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Opinion Article

Neurological Complications of Atrial Myxoma Masquerading as Lyme Disease

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DESCRIPTION

Neurological manifestations are among the most alarming and clinically significant complications that can arise in patients with atrial myxoma, yet in many instances these presentations are mistaken for other systemic conditions such as Lyme disease. This overlap in symptomatology poses a serious challenge for clinicians and creates a dangerous window where patients may be subjected to inappropriate investigations, ineffective treatments, and prolonged uncertainty. The misdiagnosis of atrial myxoma as Lyme disease is more than an academic curiosity; it represents a tangible risk to neurological health and long-term quality of life that demands urgent recognition in clinical practice.

Atrial myxomas, despite being the most common primary cardiac tumor in adults, remain exceedingly rare compared to the burden of infectious diseases. Their rarity makes them inherently less likely to be considered early in the diagnostic process, particularly in patients who present with nonspecific neurological complaints such as dizziness, confusion, headaches, or transient ischemic-like symptoms. Lyme disease has gained significant public attention over the past decades, especially in endemic regions, and is often invoked as a possible explanation when patients present with unexplained neurological symptoms including cognitive slowing, neuropathic pain, facial palsy or migratory paresthesias. This discrepancy in prevalence and public awareness tilts the clinical mindset toward infectious explanations and away from rare cardiac tumors, even when the pattern of neurological dysfunction may be atypical for Lyme disease.

The neurological manifestations of atrial myxoma are rooted in embolic phenomena and altered cerebral perfusion, which differ fundamentally from the inflammatory and infectious mechanisms seen in Lyme disease. Fragments of the friable tumor can embolize into the cerebral circulation, leading to ischemic strokes, transient ischemic attacks, or multifocal infarcts that produce diverse neurological deficits. Patients may present with sudden weakness, aphasia, visual disturbances, or seizures, symptoms that are acute and episodic in nature. In contrast, neurological Lyme disease tends to evolve more insidiously, with chronic meningoencephalitis like features, radiculopathies, or cranial neuropathies. Despite this distinction, clinical overlap exists, and the lack of immediate imaging or cardiac assessment often results in patients with atrial myxoma being misclassified as having an infectious etiology. The tragic irony is that while Lyme disease responds to antimicrobial therapy, atrial myxoma requires surgical removal, and every delay increases the risk of further embolic complications.

The cognitive and psychological toll of such misdiagnosis cannot be ignored. Patients experiencing neurological decline often cling to the hope that antimicrobial therapy will restore function, only to find themselves trapped in a cycle of ongoing fatigue, brain fog, or worsening deficits. When the eventual diagnosis of atrial myxoma emerges, it is often met with both relief and resentment relief that an answer has been found, and resentment that the answer was delayed by months of misdirection. From a rehabilitative perspective, each additional embolic event worsens long-term prognosis, leaving patients with preventable deficits such as hemiparesis, cognitive impairment, or language dysfunction. Thus, the cost of misdiagnosis extends beyond delayed surgery into permanent disability and diminished quality of life.

Neurologists may be inclined to pursue infectious or autoimmune etiologies for unexplained neurological findings, while cardiologists are rarely brought into the diagnostic loop until cardiac symptoms such as palpitations, murmurs or syncope arise. Yet in atrial myxoma, neurological manifestations may precede overt cardiac signs, leaving patients in a diagnostic limbo. This fragmented approach to patient care underscores the need for more integrated evaluation, where neurological presentations with unexplained embolic or multifocal features trigger cardiac imaging as part of standard workup.

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