Research Article

Chorea: A Sequelae of Canine Distemper

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Abstract | Disorders of animals related to movement are highly diverse and are divided into a heterogenous group with varying physical situations, such as involuntary movements without losing consciousness. Various forms of convulsions, hypersensitivity of peripheral nerves, involuntary muscle movements involving repetitive or twisting movements, writhing movement of arms or face and chorea are involved in canine movement disorders. In this consensus statement, standard terminology for describing the various movement disorders is recommended with an emphasis on paroxysmal dyskinesia, as well as a preliminary classification and clinical approach to reporting cases. In the clinical approach to movement disorders, we recommend categorizing movements into hyperkinetic vs hypokinetic, paroxysmal vs persistent, exercise-induced vs not related to exercise, and a genetic or unknown cause that involves defined movements with the help of suggested terminologies described here. Classical canine distemper is a multisystemic disease that typically affects the gastrointestinal, lymphoid and respiratory system. In many clinically and sub-clinically affected dogs, neurological manifestations of canine distemper occur 2-3 weeks later, *albeit* they may appear even months or years later. This neurological form of the disease affects the white as well as the grey matter of the CNS. Owing to the involvement of both white and grey matter, a variety of neurological signs, including behavioral changes, focal or generalized, seizures (fits), cerebellar and vestibular signs, visual deficits, paresis, paralysis, limb weakness, tremors and myoclonus may be observed. Seizures and myoclonus (chorea) are the two most common signs of neurological involvement.

Keywords | Athetosis, Fasciculations, Ballism, Chorea, Cramp, Myoclonus, Tetanus, Tremor

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INTRODUCTION

(St. Vitus Dance, myoclonus) also known as hyperkinesia in dogs is defined as the involuntary rhythmic jerking or twitching of a single muscle or muscle groups and resembling those in affection which has been described as "chorea electrica" (Prikryl et al., 2018). Although there are several causes of chorea in dogs, this condition mostly occurs as one of the sequelae of canine distemper (Canine distemper is an important disease of dogs that can occur even in properly vaccinated animals) encephalitis and often appear during the convalescent period. The condition also occurs in humans (Lowrie and Garosi, 2017).

In veterinary medicine, chorea is also symptomatically similar to paroxysmal dyskinesia. It has been found in a number of breeds (e.g., Cavalier King, Charles spaniel, Border terrier, Cairn terrier, Scottish terrier, Dalmatian and Norwich terrier, Boxer, Bichon frise, Pugs, Chinook, in which they have been 'labelled' as breed specific beings).

However, it has not been reported that similar paroxysmal movement disorders are gradually seen in other breeds, particularly Jack Russell terriers (JRT) or Labrador retrievers in the UK. Cramps or Spasms specifically in the limb, which are found as an increase in the muscle tone of limbs, are the most common appearance in affected breeds of dogs (Whittaker et al., 2022). The hind limbs are affected to a greater extent as compared to the forelimbs. However, all four limbs may be affected. During an attack, animals can be severely injured, since the spasm overcomes any attempts at voluntary movement, however, many dogs will still attempt to walk. Some factors like excitement or exercise may trigger the effect in some cases.

Etiology

It is not clear still now that why chorea disease occurs, it depends upon several factors. The precise cause is not known but mostly suspected causes include;

- Poisoning and toxicity
- Brain tumors that cause weakness of the brain portion which controls muscle movements
- Trauma to the head
- Intestinal parasites; worms may cause a lot of damage to the brain
- Canine distemper
- Vaccines that are not administered properly

Chorea-acanthocytosis is a very rare genetic disorder. It is characterized by distorted red blood cells. Different neurological abnormalities and disturbed brain functioning may result. Abnormal arm and leg movements, pelvic thrusts and shoulder shrugs are involved in chorea. Similarly, rapid and purposeless movements of the face may also occur (Wood, 1893). Dystonia may also be involved in the dogs exhibiting this form of chorea. This is characterized by involuntary muscle contractions of the mouth and face, such as involuntary belching, teeth grinding, saliva coming out of the mouth or spitting, difficult swallowing, lip and tongue biting, and vocal tics such as grunting, slurred speech or involuntary speaking.

In addition to chorea and dystonia, this condition may cause seizures, neuropathy, loss of sensation, muscle weakness, and behavioral changes (Leo, 2022).

It mostly occurs in dogs with renal, liver and splenic disorders. It is sometimes associated with lymphoma and RBC fragmentation and with altered lipid metabolism (Weiss, 2022).

Neurophysiology and Neuroanatomy of movement control

A complex integrated neuronal circuitry is termed as basal nuclei. It is a difficult task to specify a nucleus with a certain function. Basal nuclei connect individual parts with the cortex and thalamus through reciprocal loops and feedback, which plays a major role in movement gating. Functionally segregated parallel circuits form cortical connections with basal nucleus (Eberhard et al., 2022).

There are 5 cortico-BN-thalamo-cortical loops which are (1) motor, (2) oculomotor, (3) associative, (4) limbic, and (4) orbitofrontal.

The modulatory control of the basal nucleus and associated circuits are dominated by the dopaminergic projections to the putamen and caudate. D1 dopamine receptors activation increases glutamatergic activity and activation of D2 receptors decreases glutamatergic activity (Bezan and McKay, 2022). Semiotically organized "motor circuit" is most relevant to the pathophysiology of movement disorders. Nigrostriatal dopaminergic neurons regulate the primary somatosensory cortex and motor cortex which is glutamatergic (excitatory) to the caudate and putamen and mainly activates the GABAergic medium spiny neurons (Salpietro et al., 2018). There GABAergic medium spiny neurons are directly connected to the main output structure of the basal nucleus, substantia nigra pars reticulata (SNr) and entopeduncular nucleus (EPN) (Danciu et al., 2019).

There are 2 main pathways: the direct and indirect pathway. The "direct pathway" is an inhibitory neuronal connection from EPN/SNr to putamen, monosynaptic and contains y D1 dopamine receptors. the "indirect pathway" contains D2 dopamine receptors and striatal neurons.

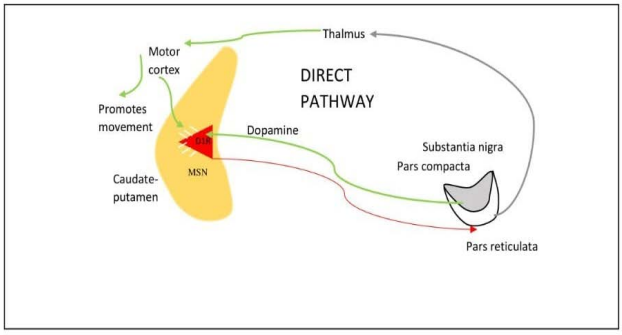
The "indirect pathway" is initiated when the caudate nucleus and putamen in medium spiny neurons send their GABAergic axons to the GP, which convey GABAergic axons to the subthalamic nucleus, which in turn directs excitatory axons (glutamate) to the EPN/SNr. This inhibits the pedunculopontine tegmental nucleus (PPTN) and the ventrolateral thalamic nucleus. The ventrolateral thalamic nucleus and the PPTN are then inhibited by the Entopeduncular nucleus/substantia nigra pars reticulata (Abdel-Saeed and Elmashad, 2020).

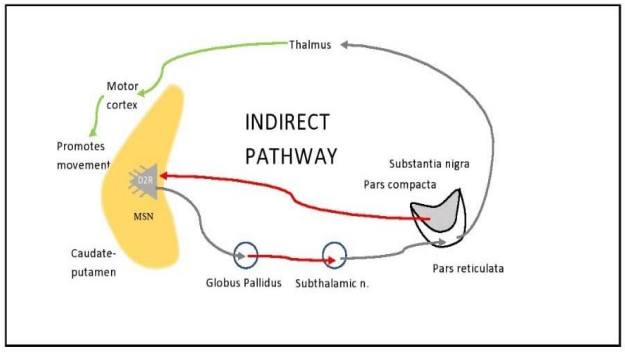
In veterinary literature:
Basal nuclei comprise of caudate nucleus, entopeduncular nucleus (EPN), putamen, amygdala, claustrum and globus pallidus (GP)

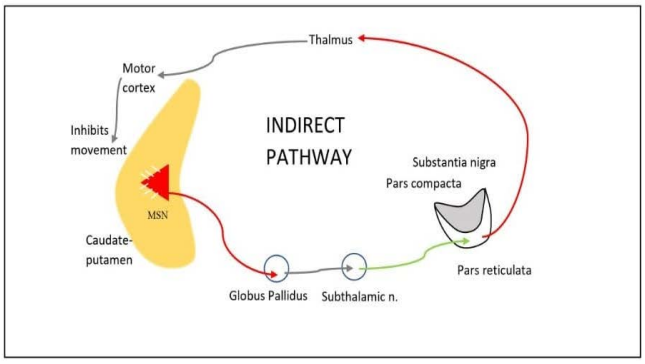
Both, the indirect and direct pathways control the output of the EPN/SNr neurons. When the direct pathway is activated neuronal firing rate is reduced in the EPN/SNr, which results in disinhibition of their projections. On the other hand, when the indirect pathway is stimulated excitation of the subthalamic nucleus is increased, which

Table 1: Differentially diagnosed diseases with chorea

Disease	Differentiation
Tremors	Systemic, involuntary but patterned oscillatory movements of a particular body part with a balanced rate in both orders of movement (i.e., sinusoidal), around a joint axis, caused by rhythmic alternating contractions of the agonist and antagonist muscles.
Tetanus	*Clostridium tetani* infections release exotoxins which cause release of glycine, as a result of which severe and sustained muscle contraction occurs and this is defined as *tetanus*. It usually involves extensor muscles and also no relaxation is observed. Different sensory changes may accompany (e.g., hypocalcemia-induced tetany).
Myotonia	Relaxation of muscles usually after voluntary contraction or percussion. It can be noticed frequently after a period of rest and recovers with sustained movement. Diminished joint flexion and hypertrophy of the affected muscles may result when walking due to myotonia. (i.e., "stiffness" of the limbs)
Neuromyotonia	Due to abnormal electrical discharges of some motor nerves, permanent muscle stiffness and the late muscle relaxation occurs. Generalized muscle stiffness and late or delayed relaxation differentiates it from myokymia. Hence, this results in collapse of the animal. Frequent muscle rippling may occur clinically. Some factors like stress or excitement may trigger it. In order to summarize, due to the supposed local spread of motor command, overflow movement "spread" of movement beyond an area of (unintended) movement to a near or adjacent area occurs.
Myoclonus	Sudden, but irregular, repeated jerk like movements which result from the involuntary contraction and relaxation of one or a group of muscles. This results in the movement of the contracted body part like limb or head movement, whereas various tremors like movements do not differ from an abrupt comeback in that myoclonic movements which usually exists independent of an abrupt stimulatory involvement and happen repeatedly (Eberhart, 1917). However, some factors like movement or stress may worsen the condition. It usually happens in sleep.
Dystonia	Production of abnormal movements or posture of particular part of the body in a longitudinal pattern only as a result of persistent or prolonged, gradual, unconscious or involuntary contraction of both type of muscles either agonist or antagonist. Consequently, curled posture of the trunk, limbs or neck appears. By the introduction of irregular posture of different parts of the body, dystonia may either create completely new movement or may stop normal movement. It is related to the varying degree or span of arrhythmic muscle contraction (Penrose, 1963).
Cramp	An abrupt, unconscious or involuntary contraction of muscles or over shortening, that is usually benign and temporary in nature which may result in severe pain (mild-to-excruciating), even complete immobilization of the muscles. Usually, the occurrence is abrupt and it may repeat after the different span of time (several seconds, minutes, or hours). It may be due to electrolyte imbalance.
Ballism	The term is defined as sudden unconscious and involuntary contractions of muscles of proximal limb (e.g., shoulders) leading to various movements of the limbs like flailing or flinging, having a large amplitude (usually unilateral). It can be differentiated from chorea or athetosis in animals (Auffret *et al.,* 2019).
Athetosis	The term is of Greek origin which means "without position or place". It is defined as slow but prolonged unconscious or involuntary contraction of the muscles as a consequence of which flexing, bending, convoluted, writhing and non-rhythmic movements of hands, fingers, toes or feet occur. All these factors result in the disturbance of a normal stable posture. Most often athetosis is confused with repetitive **choreatic** movements (i.e., choreoathetosis). Muscles of distal limb are mainly involved (less frequently the muscles of trunk, face, neck). However, individual choreatic movements can be differentiated from each other as discrete movements, whereas movements associated with athetosis usually seem to "flow" from one to another and becomes more difficult to separate from each other (Cerda-Gonzalez *et al.,* 2021).
Fasciculations	A small number of muscle fibers activate, which results in flickering or vermicular like movement under the skin due to a spot contraction of muscle fibers, the term is defined as fasciculation. A brief, sudden, involuntary contraction with primarily described in two phases, one is a unidirectional fast phase which may be either positive or negative and second is slower recovery phase. Hence, they may be termed as rhythmic myoclonus (Woma and van Vuuren, 2009).
Myokymia	A wave or vermicular like movement of the skin covering the affected muscle due to a focal or generalized continuous contraction of facial or limb myofibers (It looks like as the worms crawling the skin) (Dalling *et al.,* 1966).







results in increased inhibition from the EPN/SNr onto their projection.

Movement is promoted by dopamine as it excites medium spiny neurons by the D1 receptors in e direct pathway but is inhibited by D2 receptors in the indirect pathway. Thus, dopaminergic innervation loss leads to hypokinetic movement disorders and when indirect pathway is affected by a disease condition it leads to hyperkinetic disorders like chorea (Macalister, 1924).

In addition to these 2 pathways; the cerebellothalamic-BN pathway of the cerebellum also contributes to the pathophysiology of hyperkinetic disorders, particularly dystonia (Rosenow, 1923).

Thalamo-cortico-basal ganglia circuits are involved in movement disorders.

a) In direct pathway activation of D1R on GABAergic MSN increases inhibition of the SNr through dopaminergic innervation of the caudate/putamen. As a result, tonic inhibition is lost by the thalamus.

Excitatory feedback is increased from the thalamus to the motor cortex and caudate/putamen due to this inhibition, thus abnormal movements are promoted.

b) Indirect pathway works oppositely. GABAergic neurons are inhibited by MSN. Subthalamus is released from tonic inhibition, SNr/EPN excitation is increased and leads to thalamus inhibition which results in movement inhibition. Dopamine through D2R inhibits MSN in the indirect pathway.

c) Thus, the activity of the direct pathway is promoted by dopamine and the activity of the indirect pathway is decreased.

Abbreviations: D1R, D1 dopamine receptor; D2R, D2 dopamine receptor; EPN, entopeduncular nucleus; GP, globus pallidus; MSN, medium spiny neurons; SNc, substantia nigra pars compact; SNr, substantia nigra pars reticulata.

Clinical signs of chorea in dogs

a) Characteristic involuntary, isolated repeated twitching (jerking) of a single muscle or muscle groups of face, mastication, limbs, neck, shoulder or abdomen (Amude et al., 2010; Willard and Tvedten, 2011). Occasionally, eyelids are also affected. Some chorea affected dogs show constant bobbing up and down of the head at each involuntary muscle twitching in a helpless silly manner (Koutinas et al., 2002). Involuntary rhythmic twitching of muscles is so constant that it even persists during sleep (Amude et al., 2006).
b) Exhaustion due to constant twitching of muscles.
c) Digestive disturbances such as variable appetite, costive bowels, tumid abdomen, foul tongue and loss of body condition. Some mildly affected dogs recover spontaneously
d) Sudden collapse
e) Confusion and anxiety, as the dog feels helpless
f) Involuntary elimination as the dog cannot control its bowel and bladder
g) Varying degree of weakness, head tilt, nystagmus, paresis and ataxia with hypermetria (Cerda-Gonzalez et al., 2021).

Differential diagnosis

Different symptoms may appear in different situations at rest, also can be worsened or precipitated by movement. The condition of athetosis is more difficult to differentiate because there is a lot of difference in the anatomy and degrees of joint movement between human beings and ca-

nines (although in canines, complex movements like the movement of fingers, hands, or wrist are lesser). In sum, athetosis is hardly distinguishable from chorea and ballism and should be described simply as dyskinesia.

Pathology

A dog with chorea presented and the post-mortem examinations were such that no disease of the internal organ. No entozoan, healthy heart, no problem with muscle when examined with the naked eye or with the microscope. No lesion was found in the cerebral hemisphere or the central ganglion. On the other aspect, the spinal cord, the medulla oblongata, and the cerebellum presented extensive alteration. Evaluation of the spinal cord in a fresh state (immediately after death) revealed little change. No areas of congestion or ecchymosis were visible. The white substance appeared normal in consistency and tint (Gowers and Sankey, 1877).

Histopathology

Reveals that Nerve cells from the anterior cornea appeared somewhat swollen and unduly granular, but contained no pigment.

Extensive crippling was observed after hardening. The most apparent change was the extensive fill up of specific areas with lymphoid cells. These lymphoid cells vary in size from 1-4 inches, quite smaller and round in appearance. Most of these lymphoid cells were anucleate means there was no visible nucleus but in some cells, a large nucleus was obvious just like in plant cells covering most of the portion of the cell approximately two third. In the affected areas there was no uniformity observed in the infiltration of lymphoid cells. In the white substance, lymphoid cells were aggregated, branching and extending throughout the whole affected tract or part.

Dogs with PD are often confused with epilepsy or unusual epileptic seizures means the patient will remain conscious just like PD attack. Actual identification or correct information/ a source of the disease or neuromuscular disorder is necessary to identify the type of neuromuscular disorder. Most of the time neuromuscular disorders occur episodically. As a result of which when dogs are examined at variable times, the neurological situation appears normal.

A complete and proper history of the affected animal is necessary, because certain different conditions may appear apparently similar (Lee et al., 2021). The foremost approach to ensuring involuntary muscle movement is that the animal should maintain its normal cardinals during the span between one episode of dysfunction and the other. Usually, there is hypersalivation, urinary obstruction or bowel movements, so that must be checked. If possible, with the history, the owner should be asked to record the episodic spasm for a better assessment of the animal's condition.

Paroxysmal disorders can be differentially diagnosed in their simplicity and focal seizures. Disorders can easily be recognized with similar and repeatedly muscle movements. Otherwise, they will be mis-considered as neuromuscular disorders. Clinical differentiation is quite difficult because there are no defined standards for PD, diagnostic facilities are not available and elimination of structural diseases of the CNS is not a priority.

As a result of which to properly differentiate these disorders a brain scan, cerebrospinal fluid analysis or MRI scan of the brain is recommended. CBC, hormonal disturbances, biochemistry, urinalysis, and endocrine levels are thoroughly examined (Moore et al., 2022).

Though there are certain causes of chorea, so complete history of the case is necessary to determine the potential cause and its diagnosis. For a complete diagnosis, the following points are necessary;

- The time of symptoms beginning?
- What is the effect of certain factors on chorea severity?
- What is the effect of stress on an animal with chorea?
- What is the previous canine distemper record?
- What medications or vaccines are used previously?
- What is the feeding history of animal?
- Is there any previous injury during the fight?
- If the genetic record is available, is any previous record in the family?

Laboratory diagnosis may also indicate chorea. For instance, if there are unusual levels of copper in the body that may be an indication of Wilson's disease, which is a congenital disease and may lead to chorea. The disturbed biochemistry of blood such as thyroid and parathyroid hormones, complete blood count, and spiky red blood cells can indicate endocrine related chorea and chorea-acanthocytosis respectively. In Wilson's disease, there is an abnormality of copper levels in blood and brain that is effectively toxic for an animal and can result in chorea (Sawyers, 2022). The liver function test in animals can confirm that chorea is due to copper toxicity. The hemogram can reveal irregular erythrocytes or misshapen RBCs showing chorea-acanthocytosis.

Treatment

Most chorea affected dogs are euthanized as there is no specific and really effective treatment. A set of treatment modalities can be selected from the treatment strategies given below before a decision on euthanasia is taken:

1) Spinal tap treatment using New Castle disease Lasota virus vaccine is claimed to be effective in 50% of chorea cases. The following are the steps of this therapeutic procedure (http://kindheartsinaction.com/2009/12/17/dr-sears-describes-a-spinal-tap/):

a) The first step in the spinal tap treatment is to place an intravenous catheter and give general anesthesia (Jackson, 1872).
b) Prepare the area at foramen magnum for aseptic surgery.
c) To perform a spinal tap at the foramen magnum, there are two procedures for the proper positioning of the dog. The first procedure is to have the spine at the edge of the table and neck flexed so that the bridge of the nose is perpendicular to the spine. It is important to have the dog's nose parallel to the table. The second positioning procedure is similar but the neck is flexed as far as possible. The objective of both the positioning procedures is to open the cisterna magnum (the reservoir for CSF located at the back of the brain) as much as possible to allow an easy access to the spinal fluid. The landmarks in both positioning procedures are the same i.e., the cranial edge of C2 (axis) and the occipital protuberance (Strauss et al., 2015).
d) Depending on the size of the dog, withdraw 0.1 to 1.0 ml of CSF. This sample may be dispatched to the lab for the determination of anti-distemper antibodies.
e) Depending upon the size of the chorea affected dog, inject 0.1 to 0.5 ml of Newcastle disease virus (LaSota Strain) vaccine (1000 doses vial diluted with 6 ml of diluent) into the spinal canal. Afterwards, flush the needle with ½ to 1 ml of saline.
f) Treat the dog for the shock with fluids after giving this injection at 48 hours intervals (Greene, 2006).

2) Corticosteroids e.g., Inj. Prednisolone @ 2.2-6.6 mg/kg/day IV or IM, then tapering to 2-4 mg/kg at 48 hours intervals to counter the immunological reactions that might have caused neurological lesions.

3) Procainamide (an antiarrhythmic drug available under the brand name of Tab. Pronestyl™ Glaxosmithkline, Pakistan. Each tablet contains 250 mg procainamide) @ 125-250mg (depending on the weight of the dog) orally 2-4 times daily may reduce the severity of chorea.

4) Anticonvulsants: Phenobarbital (e.g., Tab. Debritone™ Xenon Pharma and Tab. Phenobarbitone™ Karachi Pharmaceutical Laboratories. Each tablet contains 30 mg phenobarbital) @ 2.5 mg/kg orally every 12 hours.

5) Acetazolamide: Treatment is possible, with most dogs responding to the use of a drug called acetazolamide.

6) Canine Immunoglobulin preparations such as inj. Canglob™ (marketed in Pakistan by Ghazi Brothers) is not likely to be effective in the treatment of neurological manifestations of canine distemper (Tipold et al., 1992).

7) Large doses of attenuated canine distemper vaccine: Large doses of attenuated canine distemper vaccines may be effective in the treatment of dogs suffering from nervous symptoms of canine distemper.

8) Dewormers: Chorea affected dogs should be dewormed as internal parasites are sometimes a component of the etiology of this nervous manifestation.

9) Vitamin C: IV administration of 1-2 grams of Vit. C per day for 3 consecutive days along with symptomatic treatment was reportedly effective in the treatment of 12 cases of clinical distemper, including 1 case showing nervous manifestations (Belfield, 1967; Geetha and Selvaraju, 2021).

10) Homeopathic treatments: (Moore et al., 2022) recommends the following homeopathic remedies for different manifestations of chorea in dogs.

Nux vomica*:* constipation, impaired appetite, and the other symptoms related to disorders of the stomach; trembling or convulsive jerking of the front or hind leg, or of sets of muscles; grouchy temperament, etc.

Ignatia: convulsive movements of the legs, muscles of the face, eye or eyelids that increase in severity when the animal is frightened (Raj et al., 2020).
Belladonna and Cuprum aceticum may also be of therapeutic value.

(Madrewar, 2004) recommend the following homeopathic remedies for different manifestations of chorea in dogs.

Belladonna 30 twice a day when a chorea affected dog champs with foam at the mouth, turns the head up and falls over backward.

Rhus toxicodendron 30 twice a day when the dog walks unsteadily, has frequent attacks of shivering and the convulsive twitching of the muscles in different body parts (Madrewar, 2004).

Miscellaneous treatments: Eberhart (1917) recommends the following recipe for chorea in dogs.

Strychnine	= 1 grain (64.8 mg)
Quinine	= 18 grains (1.166 grams)
Belladonna extract	= 6 grains (388.8 mg)
Gentian extract = 4 grams	

Rhubarb powder = 4 grams

Mix all the above and then divide into 48 pills. The dose for a small dog is ½ a pill b.i.d, the dose for a medium dog is 1 pill b.i.d and the dose for a large dog is 1.5 pills b.i.d. This treatment should be continued for 1-2 months.

Affected dogs should be fed carefully, properly lodged and exercised.

Galvanism (the therapeutic use of electric current) is also recommended for chorea.

Prognosis

Chorea is referred usually as a symptom or sequela of another disease. As far as chorea prognosis is concerned no statement can be confirmed. Dogs with chorea may recover completely with proper and on time treatment. However, some cases are untreatable and the dog may die. Studies support the facts that prognosis depends upon the dog, the time of disease, detection, and the patient's age. Which means lesser the duration and magnitude of neuronal damage, the greater will be the chances of recovery.

Chorea due to head injury, trauma or intestinal parasitism is treatable and the patient shows a good prognosis.

If chorea is a sequela of erroneous vaccination or canine distemper the prognosis is poor and in some cases the patient dies (Norris et al., 2006; Saks, 2005).

The chances of recovery can be enhanced with proper veterinary assistance and cure like the use of antiparasitic drugs or deworming in patients with intestinal parasitism and laxatives along fiber rich diets (oatmeal or canned pumpkin) in patients with constipation leads to a better prognosis.

On time and suitable surgical procedure in patients with certain injuries and homeopathic remedies in patients with vaccination related chorea elevates the chances of recovery.

CONCLUSIONS

Chorea is a disorder of involuntary, repeated twitching (jerking) of a single muscle or groups of muscles. Athetosis is more difficult to differentiate because there is a lot of difference in the anatomy and degrees of joint movement between canines and humans. Some factors like stress or excitement may trigger it. Dogs with PD are often confused with epilepsy or unusual epileptic seizures. Most chorea affected dogs are euthanized as there is no specific treatment.

Lasota virus vaccine is claimed to be effective in 50% of chorea cases. Some cases are untreatable and the dog may die.

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CONFLICT OF INTEREST

All of us show no conflict of interest in this paper.

NOVELTY STATEMENT

This paper is about neuromuscular disorders of the canines specifically, chorea which is almost a new thing because of a little work done on it.

AUTHORS CONTRIBUTION

All authors contributed equally to the publication process.

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