*In cooperation with*

# What Is Your Neurologic Diagnosis?

**Signalment**: A 4-year-old 52.7-kg (115.9-lb) spayed female German Shepherd Dog.

**History**: The dog was evaluated because of progressive ataxia, generalized weakness, head shaking, and a head tilt. The owners reported that the weakness along with the head tilt and head shaking began suddenly 1 week prior to the initial evaluation. In the interval prior to evaluation, the weakness had progressed and the dog was unable to stand unaided. The dog had a history of chronic ear infections bilaterally.

**Physical examination**: At the initial evaluation, the dog was sternally recumbent and unable to rise without assistance. The dog was overweight (body condition score, 6/9) and dehydrated (5%). Both ear canals were clear, and no other physical abnormalities were grossly apparent.

**Neurologic examination**

**Observation**

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Mental | Alert | | Depressed | X | Disoriented | | Stupor | | Coma | | |
| Posture | Normal | | Head tilt | X | Tremor | | Falling | X | | | |
| Gait | Normal | | Ataxia | X | Pelvic limbs | | All 4 | X | Circling | | |
| Paresis | Pelvic limbs | | Tetra | X | Hemi | | Mono | | | | |
| Other | Head tilt was to the right; ataxia appeared to be vestibular in origin | | | | | | | | | | |

**Postural reactions**

Key: 4 = exaggerated, clonus; 3 = exaggerated; 2 = normal; 1 = diminished; 0 = none; NE = not evaluated

| | LF | RF | LR | RR |
|---|---|---|---|---|
| Wheelbarrow | NE | NE | | |
| Hopping | NE | NE | NE | NE |
| Ext postural thrust | | | NE | NE |
| Proprioceptive pos | 1 | 1 | 1 | 1 |
| Hemistand/walk | 1 | 1 | 1 | 1 |
| Placing–tactile | NE | NE | | |
| Placing–visual | NE | NE | | |

**Spinal reflexes**

| | LF | RF | LR | RR |
|---|---|---|---|---|
| Quadriceps | | | 2 | 2 |
| Extensor carpi | 1 | 1 | | |
| Flexion | 1 | 1 | 1 | 1 |
| Crossed extensor | 0 | 0 | 0 | 0 |
| Perineal | | | 2 | 2 |

**Cranial nerves**

| | L | R | | L | R | Comments CN |
|---|---|---|---|---|---|---|
| II, VII–Vision menace | 2 | 2 | VIII–Nystagmus, resting | 2 | 2 | Additional problem—head tilt to the right. |
| II, III–Pupils resting | 2 | 2 | VIII–Nystagmus, change | 2 | 2 | |
| Stim L | 2 | 2 | V–Sensation | 2 | 2 | |
| Stim R | 2 | 2 | VII–Facial mm | 2 | 2 | |
| II–Fundus | NE | NE | V, VII–Palpebral flex | 2 | 2 | |
| III, IV, VI–Strabismus, resting | 2 | 2 | IX, X–Gag | 2 | 2 | |
| III, IV, VI, VIII–Strabismus, position | 2 | 4 | XII–Tongue | 2 | 2 | |

**Sensation** (Locate and describe abnormal)

| | | |
|---|---|---|
| Hyperesthesia | 0 | |
| Superficial pain | 2 | |
| Cutaneous reflex | 2 | |
| Deep pain | 2 | |

**What is the problem? Where is the lesion? What are the most probable causes of this problem? What is your plan to establish a diagnosis? Please turn the page.**

## Assessment

### Anatomic diagnosis

| Problem | Rule out location |
|---|---|
| Right head tilt, vestibular ataxia, and right ventral strabismus | Peripheral or central vestibular system |
| Tetraparesis | Cervical myelopathy, peripheral neuropathy, or myopathy |
| Decreased spinal reflexes | Peripheral neuropathy |

### Likely location of one lesion

| |
|---|
| Diffuse peripheral neuropathy |

### Etiologic diagnosis

| Rule out disease process | Diagnostic plan (in order of priority) |
|---|---|
| Inflammatory or infectious disease process | Clinicopathologic analysis and urinalysis—to establish general health status and metabolic status and investigate for evidence of hypercalcemia, hypoglycemia, or hyperadrenocorticism (high alkaline phosphatase activity, low BUN concentration, and mildly high blood glucose concentration) |
| Endocrine or metabolic disease | Assessment of serum concentration of thyroid hormones—to rule out hypothyroidism |
| | Extensive bathing—to examine for the presence of an attached tick |
| | Radiography and ultrasonography—to rule out grossly evident neoplastic disease |
| Neoplasia or paraneoplastic disease | Electromyography and assessment of nerve conduction velocity—to better characterize extent of neuromuscular abnormalities |
| | Muscle and nerve biopsy—to determine histopathologic characteristics of neuromuscular abnormalities |
| Idiopathic | CSF analysis—to evaluate for inflammatory disease |

**Comments**: On the basis of examination findings, a differential diagnosis of myasthenia gravis was discussed as an initial possibility. However, myasthenia gravis most commonly causes intermittent neuromuscular weakness that is exacerbated by exercise and is associated with apparently normal spinal reflexes. Because the dog had a persistent weakness that did not change with activity and decreased spinal reflexes in all limbs, that possibility was considered unlikely. Additional differential diagnoses included chronic organophosphate exposure (resulting in demyelination) and acute neuromuscular disease (eg, botulism). The possibilities of organophosphate toxicosis and botulism were also considered unlikely because the dog was well supervised, resided in an urban environment, and had no history of such exposure.

**Abnormal test results**:

**Abnormal laboratory data**: Abnormalities detected via CBC included lymphopenia (240.8 lymphocytes/μL; reference range, 1,000 to 4,800 lymphocytes/μL). Results of serum biochemical analyses included mild hypokalemia (3.8 mEq/L; reference range, 3.9 to 5.3 mEq/L), hypochloremia (105 mEq/L; reference range, 107 to 124 mEq/L), hypercalcemia (11.4 mg/dL; reference range, 9.2 to 11.2 mg/dL), hypophosphatemia (2.7 mg/dL; reference range, 3.2 to 6.2 mg/dL), hyperproteinemia (7.7 g/dL; reference range 5.2 to 7.1 g/dL), low alkaline phosphatase activity (12 U/L; reference range, 20 to 115 U/L), high alanine aminotransferase activity (111 U/L; reference range, 24 to 105 U/L), severe hypercholesterolemia after food had been withheld (1,740 mg/dL; reference range, 140 to 270 mg/dL), hypertriglyceridemia after food had been withheld (384 mg/dL; reference range, 24 to 115 mg/dL), and high creatine kinase activity (858 U/L; reference range, 60 to 270 U/L). A sample of urine obtained via cystocentesis was analyzed; specific gravity was 1.036, and bacteria were present (1+).

**Serum thyroid hormone analysis**: Serum total thyroxine concentration was < 0.5 μg/dL (reference range, 1.3 to 2.7 μg/dL).

**Imaging procedures, electromyography, nerve conduction velocity assessment, and muscle or nerve biopsy**: Not performed.

**Presumptive diagnosis**: For this dog, detection of severe hypercholesterolemia and hypertriglyceridemia (determined after food had been withheld) along with low serum total thyroxine concentration was highly sug-

gestive of primary hypothyroidism. Ideally, this diagnostic workup would also have included the measurement of serum thyroid-stimulating hormone concentration followed by electrodiagnostic procedures, such as an electromyography and nerve conduction velocity assessment. However, because of financial concerns, no additional evaluations were performed. Primary hypothyroidism in dogs is a relatively common endocrine disorder that typically affects middle-aged to older large-breed dogs. The common signs include lethargy, exercise intolerance, alopecia, and weight gain. Less commonly, various peripheral neuropathic and myopathic clinical signs may be evident, including diffuse neuromuscular weakness, hyporeflexia, proprioceptive deficits, and reluctance to rise.[1] In addition, unilateral cranial nerve abnormalities (most commonly associated with cranial nerves V [usually sensory dysfunction], VII, and VIII[1,2]) may develop. The precise etiology of these abnormalities is yet undetermined. It is thought that demyelination and axonal degeneration both play roles in the diffuse lower motor neuron signs in affected dogs. Compression of the selected cranial nerves by myxedematous tissue at the locations where they exit the skull and travel through the head and neck has been suggested to be an explanation for unilateral cranial nerve deficits.[2] Primary hypothyroidism–associated myopathic disease can be detected via histologic examination of tissue samples and serum biochemical analyses.[3,4] In dogs with hypothyroidism, serum creatine kinase activity can be elevated, albeit often only mildly.[4] On the basis of severe hypercholesterolemia, hypertriglyceridemia, high creatine kinase activity, and substantially low serum total thyroxine concentration along with diffuse signs of a peripheral neuropathy and unilateral cranial nerve VIII neuropathy, a diagnosis of suspected primary hypothyroidism was made.

**Prognosis with treatment**: The prognosis for improvement of primary hypothyroidism–associated peripheral neuropathy with proper administration of hormone supplements is good, although weeks to months of treatment are usually required.[1] The resolution of the vestibular abnormalities may not be complete, and signs may take several months to improve, although dogs with these signs usually compensate well over time.[1]
**Prognosis without treatment:** Because of the severity of the dog's clinical signs and clinicopathologic abnormalities, the prognosis for return to function without administration of a thyroid hormone supplement would be guarded.

**Therapeutic plan**: The owners decided to pursue treatment for the suspected primary hypothyroidism and concurrent mild dehydration. The dog was hospitalized for 6 days for supportive care and daily rehabilitation including range of motion and standing exercises. Intravenous fluid therapy (2.2 mL/kg/h [1 mL/lb/h]) was provided for 24 hours until the dog was eating and drinking normally and no longer dehydrated. Administration of a thyroid hormone supplement (levothyroxine; 0.02 mg/kg [0.01 mg/lb], PO, q 12 h) was initiated.

**Outcome**: During the following 6 days, the dog became weakly ambulatory with minimal assistance. On day 4 of hospitalization, serum cholesterol concentration was 1,109 mg/dL. Four months following initiation of treatment, the dog was fully able to ambulate with only minimal weakness; spinal reflexes were considered normal, and the head tilt and ventral strabismus had completely resolved.

## References


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