Section 1 **Neurocognitive Disorders**

Case 1

"I've Fallen … and I Can't Get Up"

Sundowner Syndrome

Mr. S is an 80-year-old gentleman with a significant medical history of a kidney transplant, diabetes, hypertension, hyperlipidemia, gastroesophageal reflux disease (GERD), atrial fibrillation, obstructive sleep apnea, and hypothyroidism. He was living at home with his wife when he had a nighttime fall with loss of consciousness. The fall resulted in a lumbar spinal burst fracture that required spinal fusion surgery. His post-op course was complicated by delirium in the intensive care unit (ICU). He would become disoriented and agitated and throw himself from his bed to the floor. He was referred to geriatric psychiatry for consultation due to concerns about his agitated behavior since his arrival at hospital two weeks prior. He had poor nutritional intake with a 30-pound weight loss. This led to a PICC line placement for total parenteral nutrition (TPN). Before discharge TPN was stopped and Mr. S had a G-tube placed for nutritional support. He was in acute rehab for nine weeks before being transferred to long-term care.

The family reported that the patient was often irritable. He had increased confusion and agitation in the evening and continued to occasionally throw himself out of bed. He had had several urinary tract infections (UTIs) over the three months prior to long-term care.

The nursing staff informed his medical provider that the patient would yell at anyone involved in his care, including physical therapists and nurses. They said he would purposely throw himself out of his wheelchair or bed when his requests were not responded to promptly. He would not engage in social activities at the facility and complained of poor energy and always feeling tired.

Mr. S had no significant psychiatric history and had never been under the care of a psychiatrist before. His sleep was described as poor. He would sleep throughout the late morning and early afternoon and not sleep well at night. He still seemed to enjoy watching baseball games at times.

His mental status exam described his mood as sad, with poor eye contact and a mostly logical and linear thought process. He was also described as irritable during portions of the interview and cooperative at other times. He was oriented to person but was not oriented to place or time. He was unable to identify his wife's sister or name his two children.

The medical provider recommended routine bloodwork and a repeat urinalysis to check for potentially reversible causes of subtle delirium. He was put on trazodone 50 mg qhs for sleep and 25 mg twice daily as needed for anxiety or agitation.

At the time of follow-up two weeks later, Mr. S was found to be mostly unchanged. He continued with regular displays of agitation and aggression toward care staff and family members that seemed to become more pronounced in the late afternoon and early evening hours. His appetite continued to be poor and his mood was mostly described as

irritable. He slept a bit better at night but would wake up in the early morning hours asking to get out of bed into his wheelchair, at which time he would be brought close to the nurse's station where he would often yell at staff.

The provider started the patient on sertraline 25 mg daily, which was soon increased to 50 mg daily. The physician recommended that the patient be brought out of his room and encouraged to participate in activities during the daytime instead of being allowed to stay in his room and sleep. Care staff were encouraged to bring Mr. S to the dining room for meals instead of allowing him to take his meals in his room. This was to encourage more socialization, combat boredom, and encourage the normalization of day/night cycles. Melatonin 5 mg qhs was also started.

At a four-week follow-up visit, Mr. S was found to be doing somewhat better. His mood was described as less irritable and he was more cooperative with care. His nutritional intake improved, and his weight loss started to level off. It was noted that care staff were taking the patient outdoors when the weather was good and he seemed to enjoy this. His family was pleased with his improvements in neuropsychiatric symptoms and encouraged by his increasing willingness to participate in therapies, with the goal of Mr. S being able to participate adequately enough in his care to potentially move back home with his family.

Teaching Points

"Sundowner syndrome" refers to a constellation of neuropsychiatric symptoms, usually accompanied by agitation that occurs late in the day in a patient with a major neurocognitive disorder (MNCD). In addition to agitation, anxiety, aggressiveness, increased confusion, and even visual or auditory hallucinations are common. Second only to wandering, sundowning is reported to be one of the most common disruptive behaviors in long-term care settings [1]. Symptoms usually emerge later in the day, classically in the late afternoon or early evening at the time of sunset. This neuropsychiatric syndrome can be very challenging for caregivers. Sundowning is a common reason for those with MNCDs to be moved from home to a long-term care facility. It is also one of the more common reasons for consultation with a geriatric psychiatrist in the long-term care setting.

Sundowning and delirium share some similarities with regard to underlying causes. Both are strongly associated with MNCDs. One key difference between sundowning and delirium is the time of day in which symptoms occur. Sundowning typically occurs and recurs in the late afternoon or evening hours whereas delirium can occur at any time of the day or night.

The treatment of delirium focuses on the identification and treatment of underlying medical issues or medications that may be contributing. In contrast, the treatment of sundowning focuses on altering environmental factors that can contribute to the condition.

The pathophysiology of sundowner syndrome is not fully understood, but there are several theories about the underlying mechanisms. These theories are not mutually exclusive.

One theory is that sundowning may be related to changes in the body's internal clock or circadian rhythm regulation. These rhythms are regulated by the suprachiasmatic nucleus (SCN), which responds to light and dark stimuli to help regulate sleep–wake cycles and other physiological processes. In individuals with dementia or other cognitive

impairments, the SCN may be disrupted, leading to changes in sleep patterns and other circadian rhythms. This disruption may contribute to the development of sundowning symptoms in the late afternoon or evening.

Another theory is that sundowning may be related to pain or fatigue that becomes more pronounced in the late afternoon or evening. Individuals with dementia or other cognitive impairments may have difficulty communicating their discomfort, leading to increased agitation or confusion as a way of expressing their discomfort.

There is also evidence to suggest that sundowning may be related to changes in neurotransmitter levels, particularly concerning the neurotransmitter acetylcholine. Individuals with Alzheimer's disease or other forms of MNCD have a loss of acetylcholine-producing neurons in the brain, which contributes to changes in cognitive function and behavior [1].

Sensory deprivation can also play a role in sundowner syndrome. Seniors in long-term care are often lacking in stimulating daytime activities. A multidisciplinary approach to creating individualized care plans for seniors with MNCD is important. These care plans should allow seniors to have regular social interactions, physical activity, family-style mealtimes, time outdoors, and religious activities.

The treatment of sundowning depends on the acuity of symptoms. All patients should have a thorough examination of environmental factors that could be contributing to late-day confusion and agitation and changes made to improve those environmental factors. Medications that could be contributing to confusion should also be identified and removed.

At the time of this writing, there has been relatively recent approval from the US Food and Drug Administration (FDA) of the atypical antipsychotic brexpiprazole for treating agitation due to Alzheimer's disease[2]. There are several other pharmacologic options for the treatment of agitation in the setting of MNCDs. For mild symptoms, several studies have demonstrated improved outcomes for those with sundowner syndrome with melatonin [1]. Melatonin levels are deficient in those with MNCDs (especially Alzheimer's disease). Melatonin is generally well tolerated and may be worth trying in those with mild symptoms. In those patients with more severe symptoms, including severe agitation or psychosis, a trial of a second- or third-generation antipsychotic may be warranted [1]. Antipsychotics are used off-label and should be used with caution and in the smallest dose for the shortest possible duration. Regular efforts to decrease the dosage of antipsychotics are also needed once symptoms are controlled. Cholinesterase inhibitors such as donepezil are known to improve behavioral disturbances in patients with dementia but there are conflicting data on the effect of cholinesterase inhibitors on sundowner syndrome [1].

Take-Home Points

- Sundowner syndrome refers to a constellation of neuropsychiatric symptoms, usually accompanied by agitation that occurs late in the day in a patient with an MNCD. In addition to agitation, anxiety, aggressiveness, increased confusion, and even visual or auditory hallucinations are common.
- Mild symptoms may respond well to melatonin 5 mg in the late afternoon. In those patients with more severe symptoms, including severe agitation or psychosis, a trial of a second- or third-generation antipsychotic may be warranted.
- All patients should have a thorough examination of environmental factors that could be contributing to the late-day confusion and agitation and changes made to improve those environmental factors. Medications that could be contributing to confusion should also be identified and removed.

– Antipsychotics are used off-label and should be used with caution and in the smallest dose for the shortest possible duration. Regular efforts to decrease the dosage of antipsychotics are also needed once symptoms are controlled.

References

1. Canevelli, M., Valletta, M., Trebbastoni, A., Sarli, G., Tariciotti, L., & Bruno, G. (2015). Sundowning in dementia: Clinical relevance, pathophysiological determinants, and therapeutic approaches. *Frontiers in Medicine*, 3. https://doi.org/10.3389/fmed.2016.00073
2. Grossberg, G. T., Kohegyi, E., Mergel, V., Josiassen, M. K., Meulien, D., Hobart, M., Slomkowski, M., Baker, R. A., McQuade, R. D., & Cummings, J. L. (2020). Efficacy and safety of brexpiprazole for the treatment of agitation in Alzheimer's dementia: Two 12-week, randomized, double-blind, placebo-controlled trials. *The American Journal of Geriatric Psychiatry*, 28 (4), 383–400.

Further Reading

Ahmad, F., Sachdeva, P., Sarkar, J., & Izhaar, R. (2022). Circadian dysfunction and Alzheimer's disease: An updated review. *Aging Medicine*, 6 (1), 71–81. doi: 10.1002/agm2.12221. PMID: 36911088; PMCID: PMC10000289.

Carrarini, C., Russo, M., Dono, F., Barbone, F., Rispoli, M. G., Ferri, L., Pietro, M. D., Digiovanni, A., Ajdinaj, P., Speranza, R., Granzotto, A., Frazzini, V., Thomas, A., Pilotto, A., Padovani, A., Onofrj, M., Sensi, S. L., & Bonanni, L. (2020). Agitation and dementia: Prevention and treatment strategies in acute and chronic conditions. *Frontiers in Neurology*, 12. https://doi.org/10.3389/fneur.2021.644317

Desai, A., & Grossberg, G. (2018). *Psychiatric Consultation in Long-Term Care. A Guide for Healthcare Professionals.* 2nd ed. (Cambridge University Press.)

Guu, W., & Aarsland, D. (2022). Light, sleep–wake rhythm, and behavioural and psychological symptoms of dementia in care home patients: Revisiting the sundowning syndrome. *International Journal of Geriatric Psychiatry*, 37 (5). https://doi.org/10.1002/gps.5712

Lin, L., Huang, Q. X., Yang, S. S., Chu, J., Wang, J. Z., & Tian, Q. (2013). Melatonin in Alzheimer's disease. *International Journal of Molecular Sciences*, 14 (7), 14575–14593. doi: 10.3390/ijms140714575. PMID: 23857055; PMCID: PMC3742260.

Menegardo, C. S., Friggi, F. A., Scardini, J. B., Rossi, T. S., Vieira, S., Tieppo, A., & Morelato, R. L. (2019). Sundown syndrome in patients with Alzheimer's disease dementia. *Dementia & Neuropsychologia*, 13 (4), 469–474. https://doi.org/10.1590/1980-57642018dn13–040015

Sharma, A., Sethi, G., Tambuwala, M. M. A., Aljabali, A. A., Chellappan, D. K., Dua, K., & Goyal, R. (2021). Circadian rhythm disruption and Alzheimer's disease: The dynamics of a vicious cycle. *Current Neuropharmacology*, 19 (2), 248–264. https://doi.org/10.2174/1570159X18666200429013041

Todd, W. D. (2019). Potential pathways for circadian dysfunction and sundowning-related behavioral aggression in Alzheimer's disease and related dementias. *Frontiers in Neuroscience*, 14. https://doi.org/10.3389/fnins.2020.00910

Section 1 **Neurocognitive Disorders**

Case 2

"I Think I Hit My Head"

MNCD Secondary to Alcohol Use Disorder

Mr. T is a 62-year-old gentleman recently admitted to long-term care after a two-week stay in acute rehab. This stay in acute rehab followed a week-long hospital stay. Mr. T presented to the hospital after being found on the floor of his home by a concerned neighbor who had not seen him on his front porch for two days. Mr. T was well known to be found on his front porch each afternoon and evening either yelling or waving at passersby.

Mr. T's neighbor let herself into the unlocked home and he was found on the floor, not able to put more than one or two words together other than, "I think I hit my head." He was at the base of a staircase with a large hematoma and dried blood on his scalp. The concerned neighbor called 911 and paramedics brought Mr. T to the local emergency department. Medical workup was significant for dehydration and mild renal failure. Blood counts showed mild anemia with mild thrombocytopenia and macrocytosis. A head CT showed a fresh subdural hematoma over the left parietal area and evidence of another more chronic subdural hematoma overlying the right frontal lobe. The head CT also showed some mild to moderate cerebral atrophy with enlarged ventricles and prominent widening of cerebral sulci.

Mr. T was admitted to hospital. Neurosurgery did an urgent craniotomy with evacuation of the subdural hematoma and he was treated in the ICU for three to four days. Mr. T's alertness was described as varying between stuporous and lethargic and at other times restless and agitated. His mental status did not improve despite the treatment for dehydration. A repeat head CT showed no unexpected findings or recurrence of the intracerebral bleeding.

On day three in hospital the ICU physician and social services successfully tracked down the patient's daughter, who lived several states away. The patient's daughter said she had not had much contact with her father in the years following the death of her mother. She said she was never very close to her father as he was often verbally and sometimes physically abusive to both her and her mother growing up. He was described as a heavy drinker.

With this new knowledge, the hospital team started treatment for alcohol withdrawal-related delirium with regularly scheduled IV lorazepam. He became calmer and gradually his alertness and orientation improved. He was moved out of the ICU. Physical therapy and occupational therapy evaluations showed that Mr. T had significant problems in multiple areas of mobility and self-care, and he was sent from the hospital to acute rehab.

The patient's physician in acute rehab found Mr. T to be generally pleasant but often confused. His therapy was slowed by difficulty following more complex instructions and

retaining knowledge from one therapy session to another. Mr. T scored 18 out of 30 on his Saint Louis University mental status (SLUMS) exam. He was also found to have significant peripheral neuropathy in his feet and legs. Over the course of the next several weeks, it became clear that Mr. T was not going to be able to return to an independent living situation. Mr. T's daughter assisted social services in making arrangements for long-term care.

Mr. T's long-term care provider felt that Mr. T's MNCD was related to chronic alcohol use disorder (AUD).

Teaching Points

The majority of cases of MNCDs can be classified as related to Alzheimer's disease, vascular mechanisms, or Lewy Body dementia. Major neurocognitive disorders related to chronic alcohol use may be underrecognized as there are no definitive diagnostic criteria. The prevalence of the condition varies largely depending on the demographics of the population being studied, as alcohol consumption patterns vary based on social and ethnic patterns of alcohol use. It is estimated that 10–24% of those who have chronic AUD will develop dementia [1]. Studies have shown that alcohol-related dementia (ARD) is responsible for about 10% of early-onset dementia and about 1% of late-onset MNCDs [2].

The pathophysiology of ARD is complex and at present not well understood. Suggested factors likely to be involved include direct neurotoxicity of alcohol, chronic oxidative stress, neuroinflammation, vitamin deficiencies, alcohol-related impacts on neurotransmitter pathways, and vascular effects [3]. See Box 2.1.

There are psychosocial comorbidities to AUD that likely influence the development of dementia at a younger age than in non-alcohol-abusing cohorts. Those with AUD often experience social isolation, financial difficulties, relationship problems, and

Box 2.1 Suggested mechanisms influencing the development of MNCD in chronic AUD

1. Direct neurotoxicity of alcohol. Alcohol can disrupt cell membrane integrity and alter ionic balance within neurons. This can lead to cell damage or death [3].
2. Oxidative stress related to alcohol. The metabolism of alcohol creates reactive oxygen species as byproducts such as superoxide radicals and hydrogen peroxide. Heavy production of these substances can overwhelm antioxidant defense mechanisms and cause oxidative damage to cell membranes, intracellular proteins, and DNA.
3. Alcohol-related neuroinflammation. Alcohol can activate immune cells in the central nervous system (CNS) and lead to the release of pro-inflammatory cytokines, which can cause damage to cells within the CNS [4].
4. Vitamin deficiencies. Chronic heavy alcohol use can lead to deficiencies of thiamine, folate, and pyridoxine. These deficiencies can lead to dysfunctional cellular energy metabolism, impaired DNA synthesis and repair, and neurotransmitter synthesis.
5. Alcohol-related effects on neurotransmitter pathways. Alcohol negatively alters some key neurotransmitter systems.
6. Central nervous system vascular dysfunction related to chronic alcohol use. Chronically high levels of alcohol use can cause disruptions of the blood–brain barrier, cerebral blood flow irregularities, endothelial dysfunction, and hypertension-related small vessel disease of the brain [5].

Box 2.2 Common medical comorbidities seen in those with AUD

1. Liver disease. This can include alcoholic fatty liver, alcoholic hepatitis, and cirrhosis.
2. Cardiovascular disease. Those with AUD show higher rates of hypertension, cardiomyopathy, arrhythmia, and increased risk of stroke and heart attack.
3. Gastrointestinal disorders. Those with AUD are known to have higher incidences of gastritis, pancreatitis, and gastrointestinal (GI) bleeding.
4. Malnutrition and vitamin deficiencies (particularly B vitamins).
5. Neurologic disorders. These can include peripheral neuropathy, Wernicke–Korsakoff syndrome, and alcohol-related brain damage.
6. Alcohol-influenced psychiatric disorders. These can include depression, anxiety, and post-traumatic stress disorder (PTSD).
7. Respiratory complications. Those with chronic alcoholism are at increased risk of pneumonia secondary to immunosuppression and increased risk of aspiration.
8. Bone disorders. Those with AUD have a higher incidence of osteoporosis and risk of fracture.
9. Endocrine abnormalities. Men can show alcohol-related hypogonadism and both men and women can have adrenal insufficiency.
10. Metabolic syndrome. Those with chronic AUD have a higher risk of metabolic syndrome (a combination of obesity, insulin resistance, high blood pressure, and dyslipidemia) [6].

housing insecurity that may contribute to earlier cognitive decline. Those with AUD are less likely to adhere to medical treatments for conditions such as hypertension, diabetes, and cerebrovascular disease, causing earlier CNS-related complications of these conditions. Those with AUD are also much more likely to have alcohol-related head injuries from falls, which is known to increase the risk of early progressive cognitive impairment.

Unlike some other MNCDs, there are no specific cerebrospinal fluid (CSF) or imaging findings that are definitive for alcohol-related MNCD. Diagnosis is usually clinical. Those with AUD-related MNCD are more likely to have an earlier onset of dementia and the cognitive impairment will be in the setting of multiple other associated medical and psychosocial comorbidities of AUD [1]. Some other common conditions associated with chronic AUD are shown in Box 2.2.

Although uncommon, providers may be faced with the situation of a patient in long-term care who continues to intermittently abuse alcohol. Cravings for alcohol may remain long after admission and those patients with relatively preserved cognition and/or help from enabling friends or family may have access to alcohol while there. Naltrexone provides a generally well-tolerated pharmacologic option for blunting the pleasurable effects of alcohol in those with AUD. It acts through blockade of the mu-opioid receptors and therefore should not be administered to those who require opiate medications. Acamprosate is a good pharmacologic alternative for those patients who require intermittent or regular opiate use [7].

There are no specific pharmacologic treatments for alcohol-related MNCD. Clinical trials exploring the efficacy of cholinesterase inhibitors specifically in ARD are limited, and the existing data on their effectiveness on this condition are inconclusive. Treatment is largely symptomatic and focuses on the often-seen medical and psychiatric comorbidities that are seen with the condition. Abstinence from alcohol is the most critical factor

in slowing the further decline in alcohol-related MNCD. By the time a patient ends up in long-term care, they have often been abstaining from alcohol for some time due to a lack of access from being in a highly controlled care setting. Cognitive rehabilitation with occupational therapy or speech therapy can produce some improvements in functionality and nutritional support is important. Those with ARD may have a long history of very poor nutrition. Psychiatric comorbidities such as depression and anxiety are common and should be treated similarly to those without a history of AUD. Social support and engagement can improve the quality of life in these patients.

The prognosis of ARD can be difficult to predict as there are many confounding variables that may influence outcomes. Those who have early recognition of the condition with early interventions are likely to fare better than those who present with more severe cognitive impairment in a later stage of the disease. Conversely, those who present with severe cognitive impairment at a younger age are more likely to have more aggressive cognitive decline. Those who cease consumption of alcohol clearly do better than those who continue to abuse alcohol and patients with fewer medical comorbidities have a longer life expectancy. In addition, those in a more controlled and supportive environment such as long-term care or assisted living with greater support of healthy living and less availability of alcohol are likely to have improved outcomes.

There are certain difficult situations that may arise in the care of those with early ARD in the assisted living or long-term care environment. Those with less severe cognitive impairments are often still able to direct their own care and may have the ability to obtain alcohol independently or via the help of others. Balancing the autonomy of the resident to potentially make bad choices with a facility's responsibility to provide a safe and healthy environment for the resident and other residents can be a difficult tightrope to navigate. The consequences of continued heavy alcohol use in one already experiencing cognitive and health declines will often progress to the point where the individual will need a surrogate decision-maker to facilitate placement in a more controlled environment, such as long-term care or memory care. Once active drinking has stopped, these patients may maintain relative medical and cognitive stability or experience only slow continued declines in cognitive abilities.

Take-Home Points

- Chronic heavy alcohol use is toxic to the brain via multiple mechanisms.
- Labs and imaging are not specific for alcohol-related MNCD. Diagnosis is usually via history and clinical findings.
- Alcohol-induced MNCD can be partially reversible with abstinence, but the degree of reversibility depends on the severity of pathology.
- Once alcohol use has stopped there can be a long period of cognitive stability.

References

1. Ritchie, K., & Villebrun, D. (2008). Epidemiology of alcohol-related dementia. *Handbook of Clinical Neurology*, 89, 845–850.
2. Cheng, C., Huang, C. L., Tsai, C. J., Chou, P. H., Lin, C. C., & Chang, C. K. (2017). Alcohol-related dementia: A systemic review of epidemiological studies. *Psychosomatics*, 58 (4), 331–342.

3. Zhang, K., & Luo, J. (2019). Role of MCP-1 and CCR2 in alcohol neurotoxicity. *Pharmacological Research*, 139, 360. https://doi.org/10.1016/j.phrs.2018.11.030
4. King, J. A., Nephew, B. C., Choudhury, A., Poirier, G. L., Lim, A., & Mandrekar, P. (2020). Chronic alcohol-induced liver injury correlates with memory deficits: Role for neuroinflammation. *Alcohol (Fayetteville, N.Y.)*, 83, 75–81. https://doi.org/10.1016/j.alcohol.2019.07.005
5. Sachdeva, A., Chandra, M., Choudhary, M., Dayal, P., & Anand, K. S. (2016). Alcohol-related dementia and neurocognitive impairment: A review study. *International Journal of High-Risk Behaviors & Addiction*, 5 (3), e27976 https://doi.org/10.5812/ijhrba.27976
6. MacKillop, J., Agabio, R., Feldstein-Ewing, S., Heilig, M., Kelly, J. F., Leggio, L., Lingford-Hughes, A., Palmer, A., Parry, C., Ray, L., & Rehm, J. (2022). Hazardous drinking and alcohol use disorders. *Nature Reviews. Disease Primers*, 8 (1), 80. https://doi.org/10.1038/s41572–022-00406-1
7. Murphy, C. E., Wang, R. C., Montoy, J. C., Whittaker, E., & Raven, M. (2022). Effect of extended-release naltrexone on alcohol consumption: A systematic review and meta-analysis. *Addiction*, 117 (2), 271–281. https://doi.org/10.1111/add.15572

Further Reading

Kaldy, J. (2023). Alcohol use in long-term care communities: Juggling choice and safety. *Caring for the Ages*, 24 (1), 1. https://doi.org/10.1016/j.carage.2022.12.013

Lohoff, F. W. (2020). Pharmacotherapies and personalized medicine for alcohol use disorder: A review. *Pharmacogenomics*, 21 (15), 1117–1138. https://doi.org/10.2217/pgs-2020-0079

Sullivan, E. V., & Pfefferbaum, A. (2019). Brain-behavior relations and effects of aging and common comorbidities in alcohol use disorder: A review. *Neuropsychology*, 33 (6), 760. https://doi.org/10.1037/neu0000557

Section 1 **Neurocognitive Disorders**

Case 3

"It's like She's a Different Person"

Neuropsychiatric Complications of TBI

Mrs. S was a 78-year-old resident of a medium-sized skilled nursing facility. She had been a resident of the facility for four years following a fall at home. Nursing staff at the facility requested that the consulting psychiatrist see the patient after she had returned from hospital where she was evaluated after a fall with a scalp laceration. Since returning from the emergency department Mrs. S had not been acting as usual. She had been showing personality changes. Prior to her fall, she could be described as fairly quiet but pleasant. She enjoyed social functions in the nursing facility and was generally liked by other residents and nursing staff alike. She had a particular affinity for music and her favorite activities involved musical performers who would come to the facility once or twice a week. Mrs. S loved to play the piano and would often entertain others in the facility after evening meals with her piano playing and singing.

After her return from the emergency room following her fall three weeks prior, Mrs. S seemed to have little interest in social activities. She had stopped attending most musical activities and had stopped playing music for other residents even after much encouragement from her friends. Her friends and staff members had noted that Mrs. S was now described as often short-tempered and irritable with others. Mrs. S's daughter was also concerned about these changes. "It's like she's a different person, what happened to my mom!" her daughter exclaimed during the provider's first visit with the patient.

On review of Mrs. S's chart before the meeting, it was noted that she had a history of obesity class 3 (BMI 43), impaired mobility, hypertension, hyperlipidemia, and psoriasis. She had polio as a young girl which left her with shortening of her left lower extremity, scoliosis, and lifelong issues with impaired mobility. For most of her life, she was fully mobile with a cane or bilateral hand crutches but as she reached her 50s and 60s she began gaining weight. Her weight gain led to a slow steady decline in mobility. Prior to her most recent fall, she was dependent mostly on a motorized scooter for mobility but was still able to self-transfer from her bed to her scooter with the aid of her hand crutches.

A review of her chart also indicated that Mrs. S had several hospital visits for falls in the past year alone. None had resulted in serious injury, only scalp contusions or lacerations. The nursing staff mentioned that Mrs. S may have lost consciousness for "a while" after her most recent fall. Her falls always happened when she tried to transfer herself to her motorized scooter or while transferring on and off the commode. She had a CT brain scan after her latest fall that showed only mild age-related small vessel ischemic changes. There was no comment on atrophy or subdural hematoma. A review of recent labs indicated that the patient's comprehensive metabolic panel (CMP), complete blood count (CBC), thyroid studies, b12, and folate levels were all within normal limits.