Peripheral Neuropathy: A Case Study

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Abstract

This paper discusses a patient seen in the outpatient neurology clinic at a county
hospital in a large metropolitan city. His chief complaint was increasing weakness in his lower
extremities over the past 3 months. Past medical history includes a four year history of multiple
sclerosis and neurogenic bladder, a one year history of diabetes mellitus, and a history of alcohol
abuse. Based on the full health history and physical examination, a diagnosis of peripheral
neuropathy was made. The etiology of the neuropathy needed to be explored. This paper discusses
three likely differential diagnoses of the peripheral neuropathy and the relevant studies that would
rule out or confirm each diagnosis. The treatment plan was based on the final diagnosis of axonal
degeneration secondary to metabolic/diabetic neuropathy.

The information that follows was obtained at an outpatient neurology clinic at a county hospital
in a large metropolitan city. The patient seen was a 46 year old black male that was at the clinic for
a follow up appointment. His chief complaint was progressive lower extremity (LE) weakness
stating his knees keep "buckling up from under" him. Vital signs: BP 135/80, HR
84, RR 20, temp 98.9 F.

HISTORY / PHYSICAL EXAMINATION

History of Present Illness: Progressive weakness started approximately 3 months ago with right
leg first and then left. Weakness is worse when first arising from chair or bed and gradually gets
stronger once he ambulates, but will worsen again if engaged in prolonged activity. Patient now
uses a cane to assist with walking as well as a leg brace on the right leg. Denies any range of
motion (ROM) limitations, but does take longer to do activities of daily living (ADL's). No
weakness in upper extremities (UE). Reports frequent episodes of numbness below calves, as well
as rare numbness in right wrist/hand. Denies pain, tremors, twitching, swelling, redness, or
tenderness in muscles or joints. Denies backaches, arthritis, or musculoskeletal trauma. Patient
believes it to be worsening of his multiple sclerosis.

Allergies: none

Medications: Ditropan 5mg PO BID, Baclofen 10mg PO TID, Colace 50mg PO prn constipation.

Past Medical History: Multiple sclerosis (MS) and neurogenic bladder for 4 years, diabetes mellitus (DM) for 1 year. Denies coronary artery disease (CAD), hypertension (HTN), or known peripheral vascular disease. Denies any previous surgeries. Hospitalized in November 1996 and Summer 1994 for exacerbation of MS symptoms and treated with intravenous (IV) steroids.

Family Medical History: Mother (69 y.o.) with HTN; father died at age 56 from industrial accident; 4 brothers (40 - 48 y.o.), one with HTN and one with alcohol abuse. Denies any known CAD, peripheral vascular disease (PVD), DM, MS, cerebral vascular accident (CVA), cancer (CA), or sickle cell anemia.

Personal/Social History: Smokes 1 pack per day (ppd) x 20 years, 3-4 beers/day, up to 6-8/day on weekends. Denies illicit drugs. Unemployed at present and lives with girlfriend and her two children whom he watches when she is at work States one child has been "a problem lately" and has added stress to their lives and relationship.

Review of Systems: General: Denies weight or appetite changes, denies generalized fatigue, chills or fever. Skin: Denies any rashes, lumps, sores, itching or changes in color.

HEENT: Denies headache (HA) or head trauma. Denies hearing loss, tinnitus, vertigo or ear discharge. Denies eye pain, exudate, excessive tearing, glaucoma, cataracts, or use of contacts/glasses. Reports occasional blurry vision and rare double vision. Reports occasional colds and sinus trouble with nasal drainage, denies any nose bleeds, chronic sinusitis, nasal trauma or change in sense of smell. Denies problems with teeth or gums other than occasional cavity, oral sores, hoarseness, sore throat/tongue or voice changes. Neck: Denies any lumps, injuries, swollen glands, stiffness or pain. Respiratory: Denies cough or sputum production, hemoptysis, asthma, wheezing, and TB. Reports frequent bronchitis problems last year but none in approximately 6 months. Denies shortness of breath or pneumonia. Cardiac: Denies any heart trouble, HTN, chest pain, palpitations, dyspnea, PND, orthopnea, masses or lumps on chest/thorax. Denies pain, tenderness, discoloration, edema or temperature changes in extremities. GI: Denies any abdominal pain, gastric ulcers, nausea/vomiting, diarrhea, rectal bleeding, bowel incontinence or change in size and color of stool. Denies any trouble swallowing, heartburn, indigestion or regurgitation. Reports occasional constipation that is easily relieved with the Colace prescribed. Denies liver or gall bladder disease, jaundice, hepatitis, or spleen problems. GU: Denies any kidney stones or kidney disease. Reports history of frequency, urgency and incontinence 4 years ago when diagnosed with MS and neurogenic bladder; currently self caths approximately 4 times daily without problems of incontinence. Denies hematuria or change in urine color. Denies hernias, discharge or sores on penis, testicular masses or pain, impotency or STD's. Denies any sexual problems or pain with intercourse. Musculoskeletal: see History of Present Illness (HPI). Neurological: Denies fainting, seizures, tingling or tremors. (See HPI). Endocrine: Denies thyroid problems, heat/cold intolerances, or excessive sweating. DM diagnosed 1 year ago; controlled with diet; does not check blood sugars at home. Psychiatric: Denies nervousness, memory problems, or depression. Reports more than usual stressed/tense emotions in past 4 months due to girlfriend's children.

Physical Examination: 46 year old black male in no distress sitting on examination table, alert and oriented (AO) x 3, cooperative, communicates well with good eye contact. Skin: Brown, dry, turgor with instant recoil, no lesions or tenderness. Nail beds pink without clubbing, brisk capillary refill. Hair coarse with normal distribution. HEENT: Scalp freely movable without lesions or tenderness, well-spaced symmetric facial features. No periorbital edema or ecchymosis, lids and lashes normal. PERRLA, internuclear ophthalmoplegia (INO) present bilaterally, full visual fields, red reflex present, no papilledema or AV nicking noted. Cornea and lens clear, retina pink, no hemorrhages or exudate. External ears normal in shape/size, tympanic membranes intact, gray and translucent, light reflex and bony landmarks present. Rinne - air > bone conduction bilaterally, Weber midline. No flaring of nares, patent bilaterally with septum slightly to right of midline, pink/moist mucosa, no lesions or polyps, no discharge. No frontal or maxillary sinus tenderness on palpation. Buccal mucosa pink/moist, no lesions, multiple fillings noted, pink gingiva. Tongue midline, no lesions, uvula midline, pharynx without lesions, drainage or erythema, gag reflex intact. Neck: No masses, lymphadenopathy; trachea midline, thyroid borders palpable without enlargement or nodules. Full ROM, + L'Hermitte's sign, no carotid bruits, stridor or tenderness. Chest: Lungs clear to auscultation bilaterally, vocal and tactile fremitus equal from apex to base, expiration = inspiration, resonant percussion throughout. Palpation of chest/thorax reveals no deformity or tenderness to ribs, sternum, clavicles or scapulae, no scoliosis of spine. Cardiovascular: S1S2 with rate of 80 beats per minute (bpm), no murmurs, rubs, clicks or extra heart sounds. PMI at 5th intercostal space, midclavicular line, no heaves, lifts or thrills. Apical and radial pulse equal. Abdomen: Rounded abdomen without scars or lesions, bowel sounds x 4 quadrants, no bruits auscultated. Liver span 9 cm at right midclavicular line by percussion, unable to palpate liver, spleen or kidneys, no tenderness, distention, rebound or guarding. Extremities: Radial, femoral, dorsalis pedis (DP) and posterior tibial (PT) pulses equal bilaterally; full ROM to shoulders, elbows, wrists, hips, knees and ankles bilaterally. Strength 5+ / 5+ in UE bilaterally, 4+ / 5+ in LE bilaterally. No swelling, tenderness, heat or erythema noted in any extremity. Neurologic: AO x 3, answers questions appropriately and timely, long and short term memory intact, good articulation and speech pattern. Cranial nerves (CN) V-XII grossly intact, no adduction on attempted horizontal gaze to one side with nystagmus in abducting eye (INO) bilaterally. Rapid alternating movements and finger-to-nose test normal, + Romberg test. Unsteady gait, uses examining table and wall for support, unable to do heel-toe walking. Dull/sharp sensation intact in UE bilaterally, but decreased in LE bilaterally, vibration sensation intact in UE but decreased at ankles/toes bilaterally. Two point discrimination intact in UE and trunk, but decreased in LE. Biceps, brachioradial, triceps, and patellar reflexes 4+ / 4+

bilaterally, achilles reflex 3+ / 4+ bilaterally, positive Babinski sign bilaterally, positive ankle clonus bilaterally.

Lab Work: No updated lab work since patient's hospitalization in November.

DIFFERENTIAL DIAGNOSIS

Subjective and objective cues that emanate from the interview and physical exam will initiate hypothesis generation of possible diagnoses for this patient. This hypothesis list will be revised and refined as the process of data gathering and interpretation evolves. Once relevant data are accumulated, one or more hypotheses are accepted as sufficiently valid to guide further testing, therapeutic, or prognostic decisions ([1]). These surviving hypotheses comprise the differential diagnosis list. The three most probable differential diagnoses for this particular patient are based on the physical exam (PE) findings and historical data, mainly, MS, DM, and alcohol use. The PE findings and chief complaint point to a peripheral neuropathy which could be an exacerbation of his MS, or a result of a new compounding problem. Therefore, the three most likely differential diagnoses for this patient are: (a) MS exacerbation with demyelination neuropathy, (b) toxic/nutritional neuropathy secondary to alcohol use, and (c) metabolic neuropathy secondary to DM.

A) MULTIPLE SCLEROSIS

Multiple sclerosis is characterized by demyelination (myelin destruction), chronic inflammation and scarring of the white matter of the brain and spinal cord. This disease is the major cause of chronic disability in young adults and is associated with exacerbations and remissions of symptoms ([2]). The sporadic patches of demyelination cause varied neurologic function throughout the body. Partial remyelination can occur after an attack which accounts for partial resolution of symptoms. These manifestations can range from a benign illness to a rapidly evolving and disabling disease ([3]). The exact etiology is unknown, but research suggests a combination of an autoimmune response, genetic susceptibility, and environmental exposures ([4]). Indirect evidence that emotional stress, overwork, fatigue, pregnancy, and acute respiratory infections precede the onset supports this theory (2). The initial symptoms of MS in order of highest to lowest occurrence are weakness, sensory loss, paresthesias, optic neuritis, diplopia, ataxia, vertigo, bladder dysfunction, L'Hermitte, pain, dementia and visual loss (3).

The clinical course usually follows one of three patterns: relapsing/remitting, chronic progressive, and relapsing/progressive. Relapsing/remitting is characterized by attacks followed by near complete or complete remissions (4). These patients may eventually develop chronic/progressive symptoms with steady gradual worsening, which is also found in patients that develop MS later in life. The relapsing/progressive course has chronic progressive disease exacerbated by acute attacks and minimal remission (4). The patient described in this case study appears to be following the relapsing/remitting course thus far. He reported near complete remissions of symptoms after the two hospitalizations and IV steroid treatments, except for continuous need of self-catheterization. Although those two attacks were characteristic of acute onset of symptoms and the current onset of symptoms was much slower, there is a possibility these symptoms are a manifestation of MS exacerbation. The patient's report of recent increased stress and anxiety in his life could contribute to an exacerbation. The small amount of Baclofen the patient is taking should not produce that much muscle weakness, and especially not limited to the lower extremities.

According to Gross ([5]), proprioceptive loss resulting in sensory ataxia and abnormal gait may occur in peripheral neuropathy or in disease of the posterior columns of the spinal cord. A positive Romberg test is a relevant positive physical finding for the two possibilities. If proprioceptive loss is from spinal cord disease, upper motor neuron findings may be present such as positive Babinski's sign and spasticity (5). In order to confirm whether the symptoms are a result of a MS exacerbation neuropathy or a type of secondary neuropathy, an electromyogram (EMG) and nerve conduction study (NCS) were ordered.

The EMG and NCS provide one of the best means of diagnosing myopathies and neuropathies ([6]). The EMG is used to detect and measure the electrical discharge, or action potential, originating in a skeletal muscle. Skeletal muscle at rest has little electrical activity. An evidence of spontaneous, involuntary electrical activity while a muscle is at rest generally indicates some neuromuscular abnormality (6). The NCS records electrical response of a muscle to stimulation of its motor nerve at two or more points along its course. This permits conduction velocity to be determined

between the points of stimulation. Sensory NCS determine the conduction velocity and amplitude of action potentials in sensory fibers between two points (3). These measurements are then compared with a published norm, such as those listed by DeLisa ($_7$).

The two major components in peripheral nerves are the axon and myelin. Therefore, a peripheral neuropathy can be classified according to the predominant pathological involvement: axonal degeneration or segmental demyelination. With peripheral neuropathy, the NCS should be done in testable nerves in both legs and in one arm as nerve conduction abnormalities are not observed uniformly in all peripheral nerves ($_8$). This patient's EMG and NCS results are described in Table 1 and Table 2.

Although the EMG findings were within normal limits, there were several abnormalities in the NCS according to those normal values reported by DeLisa (7). Sural nerves were unobtainable bilaterally with decreased sensory nerve action potential (SNAP) amplitudes. There was also decreased motor amplitudes except right ulnar, and there was no desynchronization of compound action potential (temporal dispersion). The nerve conduction velocity (NCV) can be used to differentiate between axonal degeneration and segmental demyelination. The axon is the lest effective in action potential conduction, and the myelin is the most effective due to saltatory conduction in myelinated fibers. The NCV will be mildly slow or normal (>35 m/sec) in axonal degeneration, but will be markedly slow (<30 m/sec) in segmental demyelination (8). Based on the above EMG and NCS, this patient was diagnosed with sensory motor axonal peripheral neuropathy. This finding rules out the first differential diagnosis of MS exacerbation with demyelination neuropathy.

Sensory motor axonal peripheral neuropathy can be caused by either one of the remaining differential diagnosis since both metabolic and toxic/nutritional neuropathies can be manifested as such (8). Pruitt ($_9$) listed diabetes, alcohol abuse, and B-vitamin deficiency as common causes of subacute (weeks to 1-2 years) sensorimotor peripheral neuropathies.

B) TOXIC / NUTRITIONAL

According to Oh (8), alcoholic neuropathy is one of the most common forms of peripheral neuropathy, and about 20% of alcoholic patients are known to have peripheral neuropathy. Nerve conduction abnormalities are observed in 88% of cases in at least 2 out of 4 nerves in sensory NCS and 2 out of 3 nerves in motor NCS. The predominant process in alcoholic neuropathy is axonal degeneration of the heavily myelinated fibers (8). Alcoholic neuropathy is distal, symmetric, and involves both the sensory and motor systems. B vitamin deficiencies can also contribute to the neuropathy ($_{10}$). Alcohol metabolism results in a dangerous shift of the body's acid-base balance toward acid and increased fatty acid synthesis causes the liver to become clogged with fat ($_{11}$). The liver becomes very inefficient and impairs a person's nutritional health in ways that cannot be corrected by diet alone.

An assessment of alcohol related behavior may be made with the Michigan Alcoholic Screening Test (MAST) or with the CAGE questionnaire ($_{12}$). Lab studies that would help identify impaired hepatic function include liver function tests (LFTs). Aspartate aminotransferase (AST) concentration is slightly increased in alcoholic cirrhosis, but has a three to sixfold increase in alcoholic hepatitis ($_{13}$). The sedimentation rate (ESR) will be increased in inflammatory or degenerative process. Vitamin B12 and folate levels are usually decreased in the alcoholic due to nutritional deficits ($_{14}$). If the lab results indicated alcoholic process with nutritional deficits, the treatment would be abstinence and vitamin replacement, such as EC thiamine 40mg PO QD and a multivitamin 1 PO QD ($_{15}$). The symptoms should improve over 4-6 months if the patient adheres to treatment (10). A referral to Alcoholics Anonymous should be made as well.

NCS identifies neuropathy in 76-80% of patients with diabetic neuropathy (8). Among the various electrophysiological parameters, the sensory action potentials have been shown to be the most sensitive indicator for neuropathy in diabetic patients. Median sensory conduction abnormalities were observed in 80% of patients, whereas the motor distal latency was abnormally delayed in 40% (8).

C) DIABETES MELLITUS

Diabetes Mellitus is a chronic disease of insulin deficiency or resistance, and is characterized by disturbances in carbohydrate, protein, and fat metabolism ($_{16}$). Insulin transports glucose into the cell for use as energy and storage as glycogen, as well as stimulates protein synthesis and free

fatty acid storage in the fat deposits. Insulin deficiency compromises the body tissue's access to essential nutrients for fuel and storage ([17]). Type II diabetes is often a finding discovered by chance on screening urinalysis or blood sugar ([18]) as was the case with this patient one year ago. He was advised to exercise at least 3 times weekly and eat a diet high in carbohydrates and fiber. No medication or continuous blood sugar monitoring were prescribed for him at the time of diagnosis. The patient stated that he did try to increase his exercise immediately after the diagnosis, but that only lasted a couple of months. He reports that he has not been eating "right" either, and averages only one full meal a day with multiple small snacks.

Due to the patient's lack of exercise and diet control, increased stress, and lack of blood work since November, the possibility of undetected prolonged hyperglycemia existed. The patient's glucose levels were >200 when hospitalized in November and given IV steroids. Degenerative complications of DM, such as neuropathy, may be present in the absence of symptomatic hyperglycemia ([19]) and nerve conduction abnormalities can occur early in the disease (8). Hyperglycemia-induced sorbitol accumulation in nerve tissues may lead to axonal loss and peripheral neuropathy, which is predominantly sensory in nature and usually effects the lower extremities (18).

A basic lab test to get a rough estimate of current glucose control is the urine ketone test which is a part of the average urinalysis (UA). The amount of ketones present in the urine indicates the severity of metabolic acidosis ([20]). A random blood sugar would indicate the glucose level at the time the blood was drawn, but is a poor test to assess control over time. Hemoglobin A1c (HbA1c) is the glycosylated form of hemoglobin in the red cell. Measurement of the HbA1c allows an assessment of overall glycemic control for the previous 2-3 months, which is the life span of the red blood cell (RBC) (18). Values of 11-12% correlate with glucose levels >300 mg/dL and indicates poor carbohydrate control (18). This patient's HbA1c value was 13% of total hemoglobin.

The Diabetes Control and Complications Trial [3] has demonstrated conclusively the benefit of tight glycaemic control as compared to poor control. Tight glycaemic control was defined as mean glycated Hgb concentrations of 1 and 3% above the non-diabetic reference range (17). Intensive insulin therapy with those patients who did not have neuropathy at base line reduced the appearance of neuropathy at 5 years to 3% versus 10% in the conventional therapy group. Similarly, intensive therapy reduced the appearance of already existing neuropathy from 16% to 7% at 5 years (17). It is clinically plausible to assume that such tight control in noninsulin dependent diabetes mellitus (NIDDM) would also reduce the risk of neuropathy in those patients as well. NIDDM patients respond well to sulfonylureas which act by stimulating release of insulin from the beta cell (19). Sulfonylureas increase insulin release with lowering of the blood glucose level. The insulin levels decrease as the glucose level falls, which masks the initial stimulation of insulin release since blood glucose is the major stimulus to insulin release (19). The patient could be started on glyburide 5mg PO QD and instructed on potential side effects as well as signs/symptoms of hypoglycemia (16). He needs to be instructed on how to monitor his blood sugars at home and be reminded of proper exercise/diet routine. He should be referred to the General Medicine clinic for future follow up concerning his DM as well as the diabetic educator class for more detailed teaching. In the future, the patient might also benefit from aldose reductase inhibitors (ARIs) which prevent and reverse nerve conduction deficits and biochemical abnormalities in diabetic animals. One study found increases in the numbers of regenerating fibers in sural nerve biopsies from patients treated with ARI ([21]). The results of the multi-national randomized trials are still eagerly awaited.

If the patient monitors his blood sugar regularly, adheres to the glyburide therapy, and follows his diet/exercise instructions, he might be able to eventually discontinue the glyburide. Hopefully his neuropathy will improve with better glucose control, which will make it easier to exercise as he should. He will need support from his girlfriend to assist with proper diet, as well as assist and encourage him to properly monitor his blood sugar. His MS and neurogenic bladder will be lifelong problems and he will need to continue to self catheterize as well as keep his follow up appointments with the neurology clinic. He will need emotional as well as physical support if there are any more MS exacerbations in the future. The hardest road he may have ahead of him is to seek treatment for alcohol abuse. He will need support and encouragement from his girlfriend, as well as we, the healthcare professionals.

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