receptors on the postsynaptic sarcolemmal surface.

Breeds: Occurs in dogs and cats. More common in dogs than cats. High risk breeds include Akitas, terriers, German Shorthaired Pointers, and Chihuahuas, Abysinian, Somali breeds.

Clinical Signs: Bimodal age of onset (3 years and 10 years). Common clinical signs include generalized weakness, decreased palpebral reflex, decreased menace response, decreased gag reflex, laryngeal weakness, and megaesophagus. Weakness will often worsen with activity (wax and wane).

Diagnosis: Definitive diagnosis is made by demonstrating circulating antibodies against nicotinic ACh receptor.

Treatment: Anticholinesterase drugs are the mainstay of treatment. These drugs bind ACh-esterase, thereby prolonging the availability of ACh binding to the ACh receptor. Pyridostigmine bromide (Mestinon): 0.5-3.0 mg/kg PO BID-TID. Immunosuppressive drugs have also been used.

Prognosis: Good if aspiration pneumonia or pharyngeal weakness is not present.

Generalized Idiopathic Tremor Syndrome

This tremor syndrome is often referred to as “Little White Shaker Syndrome” because it was originally described in Maltese and White Highland Terriers. This syndrome has also been called “Corticosteroid Responsive Tremor Syndrome.”

Cause: This disease is believed to be an autoimmune disease of the central nervous system (primarily of the cerebellum).

Breeds: Many breeds of different coat colors affected; affected dogs usually less than 5 years old and weigh less than 15kg.

Clinical Signs: Predominant clinical sign is a fine, whole-body tremor; other signs have been reported (decreased menace responses, head tilt, nystagmus, seizures, ataxia, paresis).

Diagnosis: Cerebrospinal fluid analysis reveals a lymphocytic pleocytosis.

Treatment: Affected dogs respond well to immunosuppressive doses of corticosteroids.
(Prednisone: 1-2 mg/kg PO BID). Once clinical signs have resolved, the dose is gradually discontinued over a 1-3 month period. Some dogs need to take a maintenance dose to control signs. Some dogs may benefit from oral diazepam (0.2 mg/kg PO TID). Prognosis: Excellent.

**Narcolepsy**

Narcolepsy is a disorder of the brain that is marked by sudden recurring attacks of sleep and cataplexy. Cataplexy is a sudden loss of muscle tone in response to stimulation (dropped jaw to complete collapse) with consciousness remaining intact.

**Cause**: Exact cause is unknown but an imbalance between cholinergic and catecholaminergic neurotransmitter systems within the CNS is suspected.

**Breeds**: Doberman Pinschers, Labrador Retrievers, Miniature Poodles, Beagles, and Dachshunds.

**Clinical Signs**: Cataplexy is the predominant sign in dogs and cats; episodes are characterized by paroxysmal attacks of flaccid paralysis without loss of consciousness and are frequently induced by eating and excitement. Episodes can last up to 20 minutes with a sudden return to normality. Excessive daytime sleepiness and fragmented sleep patterns have also been reported. Dogs do not have any autonomic signs (urinary or fecal incontinence, salivation) or muscle rigidity.

**Diagnosis**: Based on the typical clinical signs and exclusion of other paroxysmal disorders. Food-elicited cataplexy test can be used to identify mild cataplexy. Pharmacologic testing can also be done (physostigmine can induce an episode, atropine can decrease clinical signs).

**Treatment**: Tricyclic antidepressants improve signs (imipramine: 0.5 - 1.0 mg/kg PO TID; desipramine -3 mg/kg PO BID).

**Prognosis**: Good.

References available upon request.