Sleep disordered breathing (SDB) including obstructive sleep apnea (OSA), central sleep apnea (CSA), and Cheyne-Stokes breathing is common in patients with congestive heart failure (CHF). In a study of 81 males with an ejection fraction (EF) < 45%, 51% of patients had documented SDB with the majority of cases representing CSA. Studies suggest that SDB is an independent risk factor for increased morbidity and mortality in the setting of CHF.

Like OSA, CSA frequently presents with nighttime awakenings, nocturnal hypoxia, and daytime somnolence, but only OSA has been reported to present with delirium. We present a patient with clear manifestations of CSA with frank delirium that improved only after BPAP therapy.

**REPORT OF CASE**

An 82-year-old male was in his usual state of health until 2 months prior when he presented to the hospital with a myocardial infarction. Heparin was initiated, and he subsequently fell, with a resultant subdural hematoma. The subdural hematoma appeared stable on serial imaging, and he was discharged after 10 days.

Over the next 2 months, his family noted episodes of delirium, difficulty sleeping, and failure to thrive with a 30-pound weight loss. He was thus readmitted to the hospital for work-up of his delirium. A head computed tomogram (CT) showed complete resolution of the subdural hematoma.

His medical history was significant for ischemic cardiomyopathy (EF 30%), mild chronic obstructive pulmonary disease, and atrial fibrillation. His medications included digoxin, furosemide, diltiazem, enalapril, ezetimibe, and tiotropium. He had no history of alcohol or illicit drug use and was previously independent.

Physical exam revealed an elderly delirious male who was oriented to name and place only; his mini-mental status exam (MMSE) score was 19/30. Laboratory tests including complete blood count, comprehensive metabolic panel, thyroid-stimulating hormone, and urinalysis were unremarkable.

He was treated for possible pneumonia and congestive heart failure with levofloxacin and furosemide without improvement of his delirium. Magnetic resonance imaging (MRI) of the brain was unrevealing, and transthoracic echocardiogram revealed an EF of 20% to 25%.

During his hospitalization, he was noted to have apneic events lasting up to 12 seconds while sleeping. He was placed on continuous positive airway pressure (CPAP), which he did not tolerate, and was later switched to bilevel positive airway pressure (BPAP). With BPAP therapy, he began sleeping well, and his delirium improved. He was discharged after a 12-day hospitalization.

An outpatient polysomnogram revealed severe central sleep apnea (apnea index 70) with Cheyne-Stokes breathing (Figure 1) and oxygen desaturation with a nadir of 69%. The patient has continued on BPAP therapy with complete resolution of his delirium, with a repeat MMSE of 27/30. He sleeps 9 hours per night without daytime somnolence and has regained his lost weight.

**DISCUSSION**

To our knowledge, this is the first reported case to link central sleep apnea and delirium, although obstructive sleep apnea has been reported to cause delirium. One of the first documented cases was described in 1998, when a patient presented with acute delirium and did not improve until diagnosis and treatment of his severe OSA. A recent abstract at the American Society of Anesthesiologists demonstrated that in a population of non-demented elderly patients undergoing elective knee arthroplasty, preexisting OSA was the main risk factor for the occurrence of postoperative delirium.

The correlation of sleep apnea and cognitive impairment may reflect any number of physiologic mechanisms. Watershed areas with poor cerebral blood flow due to conditions such as stroke or vascular stenoses may be especially susceptible to hypoxic events. In contrast, hypercapnia may reduce blood flow in damaged intracerebral vessels by dilating normal vessels. In
a prospective cohort study of 153 patients with acute ischemic stroke, 15% of patients were found to have an intracranial steal phenomenon on transcranial Doppler with worsening neurologic deterioration during sleep apneic events. This phenomenon, coined the “Robin Hood Syndrome,” may further exacerbate delirium in patients with intracerebral vascular damage. Finally, respiratory acidosis, increased adrenergic tone, and worsened cardiac instability may all affect various organ systems to exacerbate delirium during apneic events.

Delirium has a broad differential and sleep apnea, obstructive and central, should be included in the list. CHF is also known to cause delirium. Our patient had several confounding variables; however, after resolution of his heart failure, subdural hematoma, and pneumonia, his delirium persisted until his CSA was treated. Therapies for CSA include oxygen, CPAP, and BPAP. CPAP improves not only the apnea index but also the left ventricular EF in patients with CSA and heart failure. Thus, early recognition and treatment may lead to improvement in cardiac function and, in the case of our patient, a dramatic improvement in delirium and quality of life.

**REFERENCES**


**SUBMISSION & CORRESPONDENCE INFORMATION**

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