signifcantly in developed countries with widespread use of antibiotics. However, its persistence in certain populations and its potential resurgence in the context of evolving antibiotic resistance warrant continued vigilance

Treponema pallidum, a spirochete capable of invading the human body

In od c ion

Neurosyphilis, an insidious complication of untreated syphilis infection, represents a poignant intersection of infectious disease and neurology. Historically known as the "great imitator," syphilis has intrigued and perplexed clinicians for centuries with its diverse and often unpredictable manifestations. Among these, neurosyphilis stands out as a profound example of the disease's ability to invade and disrupt the central nervous system (CNS) [1].

Syphilis itself is caused by the spirochete bacterium *T, e, , nema , allid m*, initially entering the body through mucous membranes or breaks in the skin. If left untreated, the infection progresses through distinct stages: primary and secondary lesions, followed by a latent phase [2]. In some individuals, particularly if untreated for years or decades, *T. , allid m* can disseminate throughout the body, including the CNS, leading to neurosyphilis.

The pathogenesis of neurosyphilis involves *T. , allid m* crossing the blood-brain barrier and infecting the meninges, brain parenchyma, spinal cord, and cranial nerves [3]. This invasion triggers a spectrum of neurological disorders, ranging from asymptomatic forms to severe and debilitating conditions affecting cognition, motor function, and sensory perception [4].

Clinically, neurosyphilis can present with diverse symptoms, including meningitis, stroke-like episodes, cognitive impairment resembling dementia, and sensory deficits such as ataxia and lancinating pains [5]. The variability and often delayed onset of these symptoms complicate diagnosis, necessitating a high index of suspicion in at-risk populations [6].

Me hodolog

The clinical presentation of neurosyphilis is diverse and can mimic various neurological disorders, making diagnosis challenging. Common manifestations include:

Meningi i : Headache, neck stiffness, and cranial nerve palsies.

Meningo a c la phili : Stroke-like symptoms due to inflammation and damage to blood vessels supplying the brain.

Gene al pa e i : Cognitive impairment, personality changes, and psychiatric symptoms resembling dementia.

Tabe do ali : Damage to the dorsal columns of the spinal cord, leading to sensory ataxia, lancinating pains, and bladder dysfunction.

These manifestations can occur years to decades after the initial syphilitic infection, highlighting the chronic and progressive nature of untreated neurosyphilis.

Diagnosing neurosyphilis relies on a combination of clinical suspicion, serological tests, and CSF analysis:

Serological tests include non-treponemal (e.g., RPR, VDRL) and treponemal (e.g., FTA-ABS, TP-PA) tests, which detect antibodies against *T. , allid m*.

Cerebrospinal fluid (CSF) analysis shows lymphocytic pleocytosis, elevated protein levels, and positive treponemal tests (e.g., TPPA) in the CSF.

Neuroimaging, such as MRI or CT scans, may reveal abnormalities suggestive of neurosyphilis, although findings can be nonspecific.

T ea men and managemen

The cornerstone of neurosyphilis management is antibiotic therapy. Penicillin remains the treatment of choice, with various regimens depending on the stage and severity of the disease. For

Advancements in medical understanding and treatment have transformed the prognosis of neurosyphilis, particularly with the advent of antibiotics such as penicillin. Early detection and prompt treatment can halt disease progression and, in some cases, reverse neurological deficits. However, challenges such as antibiotic resistance and the intersection with HIV co-infection necessitate ongoing vigilance and adaptation in clinical management.

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