CLINICAL VIGNETTE

Bradycardia and Hypothermia in a Patient with Dementia

Marie Adachi, MD and Erin A. Cook, MD

Case

A 92-year-old female with dementia with Lewy bodies (DLB), frequent falls, and hypothyroidism presented to the Emergency Room from home with increased agitation and hallucinations. At baseline, the patient had a one-year history of fluctuating mental status. She would alternate between periods of agitation and confusion, including times when she pounded on the door of her home to get out as she did not believe it was her real home, and periods of lethargy, during which she would sleep most of the day. Her family had also noticed frequent hallucinations during which she conversed with people who were not there. These symptoms had worsened over the last several weeks.

The evaluation for behavioral symptoms included a WBC of 10 x10E3/uL, BUN 46 mg/dL, Creatinine 2.7 mg/dL (baseline 0.7-0.9 mg/dL). Chest x-ray did not show any evidence of pneumonia. Urinalysis revealed >1000 WBCs/uL, 349 RBCs/uL, WBC clumps, pH 5.1, 1+ protein, 2+ leukocyte esterase, and positive nitrite. She was started on IV ceftriaxone for presumed urinary tract infection and IV fluids for her acute kidney injury. Her home quetiapine dose of 50mg twice a day was held. Her agitation gradually improved with treatment of the infection, requiring only 12.5mg of quetiapine 1-2 times a day, but she continued to have fluctuations in cognition. The urine culture eventually revealed >100,000 CFU/mL of both Pseudomonas and Enterococcus, and based on the sensitivity data, she was transitioned to oral ciprofloxacin to complete a seven-day course of antibiotics for her urinary tract infection. At that time, the patient appeared stable for discharge, and plans were made to transition her to a skilled nursing facility for rehabilitation for her deconditioning.

However, on hospital day seven, she developed new bradycardia to the 40s, hypothermia with axillary temperature of 92.8F and rectal temperature of 94.3F, and somnolence with respiratory rate of 11. Given concern for sepsis, a repeat infectious workup was done, but the results were unrevealing and her symptoms and vital sign abnormalities resolved without further intervention. Her family stated that there were similar episodes in the past that also resolved spontaneously. She was monitored for an additional 24 hours without any new symptoms or abnormalities in her vital signs and was discharged to a skilled nursing facility for rehabilitation.

Three days after discharge, the patient presented again to the Emergency Room from the skilled nursing facility for a recurrent episode of somnolence and hypothermia with axillary temperature of 93.4F. Repeat infectious workup was significant for a consolidation on chest x-ray, most likely due to aspiration. She was initially treated with IV ampicillin/sulbactam, and eventually transitioned to oral amoxicillin/clavulanic acid to complete a five-day course of treatment. During that admission, it was also discovered that the patient had stopped taking her levothyroxine pills for the past month given difficulty with swallowing pills. TSH at the time of readmission was 22.6 mcIU/mL (normal 0.3-4.7 mcIU/mL), with free T4 of 0.7 ng/dL (normal 0.8-1.6 ng/dL) and free T3 of 190 pg/dL (normal 222-383 pg/dL). Endocrinology was consulted for possible myxedema coma, but upon further chart review, the patient was noted to have similar episodes in the past with a normal TSH, and her current degree of TSH elevation was not high enough to be consistent with the diagnosis. She was resumed on her home dose of oral levothyroxine 50mcg daily. She was also noted during the admission to have heart rates as low as 39. The hypothermia, bradycardia, and somnolence resolved with the treatment of the pneumonia and hypothyroidism. Ultimately, it was concluded that these symptoms were likely driven by underlying autonomic dysfunction from her dementia with Lewy Bodies that were exacerbated by infection and hypothyroidism. She was discharged to a skilled nursing facility for rehabilitation, after which she was able to return home with her family.

Discussion

While Alzheimer's Dementia is by far the most common type of degenerative dementia, dementia with Lewy Bodies (DLB) accounts for about 5% of all dementia cases.[1] DLB is characterized by a progressive decline in cognitive function along with fluctuations in cognition, hallucinations, REM sleep disorders, and parkinsonism. Consensus criteria for the diagnosis of DLB have been developed to aid in the diagnosis of this disease,[2] and the core clinical features are summarized in Table 1. A clinical diagnosis of probable DLB is made if two or more core clinical features of DLB are present.

Proper diagnosis of DLB is key as there are unique strategies in management. For example, DLB patients often respond well to cholinesterase inhibitors such as donepezil or rivastigmine,[3] but are more sensitive to the side effects of neuroleptic medications such as haloperidol.[4] It is important to provide guidance to the

families and caregivers of patients with DLB as symptoms may become more prominent with progression of the disease.

Making the proper diagnosis can be challenging as there may be significant overlap in the presentation between DLB and other neurodegenerative disorders, such as Alzheimer's dementia and Parkinson's dementia. Like Alzheimer's dementia, the most common initial presentation of DLB is cognitive dysfunction. However, unlike Alzheimer's dementia, which often presents with early short-term memory loss, DLB presents with early impairments in visuospatial impairment.[5] In a retrospective study of patients with autopsy-proven dementia with Lewy Bodies or Alzheimer's dementia, visual hallucinations at the time of initial presentation has a specificity of 99% for DLB.[6] DLB can also present with motor symptoms of parkinsonism, creating overlap in symptoms with Parkinson's dementia. The key differentiating feature between Parkinson's dementia and DLB is that the motor symptoms of parkinsonism precede the cognitive symptoms by at least one year in Parkinson's disease. In contrast, in DLB, the motor and cognitive symptoms usually occur within one year of each other.

Even with the proper diagnosis, management of DLB can also pose a challenge as features of DLB can mimic other acute medical conditions, such as seizure disorders, TIAs, sepsis, delirium from another underlying medical causes, and side effects of medications (i.e. antispychotics). In this patient, there was significant overlap in the symptoms and vital sign abnormalities caused by her infections and hypothyroidism, and those caused by autonomic dysfunction from her underlying dementia.

Table 1. Core Clinical Features and Supportive Clinical Features from the Revised Criteria for the Clinical Diagnosis of Dementia with Lewy Bodies (summarized from [2])

Core Clinical Features	Supportive Clinical Features
• Fluctuating cognition with pronounced variations in attention and alertness • Recurrent visual hallucinations that are typically well formed and detailed • REM sleep behavior disorder, which may precede cognitive decline • One or more spontaneous cardinal features of parkinsonism (bradykinesia, rest tremor, rigidity)	• Severe sensitivity to antipsychotic agents • Postural instability • Repeated falls • Syncope or other transient episodes of unresponsiveness • Severe autonomic dysfunction • Hypersomnia • Hyposmia • Hallucinations in other modalities • Systematized delusions • Apathy, anxiety, and depression

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