

## Evaluation of facial paralysis with electromyography (EMG) associated with hypothyroidism in a dog

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**Abstract:** A 7-year-old male American Cocker was referred to İstanbul University Faculty of Veterinary Medicine Surgery Policlinics with imbalance, flabbiness in the left cheek muscles, increased salivation, alopecia, and hyperpigmentation. Complete blood count, blood biochemistry, and thyroid profile were recorded. Hypothyroidism and hypercholesterolemia were detected. Levothyroxine was applied orally. With regular drug therapy, thyroid hormones levels returned to normal ranges. The facial paralysis also disappeared completely, and the skin problems healed. This was the first patient with facial paralysis that originated from hypothyroidism and was diagnosed with clinical findings, laboratory methods, and electromyography in Turkey.

**Key words:** Hypothyroidism, facial paralysis, electromyography, dog

### 1. Introduction

Hypothyroidism is the most common endocrine disorder of the dog (1–8) and is characterized by various cutaneous and noncutaneous clinical signs associated with a deficiency of thyroid hormone activity (1). The thyroid, under the influence of thyrotropin (thyroid-stimulating hormone-TSH), produces the hormone thyroxine (T4) and 3,5,3'-triiodothyronine (T3) (2).

The clinical signs associated with hypothyroidism are many and varied, involving multiple organ systems (1,9). The veterinary literature includes large lists of noncutaneous abnormalities associated with hypothyroidism (1). Neurologic, reproductive, ocular, hematologic, and cardiovascular abnormalities have been reported in dogs with hypothyroidism (5,10). Localized neuropathies often cause vestibular and facial nerve paralysis (5). Central nervous system signs can include seizures, disorientation, circling, and coma (1).

Neurological manifestations of hypothyroidism are being recognized with increasing frequency (11). A hypothyroid-associated neuropathy does appear to exist in mature to middle-aged dogs, usually in large breeds, and may be recognized without the usual signs of hypothyroidism (1). Peripheral neuropathy is the best documented neurologic manifestation of hypothyroidism (10). Peripheral neuropathies may also affect the cranial nerves, causing abnormalities such as head tilt, facial

nerve paralysis, strabismus, nystagmus, decreased facial sensitivity, and laryngeal paralysis. The early stages are often missed because the signs are mild (1). Once a diagnosis has been made, this disease can be easily controlled with thyroid hormone supplementation (2). Dogs improve rapidly within a few days after the start of treatment, and most dogs are neurologically normal after 1 to 2 months of treatment.

Electromyography (EMG) is the measurement of electrical activity within a muscle and is performed using electrodes (12,13). Electrodiagnostic examination is a fairly noninvasive method of assessing functional neuromuscular disorders (14). Electrical activity elicited when the electrode is inserted and the duration and amplitude of the potential at the motor unit can be used to evaluate the integrity of the motor unit. A normal muscle at rest is otherwise electrically inactive. In pathological conditions, different forms of spontaneous electric activity can be seen following spontaneous depolarization, which progress along the muscle membranes. This spontaneous activity can be registered as different types of wave-formed curves. Fibrillation potentials and positive sharp waves can be seen following denervation, and in some cases complex repetitive discharges as well (13).

### 2. Case history

A 7-year-old, 18 kg, intact male American Cocker was referred to İstanbul University Faculty of Veterinary

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Medicine Surgery Department Polyclinics with imbalance, flabbiness in left cheek muscles, increased salivation, alopecia, hyperpigmentation, and facial paralysis (Figure 1). There was no trauma or exposure to a toxic agent. The dog had received all of its vaccinations and had no previous medical problems.

The clinical signs were characterized by left ear drooping, lip commissural paralysis, lagophthalmia, and abnormal gait. The physical examination revealed no abnormalities. The neurological examination revealed menace response and palpebral reflex absence and vestibular ataxia. Concentric needle EMG of the major muscle groups of the head was performed (Figure 2). It showed moderate abnormal spontaneous electrical activity where fibrillation potentials and positive sharp waves were noted (Figure 3). Magnetic resonance imaging (MRI) of the brain and internal acoustic canal was normal (Figure 4). Laterolateral (LL) and ventrodorsal (VD) X-rays of the skull and tympanic bullae were normal. Otoscopic examination revealed that the tympanic membranes were intact and normal in appearance.

Then the patient was referred to Internal Medicine Department Polyclinics for general condition status control and the existing skin problems. Upon physical examination, no abnormalities were detected but alopecia on the trunk and hyperpigmentation were clearly seen (Figure 5). Blood was taken for a complete blood count (CBC), and the serum biochemical profile revealed hyperlipidemia,



**Figure 1.** Facial paralysis of the dog.



**Figure 2.** EMG application in the dog.

hypercholesterolemia, and hypertriglyceridemia (triglyceride: 197 mg/dL, cholesterol: 398 mg/dL).

Because of the skin problems and the lipid profile, hypothyroidism was suspected. To detect this disease, thyroid function tests were performed. Measurement of the endogenous TSH level was achieved using a canine assay. T3, T4, Free T4 (fT4), and TSH were detected with a chemiluminescent immunoassay system. The values of the parameters are given in the Table below.

The diagnosis of hypothyroidism was made. The dog was treated with levothyroxine sodium (Levotiron; Abdi İbrahim, İstanbul, Turkey) 0.02 mg/kg, per os (PO), q12h; cod liver oil (Pulse; Seven Seas, Abdi İbrahim, İstanbul, Turkey) 500 mg/day (1 capsule), PO, q24h; and B1&B6 vitamins (Benexol; Bayer, İstanbul, Turkey) 250 mg/day, PO, q24h. The dog's home-made diet was also changed to Dermatologic Management® Canine Formula, Purina Veterinary Diets. The owner was informed that the serum T4 levels should be re-assessed in 4 to 8 weeks.

Two weeks after the onset of clinical signs and initiation of treatment, the owner reported an improvement in palpebral hang down and hyperpigmentation on the trunk.

The second referral was 8 weeks after the initiation of thyroxine supplementation. At this time, T4 levels



**Figure 3.** Positive sharp wave and fibrillation potentials.



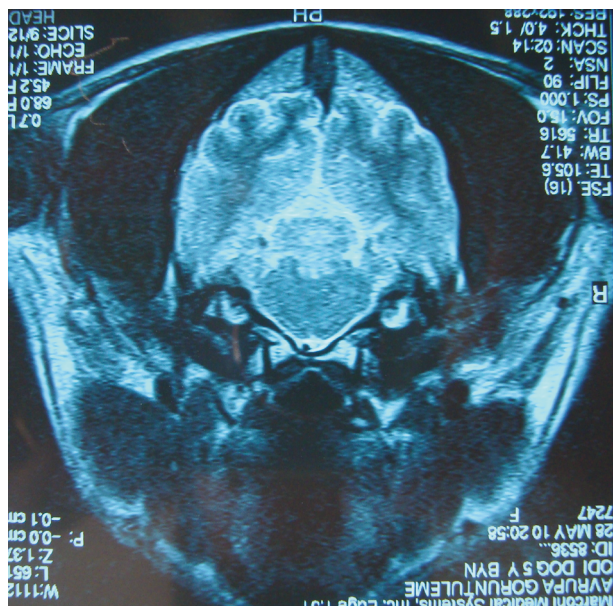


Figure 4. Magnetic resonance imaging of the dog.

were detected in normal ranges, which can be seen in the Table. The recommendation was that the dog should be maintained on levothyroxine sodium, q12h, until all clinical signs were resolved, before an attempt was made to medicate once daily. In this case, the dog was kept on levothyroxine sodium, q12h, even though the clinical signs had resolved. The triglyceride and cholesterol levels of the dog were decreased (triglyceride: 115 mg/dL, cholesterol: 250 mg/dL). When the bodyweight was measured, the dog was found to weigh 14.5 kg after the implementation of levothyroxine sodium.

After the clinical signs were resolved, the dog was medicated once daily at a dosage of 0.01 mg/kg. The patient's owner was instructed to have the thyroid hormones checked every 3 months.

Following the treatment, facial paralysis and the clinical findings of the patient were healed (Figure 6). Reinnervation findings of the EMG, polyphasic potentials, increasing in the number of phases of a motor unit, were observed in head muscles (Figure 7). These EMG findings indicated the healing of the patient.

### 3. Results and discussion

The diagnosis of neuropathy caused by hypothyroidism should be based on a combination of history; clinical signs including neurological findings, hematologic and biochemical changes; the results of thyroid gland function tests as well as the results of electrophysiological examinations and analysis of muscle and nerve biopsies (13). Evaluation of the canine TSH level is a good confirmatory test and has a specificity of 98% when it is used in conjunction with evaluation of the total or free T4



Figure 5. Hyperpigmentation of the dog.

levels (2). In our case, canine TSH, total T4, and fT4 were detected.

A neurologic examination must be performed to establish the localization of the lesion and thereby the peripheral and/or central localization. Electrophysiological testing such as EMG and nerve conduction studies can be used in diseases of the peripheral nervous system and musculature in order to establish the peripheral localization and to differentiate a myopathy from neuropathy (13).

Table. Thyroid profile of the dog at the first and second referrals.

	First referral	Second referral	Reference ranges
T3 (ng/dL)	31	67	50-160
T4 (µg/dL)	0.8	1.8	1.3-2.9
TSH (ng/mL)	0.34	0.13	<0.5
fT4 (ng/dL)	0.45	1.3	0.7-2.1



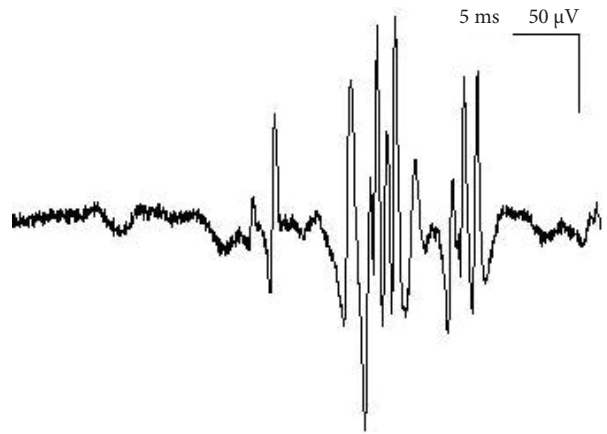
**Figure 6.** After the treatment, normal facial view.

As EMG findings, the occurrence of fibrillation potentials positive sharp waves and complex repetitive discharges has been described in dogs with hypothyroidism (13). Dysfunction of multiple cranial nerves (facial, trigeminal, and vestibulocochlear) with or without abnormal gait and postural reactions has been reported in dogs with hypothyroidism (10). Facial nerve damage and vestibular dysfunction were also seen in our patient. These findings were supported by the literature information.

Cranial nerve deficits associated with hypothyroidism can involve single or multiple nerves, but those most commonly affected are the vestibular, trigeminal, and facial nerves. Clinical manifestations can be unilateral or bilateral; they include head tilt, nystagmus, strabismus, decreased facial sensation, and facial paralysis. (6). The clinical findings of the patient were similar to the symptoms in the literature.

Peripheral neuropathy caused by hypothyroidism affects primarily middle aged and older individuals, especially of middle to large sized breeds (6,10,15). In our case, the patient was a middle sized breed (American Cocker) and 7 years old.

There is no proven association that supports a connection between hypothyroidism and the occurrence of facial paralysis as a single sign in dogs. One source



**Figure 7.** Polyphasic motor unit potentials.

indicates that the facial dysfunction seldom improves on treatment using levothyroxine, which gives some reason to question if hypothyroidism causes facial paralysis alone (13). Treatment of dogs with hypothyroidism is easily achieved with oral levothyroxine supplementation (2,6). Dogs improve rapidly within a few days after the start of treatment (1). The majority of dogs with hypothyroidism with neurological deficits will show partial or complete resolution of their neurologic signs over 2–4 months, with improvement often noted within the first week of treatment (6). The patient had showed improvement in the 2 weeks after the use of oral levothyroxine. With appropriate treatment, hypothyroidism can be well managed and have an excellent prognosis (2). The dog's facial paralysis was improved within 2 months of the administration of levothyroxine sodium. Treatment with thyroxine supplementation is successful in most cases of polyneuropathy and peripheral vestibular syndrome (13).

Hypercholesterolemia, hypertriglyceridemia, and hyperlipidemia are commonly noted in serum biochemistry results in hypothyroidism (3,11). In our case, the dog had hypercholesterolemia, hypertriglyceridemia, and hyperlipidemia when the disease had been first recognized.

Eventually, dogs with facial paralysis must be evaluated for thyroid functions to be certain of the cause and to help determine treatment duration. Further studies are necessary to determine the disturbances caused by hypothyroidism.

## References

1. Suraniti, A.P., Gilardoni, L.R., Rama Llal, M.G., Echevarria, M., Marcondes, M.: Hypothyroid associated polyneuropathy in dogs: Report of six cases. *Braz. J. Vet. Res. Anim. Sci.*, 2008; 45: 284–288.
2. Scott-Moncrieff, J.C.: Clinical signs and concurrent diseases of hypothyroidism in dogs and cats. *Vet. Clin. North. Am. Small Anim. Pract.* 2007; 37: 709–722.



3. Finora, K., Greco, D.: Hypothyroidism and myxedema coma. *Compendium*, 2007; 29: 19–32.
4. Dixon, R.M., Reid, S.W.J., Mooney, C.T.: Treatment and therapeutic monitoring of canine hypothyroidism. *J. Small Anim. Pract.*, 2002; 43: 334–340.
5. McKeown, H.M.: Hypothyroidism in a boxer dog. *Can. Vet. J.*, 2002; 43: 553–555.
6. Blois, S.L., Poma, R., Stalker, M.J., Allen, D.G.: A case of primary hypothyroidism causing central nervous system atherosclerosis in a dog. *Can. Vet. J.*, 2008; 49: 789–792.
7. Dodds, W.J.: Thyroid disease and autoimmune thyroiditis. <http://www.homevet.com/pet-care-library/item/336-thyroid-disease-and-autoimmune-thyroiditis-with-dr-dodds>.
8. Taeymans, O., Daminet, S., Duchateau, L., Saunders, J.H.: Pre- and post-treatment ultrasonography in hypothyroid dogs. *Vet. Radiol. Ultrasound*, 2007; 48: 262–269.
9. Dixon, R.M.: Canine hypothyroidism. In: Mooney, C.T., Peterson, M.E., Eds. *BSAVA Manual of Canine and Feline Endocrinology*. 3rd ed., BSAVA, Gloucester. 2004; 76–94.
10. Jaggy, A., Oliver, J.E., Ferguson, D.C., Mahaffey, E.A., Glaus Jun, T.: Neurological manifestations of hypothyroidism: a retrospective study of 29 dogs. *J. Vet. Intern. Med.*, 1994; 8: 328–336.
11. Panciera, D.L.: Is it possible to diagnose canine hypothyroidism? *J. Small Anim. Pract.*, 1999; 40: 152–157.
12. De Luca, C.J.: The use of surface electromyography in biomechanics. *J. Appl. Biomech.*, 1997; 13: 135–163.
13. Fors, S.: Neuromuscular manifestations of hypothyroidism in dogs. *Svensk Veterinärtidning*, 2006; 14: 11–17.
14. Holiday, T.A.: Electrodiagnostic examination: somatosensory evoked potentials and electromyography. *Vet. Clin. North. Am. Small Anim. Pract.*, 1992; 22: 833–857.
15. Jaggy, A., Oliver, J.E.: Neurologic manifestations of thyroid disease. *Vet. Clin. North. Am. Small Anim. Pract.*, 1994; 24: 487–494.