Pathophysiology: guillain-barre syndrome and cauda equina syndrome

1. Possible Diagnosis (listed in order of likelihood)	Signs and Symptoms seen in This patient that make you consider this as a diagnosis
2. Guillain-Barre Syndrome: Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP)	Difficulty with bladder/bowel function Back pain that may get worse at night Weakness Unsteady walking Tingling and

		numbness in hands and feet
		Sensorimotor disruption
3.	Cauda Equina Syndrome	Lower back pain
		Burning and numbness sensation felt down the buttocks, lower legs and feet
		Bladder incontinence
		Weakness
		Reduced reflexes in

		the lower extremities
		Radiating back pain
4.	Multiple Sclerosis	Nerve pain, burning, numbness and tingling
		Weakness
		Malaise
		Bladder incontinence
5.	Spinal Tumor	Back pain
		Muscle weakness
		Difficulty walking
		Loss of

		sensations
		Urinary incontinence
		Fever
		Trouble walking
6.	Epidural Abscess	Weakness
		Back pain
		Bladder incontinence
7.	Vertebral Osteomyelitis	Radiating back pain, worse at night
		Nerve pain
		Burning, numbness and tingling

		Weakness Bladder incontinence Numbness/ Tingling in hands and feet
8.	Diabetic Neuropathy	Weakness Loss of Reflexes Bladder Incontinence
9.	Transverse Myelitis	Back pain that radiates down the leg Weakness Urinary incontinen

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Numbness, tingling and burning sensation.

1. Differential Diagnosis Table

2. Most Likely Diagnosis

1. Guillian-Barre Syndrome (GBS):

1. Guillian-Barre Syndrome (GBS): A neurological condition where the body’s immune system destroys the peripheral nervous system and damages nerve tissue. Damage is done by invading macrophages that strip axons and damage myelin sheaths (McCance & Heuther, 2019). Typical nerve impulses and innervation of the correct sensory and motor reflexes are hindered. Endoneurium edema, inflammation of the lymphocytes and demyelination cause damage to the spinal roots and nerve terminals via infiltration from the T-cells (McCance & Heuther, 2019).
2. The pathophysiology of this disease is possible for this patient as evidenced by: This patient’s history includes a recent exposure and symptoms of a bacterial gastrointestinal (GI) infection that left him with nausea and vomiting for several days. Campylobacter Jejuni is a common bacterium found in undercooked poultry and can trigger the onset of GBS. A recent infection may also contribute to the

presentation of a temperature of 100. 8. Among these contributing factors are the following symptoms that would lead to the further testing, labs, and imaging to rule out GBS as a diagnosis.

1. Inability to walk: The axons, covered by the myelin sheath allow for effective afferent and efferent pathways. Demyelination of the axons caused by the immune system, stall and destroy this impulse which causes weakness and disrupted proprioception.
2. Numbness in fingers and toes: Paresthesia is felt in the fingers and toes due to abnormal sensations and disrupted messages sent by demyelinated axons to the cerebral cortex. Motor nerve deficits, impairment of pain, slow nerve conduction, and denervation cause problems with the central nervous system and lead to peripheral neuropathy. Peripheral neuropathy can have several sources and labs should be done to rule out causes such as GBS (Misra, Kalita & Nair, 2008).
3. Back pain: Alteration in the peripheral nerves and the inability for the Aβ, Aδ and C fibers of the somatosensory system to transmit signals to the brain for further processing can cause complications like those seen in this patient. The somatosensory system nerves that receive signals will transport these messages to the cerebral cortex via a signal by the thalamic nucleus. A disease process that interrupts this transmission such as peripheral neuropathy can create an altered signal, felt as pain (Colloca et al, 2017).
4. Urinary Incontinence: Autonomic dysfunction can cause occasional incontinence. Bladder disruption from retention may

increase the likelihood of overflow incontinence. Lumbosacral nerves that are either hypo or hyper active or lacking contractility of the detrusor muscle will interrupt the sensation and brain signals that tell the patient to urinate. (Amatya, Khan, Whishaw & Pallant, 2013).

5. Sensorimotor Disruption: 2+/5 toe flexion and extension in this patient and 3+/5 rating in the quadriceps from decreased myotatic reflexes and nerve impulses in the lower extremities. Hyporeflexia from demyelination of the axons and poor transmission is evident.

3. Emphasize and discuss the pathophysiology of this diagnosis.
    1. What causes GBS: Sometimes precipitated by a bacterial infection of the gastrointestinal tract, like this patient who recently had nausea and vomiting after eating chicken. Infectious organisms can cause inflammation of the nerve tissue and destruction of the myelin in AIDP. Surgery, immunizations and viral infections can also lead to the onset of symptoms once the immune response is triggered (MCance & Heuther, 2019). The microorganisms molecularly mimic the peripheral nerve, B-cell and T-cell activation target the healthy myelin and Schwann cells instead and result in peripheral nerve damage and GBS symptoms (Kuwabara, 2004).
    2. How does it develop: After exposure to a bacterial or viral infection, if the microorganism is similar to cells found on the myelin sheath, or the chemical structure is changed, lymphocytes and macrophages, CD4 and CD8 types may

incorrectly attack them in an autoimmune response (Guillan-Barre Syndrome Fact Sheet, 2018). Antibodies against the gangliosides GM1, GM1b, GD1a or GalNAc-GD1a are seen in high volumes in blood samples taken from patients with GBS. It appears the bacterial epitopes and those on the gangliosides mimic each other.

3. Do genetics have a role in this disease: No specific genetic links have been found for the development of GBS. The prevalence is sporadic and multiple conditions can cause the development, however genetic predispositions may lead to its advancement (Guillan-Barre Syndrome Fact Sheet, 2018).
4. Body systems effected/ Alteration of the Anatomy and Physiology/Body Function Compromises: Bilateral, symmetrical numbness and weakness begins in the peripheral limbs and advances proximally. The demyelination of the motor neurons effects the musculoskeletal system and the peripheral nervous system (PNS). Genitourinary, gastrointestinal, and the PNS systems are primarily affected. Autonomic complications such as high blood pressure and cardiovascular dysfunction may occur.
5. Potential Complications: If the condition develops further, respiratory function and paralysis can develop. Mortality from respiratory failure is possible (Walling & Dickson, 2013).

4. Who is at risk for developing this pathological condition?
    1. Who is at risk: Men are at higher risk for developing this autoimmune disease, and older or elderly patients can be more

susceptible to bacterial and viral infections that contribute to the pathogenesis of GBS (Kuwabara, 2004).

2. Prevention: Hand hygiene and proper cooking of poultry and other food can prevent infection. Understanding the risks involved and being aware of symptoms can prevent severe complications from developing.
3. Risk for this patient:

3. Second Most Likely Diagnosis

1. Cauda Equina Syndrome

1. Cauda Equina Syndrome: The tail-like bundle of axons at the end of the spinal column is the cauda equina. The spinal nerves include, 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal, starting below the foramen magnum down the length of the spinal cord and connecting to the peripheral nerves. The bundle of nerve roots that make up the cauda equina exist in the subarachnoid space from the lumbar, sacral and coccygeal nerves and provide sensory and motor innervation to targets along the way (Dawodu et al, 2018). Cauda Equina Syndrome is damage to this bundle of nerve roots.
2. The pathophysiology of this disease is possible for this patient as evidenced by:

1. Weakness in his feet and ankles as well as hindered reflex of the lower extremities: Nerve roots L3-L4 compressed in the CE create a motor deficit present in the quadriceps and compression of the S1-S2 nerve roots in the CE cause motor deficit of bilateral toe flexion as evidenced

by 2+/5 toe flexion and extension in this patient and 3+/5 rating in the quadriceps (Dawodu et al, 2018).

2. Inability to walk: The muscles that are innervated by the nerve roots in this area can cause extreme weakness. Compression and cellular death of the axons and nerve roots will progressively become worse.
3. Numbness and tingling in feet and toes: A peripheral nervous system injury, like that to the nerves in the CE, can present with motor neuron disruption in both the upper and lower sections (Dawodu et al, 2018).
4. Low back pain: Compression of the lumbosacral nerve roots in the lower back radiate down the sciatic area. Muscles in the lower body are innervated by nerves in the CE to provide somatic sensations and carry nerve impulses to sense pain and proprioception.
5. Urinary incontinence: The pelvic splanchnic nerves that are innervated by the nerves from the CE can produce incontinence. The detrusor muscles of the urinary bladder are innervated by parasympathetic, pelvic splanchnic nerves included in the cauda equina region that innervate from the S2-S4 region.

3. Emphasize and discuss the pathophysiology of this diagnosis.

1. What causes CE: CE causes include herniation of the cerebral disc, trauma, spinal stenosis, spinal tumor, infection and even osteoporosis. Infection or an undiagnosed herniated disc could have precipitated this patient’s pain.
2. How does it develop: When the bundle of axons in the spinal column exit into the lumbosacral spinal canal they are at increased risk for trauma and lack protection. Based on the nerve targets along the

spine, if the CE is impinged and compressed into the surrounding tissue by a herniated disc or trauma, symptoms of weakness, sensorimotor deficiency, and pain can persist.

3. Do genetics have a role in this disease: Cauda Equina Syndrome is not genetic and is precipitated by physiological problems from environment, trauma, infection, or other injuries that do not stem from genes or predisposition.
4. Body systems effected/ Compromised: Compression of the tissue on the CE region creates problems throughout the body system.
5. Alteration of Anatomy and Physiology: The epineurium that surrounds the nerve roots is not fully developed and lacks protection necessary. The nerve roots in this area have a higher risk of being damaged and can cause significant complications if not treated, such as paralysis (Dawodu et al, 2018). The nerves innervate targets along the spinal cord, from the conus medullaris to the coccyx, compression of these nerves can lead to neuronal death. The body systems affected are those that have nerve innervation at the point of compression. Genitourinary, gastrointestinal, sciatic and peripheral nerve systems are mainly disrupted.
6. Potential Complications: Paralysis is a likely complication from the compression of the CE region. If neuronal death occurs from constant stress and inadequate interventions in place then the damage may become permanent.

4. Who is at risk for developing this pathological condition?

1. Who is at risk: Populations at risk are older patients who may be susceptible to infections and live a lifestyle that cause trauma or disc herniation primarily.
2. Prevention: Exercise, smoking cessation, and a more active and less sedentary lifestyle can help alleviate problems with the spinal column.
3. Risk for this patient: This patient enjoys working in his yard, engages in physical activity such as softball and basketball as well as chores such as cutting and stacking wood. The strain and motion placed on the spine can increasingly support the
4. likelihood of CES from occurring. Smoking and lack of constant physical exercise to stimulate and increase muscle strength can also contribute to the recent onset.

4. Third Most Likely Diagnosis

1. Multiple Sclerosis

9. Multiple Sclerosis: An autoimmune, inflammatory disease process that attacks the

myelinated axons within the Central Nervous System (CNS) which will cause neurological deterioration and effect several of the body systems via degeneration.

2. The pathophysiology of this disease is possible for this patient as evidenced by:
    1. What causes MS: Proteins found on the surface of the pathogen trigger the autoimmune response to destroy the antigen or healthy tissue in the area.

2. How does it develop: Typically, this disease is caused by a viral infection, lesions or inflammatory processes that attack the CNS when found upon autopsy can reveal lymphocytes and macrophages present at the damaged axons. Scar formation on the non-neuronal cells in the CNS, as well as loss of oligodendrocytes are cased by autoimmune created antibodies that cause demyelination of the myelin sheath.
3. Do genetics have a role in this disease: Certain genetic predispositions can create an environment where the body may develop this disease.
4. Body systems effected:
5. Alteration of Anatomy and Physiology/ Body Function Compromise: Lesions on non-neuronal cells in the CNS and loss of myelin sheaths to protect the axons will disrupt the nerve conduction. The inflammatory response to the neurons in the white and gray matter lack oxidation to cause further injury from hypoperfusion (McCance & Heuther, 2019).
6. Potential Complications: With the lack of relapse in this disease the damage to the neurons can become permanent, atrophy to the brain and demise of the cells create more severe symptoms and lasting complications.

3. Emphasize and discuss the pathophysiology of this diagnosis.
    1. Who is at risk? Smoking as this patient does and vitamin D deficiency can increase risk.
    2. Prevention:
    3. Risk for this patient:

5. Education/Counsel on these Diagnosis

a. Guillain-Barre Syndrome: The slowing of nerve conduction can be found by electrodiagnostic

testing to verify demyelination of motor neurons (Kuwabara, 2004). Complete blood counts (CBC, metabolic panels, and electromyography can support this diagnosis. Neurology specialty needs to be consulted and a spinal tap ordered to look for CSF protein (> 0. 55 g/L), magnetic resonance imaging (MRI), and computed tomography (CT) will help to rule out other potential problems. Admission to the hospital is necessary to monitor for respiratory involvement, cardiovascular complications and careful consideration for bowel or bladder incontinence and retention. Potential transfer for aggressive occupational, speech and recreational rehabilitation to avoid prolonged effects the disease need to be discussed. Intravenous immunoglobulin (IVIG), plasma exchange and immunomodulatory treatment, corticosteroids and pain management should be given as options and follow up for these issues scheduled (Andary, 2016).

2. Cauda Equina Syndrome: As a result of this condition the patient may need to be taught how to self-catheterize, use suppositories or perform disimpaction with a bowel protocol in place. Prevention of skin impairment and physical therapy to maintain function and strength is necessary via physical and occupational therapy. Medication management is extremely important, the use of anti-inflammatory, corticosteroid, and pain medications will be prescribed and appropriate

lab work such as CBC, sedimentary rate, fasting blood sugar and possible cerebrospinal fluid evaluation ordered (Dawodu et al, 2018).

3. Multiple Sclerosis: The decrease of flare-ups and the maintenance of symptoms are the focus points if diagnosed with this disease. Medication management to control inflammation and inhibiting the immune response. Drugs available now can help promote remyelination, suppress the B-cell and -cell functions that play a large role in the adaptive immune response.

References

- Andary, M. (2016). Guillian-Barre Syndrome. *Medscape* . Retrieved on 10/11/16 from https://emedicine. medscape. com/article/315632-overview
- Amatya, B., Khan, F., Whishaw, M., & Pallant, J. F. (2013). Guillain-Barré Syndrome: Prevalence and Long-Term Factors Impacting Bladder Function in an Australian Community Cohort. Journal of Clinical Neurology (Seoul, Korea), 9(3), 144–150. http://doi. org/10. 3988/jcn. 2013. 9. 3. 144
- Berg, E. J., & Ashurst, J. V. (2018, September 19). Anatomy, Back, Cauda Equina. Retrieved October 9, 2018, from https://www. ncbi. nlm. nih. gov/books/NBK513251/
- Colloca, L., Ludman, T., Bouhassira, D., Baron, R., Dickenson, A. H., Yarnitsky, D., Raja, S. N. (2017). Neuropathic pain. Nature Reviews. Disease Primers, 3, 17002. http://doi. org/10. 1038/nrdp. 2017. 2
- Dawodu, S., Bechtel, K., Beeson, M., Hodges, S., Humphreys, S., & Kellam, J. (2018, June 14). Cauda Equina and Conus Medullaris

Syndromes: Background, Anatomy, Pathophysiology. Retrieved October 9, 2018, from https://emedicine. medscape. com/article/1148690-overview

- Kuwabara, S. (2004). Guillain-Barré syndrome: Epidemiology, pathophysiology and management. Drugs, 64(6), 597-610. doi: 10. 2165/00003495-200464060-00003
- Misra, U. K., Kalita, J., & Nair, P. P. (2008). Diagnostic approach to peripheral neuropathy. Annals of Indian Academy of Neurology, 11(2), 89–97. http://doi. org/10. 4103/0972-2327. 41875
- Guillain-Barré Syndrome Fact Sheet. (2018, June). Retrieved fromhttps://www. ninds. nih. gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Guillain-Barre-Syndrome-Fact-Sheet
- Walling, A., & Dickson, G. (2013). Guillain-Barré Syndrome. *American Family Physician* , 1(87), 3rd ser., 191-197. Retrieved October 11, 2018, from https://www. aafp. org/afp/2013/0201/p191. html#sec-3.