Learning In 10: Subacute Combined Degeneration of the Spinal Cord
Neurology
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Slide 1:
This is a presentation on subacute combined degeneration of the spinal cord.

Slide 2:
At the end of this presentation, one should have a broad perspective of subacute combined degeneration of the spinal cord, in particular to know about its etiology, clinical presentation, and clinical cause. One should be aware of the workup and differential diagnosis that should be considered. It is important to learn about and be able to recognize this condition as it is one of the few neurological conditions that are ameliorated with early and appropriate treatment.

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Subacute combined degeneration of the spinal cord as its name implies is progressive degeneration of the spinal cord due to B12 deficiency. This disorder affects about 1 in 10,000 people and is more common in individuals older than 40.

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Vitamin B12 deficiency can be due to impaired absorption and dietary insufficiency. The most common cause for impaired absorption is pernicious anemia and autoimmune atrophic gastritis, leading to achlorhydria and limitation of intrinsic factor secretion.

Surgical procedures, such as gastrectomy and ileal resection, are also causes for vitamin B12 deficiency. Other causes of impaired vitamin B12 absorption include bacterial overgrowth, tapeworm, and tropical sprue. Vitamin B12 is particularly concentrated in some foods, such as meat products, tuna, and liver. As a result, vegans are at high risk for vitamin B12 deficiency.

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Patients with vitamin B12 deficiency present with combination of peripheral and central nervous system and hematological problems. Not all are present in each patient. The common symptoms are incoordination or unsteadiness, weakness, numbness, and fatigue. Neurological symptoms commonly develop prior to hematological abnormalities.

Vitamin B12 deficiency typically leads to degeneration of a dorsal column, resulting in loss of joint position and vibration. It can cause corticospinal tract degeneration, resulting in progressive weakness. The classical triad signs of subacute combined degeneration of the spinal cord are extensor plantar response, absent ankle, and brisk knee jerks due to a combination of upper and lower motor neuron signs. The extensor plantar response and brisk knee jerks are caused by spinal cord involvement or the absence of ankle jerk is secondary to peripheral neuropathy.

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During physical examination, do not forget to look up for features of B12 deficiency, such as visual loss from optic neuropathy and pallor due to anemia. Glossitis and stomatitis might be present as well. The patient may also have confusion due to cerebral involvement.
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Vitamin B12 deficiency can be documented if low serum B12 level. Measurement of methylmalonic acid and homocysteine provides even greater diagnostic sensitivity as compared to serum B12 levels. Peripheral blood film may show megaloblasts and hypersegmented polymorphs. As pernicious anemia is one of the most common causes of B12 deficiency, investigations for this condition should be done in patients with vitamin B12 deficiency. Parietal cells antibodies are found in 90% of patients with pernicious anemia. Intrinsic factor antibodies are more specific for pernicious anemia, but have lower sensitivity. The Schilling test is used to determine whether the body is able to absorb vitamin B12 normally.

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Imaging with magnetic resonance imaging may show abnormalities on T2 weighted imaging consistency with the lesion. It typically affects the cervical and thoracic spinal cord with T2 signal mainly seen in dorsal column, lateral, and ventral white matter. These lesions may improve with treatment.

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Differential diagnosis that we should consider for subacute combined degeneration are diseases that can give rise to both upper motor neuron and lower motor neuron signs and conditions that produce multifocal abnormalities. Multiple sclerosis can present with a combination of cerebellar and corticospinal dysfunction. Cognitive dysfunction can develop, although this is usually with advanced disease.

Polyneuropathy is not expected. Vasculitis can produce multifocal central and peripheral findings and should be considered as a differential diagnosis. Differentiation is on laboratory results and MRI appearance. Biopsy is often needed to meet the definitive diagnosis of vasculitis.

Tabes dorsalis is another differential diagnosis. It typically affects the dorsal column and thus can mimic subacute combined degeneration. However, tabes dorsalis may give rise to additional signs, such as the Argyll Robertson pupil.

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B12 replacement and treatment of the underlying cause of vitamin B12 deficiency are key in managing subacute combined degeneration. Most patients show some improvement in the symptoms and signs of each B12 deficiency with replacement. Peripheral neuropathy usually improves within 3 to 6 months. However, the deficits due to subacute combined degeneration of the spinal cord show minimal response to vitamin B12 replacement. Importantly, if left untreated, the disorder of subacute combined degeneration can progress further, worsening symptoms, signs, and disability.

Slide 11:
Thank you for listening. We hope you have enjoyed learning about subacute combined degeneration of the spinal cord through this presentation.