

Bilateral Lower Extremity Weakness

Hello, Clinical Problem Solvers. My name is Gabriela Pucci, and I'm a Neurology resident from Brazil. Today, I'll guide you to an approach to Bilateral Lower Extremity Weakness.

There's a lot of possible causes for bilateral lower extremity weakness.

The Neurodx is equal to the localization times time course. First, let's try to localize it and then talk about the tempo.

Weakness is a symptom that can indicate injury to one of the various parts of the motor system. The upper motor neurons are situated in the precentral gyrus in the frontal lobe, and their axons go all the way to the corona radiata, internal capsule, anterior brainstem, crossing over at the cervical medullary junction, down to the spinal cord, and finally making a synapse with the lower motor neuron. This one leaves the spinal cord through roots, plexus and forms the peripheral nerve, that controls the muscle. Between them, there's the neuromuscular junction.

We separated our approach into three buckets.

To evaluate a person with bilateral lower extremity weakness, you first have to think in the upper (UMN) and lower motor neurons (LMN) and look for signs of them when examining the patient.

Our professor Aaron Berkowitz has taught us a great way to remember their signs: in the UMN lesion, the signals go "up": hyperreflexia (up or increased reflexes), the big toe goes up when you do the plantar reflex (called Babinski sign), and the tone goes "up" or increased, causing spasticity. In the LMN lesion, the signs are "low": hyporeflexia (or areflexia, reflexes are low), big toes go down in the plantar reflex (normal response), tone goes "low" (decreased tone). Another sign of LMN is fasciculation. This helps us in the localization.

So, if we see UMN signs, we can think there's a disease in the brain or spinal cord.

In the brain, the lesion has to be medial or parasagittal to cause bilateral lower extremity weakness. If you check the homunculus- motor, you will see that the legs are in the parasagittal brain.

Now that we localized the injury in the brain, we have to look for the other important factor in our equation: the time course or tempo.

Usually, in Neurology, when we think about sudden onset, we have to think about vascular diseases. The sudden onset is resultant of an abrupt blockage of the blood to a specific brain area. So, with a sudden onset of bilateral lower extremity weakness usually associated with cognitive change and bladder incontinence, we have to think in a **bilateral anterior cerebral artery stroke**, which is quite rare, but it can happen. If subacute or chronic, you have to think about a **parasagittal meningioma**.

On the other hand, if the patient has UMN signs and usually sensory change, you can sometimes see a sensory level that is even more favorable of a spinal cord disease.

Sudden causes are **vascular** (like, for example, the anterior spinal cord syndrome caused by an occlusion in the anterior spinal artery) and **disc and vertebral collapse**. The last usually is accompanied by back pain. Acute causes **include epidural abscess** (that usually will have back pain and fever) and **transverse myelitis** (sensory or motor symptoms

associated with autonomic dysfunction, a clear defined sensory level, and inflammation in the CSF). It's also important to remember that transverse myelitis can be a presentation of multiple sclerosis, so asking for previous acute neurological symptoms that were reversible spontaneously can help with this diagnosis. Subacute causes of bilateral lower extremity weakness associated with UMN signs in the spinal cord are **spondylosis (arthritis of the spine) or structural**, which compresses the spinal cord causing the symptoms, and **neoplasms** (first cause are metastasis, which are much more common than primary bone tumors). You also have to think about 2 deficiencies here: **deficiency of B12** (called subacute combined degeneration when it affects the spinal cord, causing weakness, paresthesia, and sensory ataxia) and **deficiency of copper** (similar symptoms to subacute combined deficiency of B12). **Tuberculosis** can also produce myelopathy and usually courses with the classical signs of fever, back pain, and weight loss. This infection can directly cause a tuberculoma within the spinal cord, or Pott's disease when the bacteria infects the vertebral body.

Dural arteriovenous fistula can present with some fluctuations of the symptoms and evolve with sensory disturbances and sphincter disturbances.

The last part of the spinal cord causes of bilateral leg weakness is the chronic causes. You can also find **spondylosis and structural causes** (which can also present subacute, as previously explained). Other important causes are **hereditary** - most commonly hereditary spastic paraplegia. And the pearl here is that most of the patients have a familiar positive history of a similar presentation. **Radiation** can also induce myelopathy by damaging the white matter of the spinal cord. If presented earlier, it can be reversible (especially before 6 months), but it can be persistent if presented later than 6 months. Infections can lead to chronic myelopathy, as well. The most important are **AIDS** (that can be associated with dementia as well) and **HTLV1** (also called tropical spastic paraparesis, usually not common in the US and in Europe, but more common in Japan, Middle East, Africa, South America and the Caribbean, Melanesian Islands and Papua New Guinea).

Ok, so now we finished the diseases that typically present with UMN signs. Let's talk about LMN signs.

LMN signs help us localize especially in the roots and nerves. The roots of cauda equina, for example, can be damaged and cause cauda equina syndrome (bilateral leg weakness associated with saddle anesthesia and bowel/bladder dysfunction). Other factors that indicate root injury are **compressive** (tumor, for example, or acute disk herniation) and inflammatory causes like **sarcoidosis**. Infections can also cause cauda equina syndrome, and two examples are **HSV2 and TB**. HSV2 causes the Elsberg syndrome - which is defined as cauda equina syndrome plus myelitis due to infection of Herpes Simplex 2 Virus. **Tuberculosis** can cause both UMN as we saw earlier, but the bacteria can also infect the neural roots causing cauda equina syndrome as well. **Lymphoma** is the most common cause of malignancy related to this. Injury to nerves also causes LMN signs. The most important is **Guillain-Barre syndrome**, an acute cause that classically presents with ascending paralysis beginning in the legs and can cause even quadriplegia and respiratory failure. The paraparetic variant is rare. Even in this variant, subtle arm abnormalities are common (such as absent reflex despite no muscle weakness or altered electrodiagnostic studies).

We talked about 2 buckets that can cause bilateral lower leg weakness, the UMN and LMN signs. Last, if we don't see UMN or LMN signs, we can think about **muscle injury**. Usually, this can cause proximal paralysis and preserved reflexes (unless severe myopathy is present).

To summarize, another pearl that we can use is the presence of **bowel and bladder symptoms**. In spinal cord and cauda equina injury, they are usually involved. In brain injury, the bladder may be involved, and in nerve and muscle injuries, they are usually not involved.

We hope you enjoyed the schema. Until next time, Clinical Problem Solvers!