VETCT
CONSULTANTS IN TELEMEDICINE

REPORTING SERVICE: NEUROLOGY

Report number: TELE-XXXXX Report date: XXXXX

Referring Veterinarian: XXXXX

Referring Practice: XXXXX

Email address: XXXXX

Owner: XXXX Patient: XXXX

Species: Canine Breed: Crossbreed Sex: Male Entire Age: 1 year, 7 months

Associated cases: VETCT-XXXX

Clinical History:

We would like to receive a second opinion on this patient.

Scampy is a 1y 7m o M Crossed breed (JRT x Chihuahua) that was seen the first time by Dr. Richard and me on **07.08.14** with a possible history of chocolate intoxication. He was showing GI signs (V) and neuro signs as described hereby.

Physical examination was normal.

Neuro examination revels what I think is a cerebellar ataxia, with normal mentation, a wide-based stance, intentional tremors of the head, loss of balance and truncal sway, moderate hypermetric gait.

Proprioception: mild delay on front, moderate/severe on back.

Cranial nerves and menace response bil wnl.

The clinical signs seemed getting worst with the exercise and the excitement.

O refers that the dog had always be mild paraparetic since he was a puppy.

Routine CBC, biochemistry and lactate levels were without any significant abnormality.

The patient slightly improved the first 24hs but then remained stable.

This report is based on the available history and radiographic interpretation only and not on a physical examination of the patient.
It must therefore only be interpreted by a currently licensed and registered veterinary surgeon responsible for the care of this patient.

Based on this a brain MRI and CSF tap were performed.

The MRI showed mild bilaterally symmetrical meningeal contrast enhancement, that, seen the CSF's analisis, was considered normal.

>CSF's analysis was wnl. (Gross analysis, manual count 2 cells/ul, cytology on sediment and Tpwnl. We didn't run any infectious disease).

(Laboratory reports and MRI reports are enclosed herewith).

Based on these results, no treatment was given. Since theme we saw the patient twice and he seems stable vs very mild improved.

Finally the patient represented yesterday **07.12.2014** at hour hospital with the same clinical sings (both GI and neuro, with possible chocolate intoxication that had never been confirmed.).

Questions to be answered:

In your opinion which further diagnostic test would you suggest?

Do you think this could be a case of hereditary ataxia (Wessmann 2004)?

We look forward to receiving your comments and advice in due course.

Report:

This is a very interesting case. It would be interesting to know since when Scampy is showing this type of gait. Chocolate intoxication (theobromide) usually presents with restlessness, hyperactivity, hyperreflexia and seizure activity in severe cases, however spinocerebellar ataxia is not a feature of this type of intoxication.

Based on the video, I would consider hereditary ataxia of the Jack Russell Terriers as the most likely cause of his gait, however this is very unlikely to cause paraparesis and postural reaction deficits. Dorsal cervical lesions (such a subarachnoid diverticulum) could cause spinocerebellar ataxia and progressive tetraparesis but this would be also unlikely to have an acute onset. If the dog still presents with paraparesis, it could be that the dog suffers from an unrelated T3-L3 myelopathy.

Did the dog have another chocolate intoxication in December? It could also be possible that this dog could have suffered from suffers from episodes of **neuromyotonia** (increased muscle tone, muscle stiffness and persistent contraction) and **myokymia** (involuntary rippling of muscles, tremors) rather than intoxications as this has been reported in JRT with spinocerebellar ataxia. It has been reported that some owners from affected cases realised that the ataxic gait in their dog became clearly obvious after the first neuromyotonic attack.

It would be late onset of signs if the first reported clinical signs were noticed when the dog was 15 months of age. Did the owner ever reported any uncoordinated gait prior to the first episode of intoxication?

There have been two different mutations discovered with spinocerebellar ataxia in Russell Terriers

1. one reported in Jack Russell Terrier with onset from 2 to 6 months of age
2. another one reported in Parson Russell Terriers with an onset from 6 months of age to 1 year of age

I would recommend testing the dog for these mutations (http://www.aht.org.uk/cms-display/genetics_ca.html) (http://www.aht.org.uk/cms-display/genetics_loa.html). Each test cost £40.

The fact that the dog is a cross JRT, it could also be that it has a different mutation.
If the Hereditary Ataxia of the JRT is confirmed on genetic test, they usually have no progression on his spinocerebellar ataxia and can potentially have a good quality of life. About 33% of these affected dogs can develop seizure activity. Depending on the frequency and severity of the seizure activity, then antiepileptic medication might be required.

Hyperthermia can be potentially life-threatening during episodes of neuromytonia and myokymia and some dogs had died spontaneously during a collapse. I have attached a paper describing these episodes and potential treatments, however if the frequency is not too high, I would wait on starting any medication at this stage.

Please do not hesitate to contact me if you require further information,

Kind regards.

XXXXXX, Ldo Vet, MRCVS, DipECVN

If you have any queries regarding this report then please "Add a comment" on the VetCT platform or contact info@vetctspecialists.com

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