Nocturnal Onset Ischemic Stroke Provoked by Sleep-Disordered Breathing Advanced With Congestive Heart Failure

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Recently, sleep-disordered breathing and nocturnal hypoxia have been recognized to increase the risk of cerebrovascular disease. Kirkham et al reported that nocturnal hypoxemia was a predictor of future cerebrovascular events in sickle-cell disease. However, it remains unclear whether nocturnal hypoxic episodes directly lead to nocturnal onset stroke, because other predisposing conditions might confound the association between sleep-disordered breathing and the risk of stroke. Congestive heart failure is often accompanied by central sleep-disordered breathing. We report here a case of nocturnal onset ischemic stroke directly provoked by sleep-disordered breathing, newly developed together with congestive heart failure.

Case Report

The patient was a 67-year-old man who was diagnosed with congestive heart failure April 17, 2001. He had a history of coronary artery bypass grafting because of acute myocardial infarction (three-vessel disease) 11 months previously, as well as Leriche syndrome. He had been given anticoagulation therapy with a prothrombin time of approximately 1.6 international normalized ratio (INR), antiplatelet therapy, nitrate, and long-acting calcium antagonists, and his blood pressure (BP) levels had been controlled from 130 to 160 mm Hg for systolic BP, and from 75 to 90 mm Hg for diastolic BP. Because of the development of mild congestive heart failure, administration of a diuretic (spironolactone) was started. Mild left hemiparesis and sensory disturbance were present when the patient awakened 15 days after starting diuretic intake, and this neurologic deficit continued until the next morning. One month after the episode, brain magnetic resonance (MR) imaging revealed multiple cerebral infarcts on T2-weighted imaging (Fig. 1A) and flair imaging (Fig. 1B). Diffusion MR imaging revealed a new infarct corresponding to the patient’s neurologic deficit (Fig. 1C, arrow). Brain MR angiography showed total occlusion of left internal carotid artery (Fig. 2, arrow). When compared with the BP variables obtained from ambulatory BP monitoring 3 months before the episode, the BP variables 1 month after the episode were significantly lower (after versus before the episode, 24-h BP: 111/70 v 125/72 mm Hg; awake BP: 114/72 v 133/77 mm Hg; sleep BP: 106/67 v 111/66 mm Hg). In addition, over-
night pulse oximetry one month after the episode newly revealed moderate sleep-disordered breathing with a high frequency of 4% desaturation episodes (29.2/h) during sleep, whereas mean awake oxygen saturation was 97%. Polysomnography disclosed that the patient’s apnea–hypopnea index was 55/h (central apnea dominant, 71% of total apnea). Three months before the episode, his frequency of 4% desaturation episodes during sleep had been only 4.8/h.

**Discussion**

In this patient, MR angiography examination revealed occlusion of internal carotid artery; however, he had noted no clinical neurologic deficits. Thus, we considered that nocturnal hypoxia, which developed along with congestive heart failure, directly triggered transient ischemic attack with a new infarct verified by MR imaging of the brain. A previous case-control study showed that sleep apnea was fivefold more frequent in patients with transient ischemic attack than in a normal control group (62.5% v 12.5%). In addition, nocturnal BP reduction (5 mm Hg reduction for systolic BP) caused by a diuretic might reduce cerebral perfusion and trigger a nocturnal ischemic episode. We have recently reported that elderly hypertensive patients with marked nocturnal BP fall (extreme dipping pattern) reduction have a higher risk of stroke than with appropriate nocturnal BP fall (more normal dipping pattern). During an apneic episode, cerebral perfusion pressure was found to decrease by approximately 11.2 ± 7.7 mm Hg (mean ± SD) from baseline, and a significant reduction in middle cerebral artery blood flow velocity has been reported. In addition to the direct effect of hypoxia, these intracranial hemodynamic changes in patients with marginal circulatory reserve would contribute to increase the risk of ischemic stroke.

In this patient, onset of congestive heart failure per se triggered nocturnal hypoxia. The use of diuretic therapy for congestive heart failure may have independently contributed to nocturnal hypoxia. Thus, this implies that initial therapy of heart failure might emphasize nondiuretic options.

In conclusion, if congestive heart failure develops in high-risk patients with severe systemic atherosclerosis, simple evaluation of nocturnal hypoxemic episodes using pulse oximetry should provide valuable information for predicting the risk of stroke.

**References**