Dysphagia and Obstructive Sleep Apnea in Acute, First-Ever, Ischemic Stroke

Anna Losurdo, MD,* Valerio Brunetti, MD,* Aldobrando Broccolini, MD, PhD,* Pietro Caliandro, MD, PhD,* Giovanni Frisullo, MD, PhD,* Roberta Morosetti, MD, PhD,* Fabio Pilato, MD, PhD,* Paolo Profice, MD, PhD,* Nadia Mariagrazia Giannantoni, MD,* Maria Luisa Sacchetti, MD,† Elisa Testani, MD, PhD,* Catello Vollono, MD, PhD,* and Giacomo Della Marca, MD, PhD*

Background: Obstructive sleep apnea (OSA) and dysphagia are common in acute stroke and are both associated with increased risk of complications and worse prognosis. The aims of the present study were (1) to evaluate the prevalence of OSA and dysphagia in patients with acute, first-ever, ischemic stroke; (2) to investigate their clinical correlates; and (3) to verify if these conditions are associated in acute ischemic stroke. *Methods:* We enrolled a cohort of 140 consecutive patients with acute-onset (<48 hours), first-ever ischemic stroke. Computed tomography (CT) and magnetic resonance imaging scans confirmed the diagnosis. Neurological deficit was measured using the National Institutes of Health Stroke Scale (NIHSS) by examiners trained and certified in the use of this scale. Patients underwent a clinical evaluation of dysphagia (Gugging Swallowing Screen) and a cardiorespiratory sleep study to evaluate the presence of OSA. *Results:* There are 72 patients (51.4%) with obstructive sleep apnea (OSA+), and there are 81 patients (57.8%) with dysphagia (Dys+). OSA+ patients were significantly older ($P = .046$) and had greater body mass index (BMI) ($P = .002$), neck circumference ($P = .001$), presence of diabetes ($P = .013$), and hypertension ($P < .001$). Dys+ patients had greater NIHSS ($P < .001$), lower Alberta Stroke Programme Early CT Score ($P < .001$), with greater BMI ($P = .030$). The association of OSA and dysphagia was greater than that expected based on the prevalence of each condition in acute stroke ($P < .001$). *Conclusions:* OSA and dysphagia are associated in first-ever, acute ischemic stroke. **Key Words:** Stroke—sleep apnea—obstructive sleep apnea—sleep—dysphagia.

From the *Institute of Neurology, Catholic University, Rome, Italy; and †Department of Neurology and Psychiatry, Sapienza University, Rome, Italy.

Received June 14, 2017; accepted September 24, 2017.

Author contributions: Dr. Losurdo: study concept and design, drafting the manuscript, interpretation of data, accepts responsibility for conduct of research, and acquisition of data. Dr. Brunetti: drafting the manuscript, interpretation of data, accepts responsibility for conduct of research, and acquisition of data. Drs. Broccolini, Caliandro, Frisullo, Morosetti, Pilato, Profice, Giannantoni, Sacchetti, Testani, Vollono: interpretation of data, accept responsibility for conduct of research, and acquisition of data. Dr. Della Marca: interpretation of data, accepts responsibility for conduct of research, study supervision, revising the manuscript, and final approval, guarantor of the paper, taking responsibility for the integrity of the work as a whole, from inception to published article.

Study funding: No targeted funding reported.

Disclosure: All authors report no disclosures relevant to the manuscript.

Potential conflicts of interest: The authors have no conflicts of interest to disclose.

Address correspondence to Valerio Brunetti, MD, Institute of Neurology, Catholic University, Largo A. Gemelli no. 8, Rome 00168, Italy. E-mail: v.brunetti2509@gmail.com.

1052-3057/$ - see front matter

https://doi.org/10.1016/j.jstrokecerebrovasdis.2017.09.051

Introduction

Obstructive sleep apnea (OSA) is the intermittent reduction or cessation of airflow during sleep due to partial or total obstruction of the upper airway. The prevalence of OSA in the general adult population is 2%-4%,[1] and OSA is an independent risk factor for stroke.[2]

The prevalence of OSA is higher in patients with acute stroke,[3] regardless of the severity and the site of the brain lesion.[4,5] In a systematic review of 29 studies, which included over 2000 patients, the prevalence of OSA in acute stroke was 72% when defined as an apnea hypopnea index (AHI) of greater than 5 events/h, 63% for AHI greater than 10, and 38% for AHI greater than 20.[6] OSA has also been associated with the onset of stroke during sleep (wake-up stroke) and with paradoxical embolism.[7] The presence of OSA may have a negative impact on the outcome of patients with stroke.[8]

OSA is a multifactorial pathology, characterized by a reduced ability to keep upper airway patency; obstructive events are due to the collapse of pharyngeal walls during sleep. Pharyngeal muscle dysfunction during the acute phase of stroke is common, and dysphagia has been demonstrated in 38.5% of patients with acute stroke.[9,10] In the first 3 days after the onset of stroke, clinically evident dysphagia, assessed with bedside screening techniques, is reported in 37%-45% of patients[10]; its occurrence is associated with the extension of the lesion, the clinical severity, and age. Dysphagia has generally a good prognosis: swallowing generally improves within 2 weeks, and only a minority of patients needs permanent tube feeding.[10] Dysphagia increases the risk of aspiration pneumonia, and it is associated with worse clinical outcome.[11] The high prevalence of OSA and dysphagia in acute stroke, and the presence of pharyngeal dysfunction in both conditions, suggest that these conditions may share a common pathogenetic mechanism.

The prevalence of the association between OSA and dysphagia in acute ischemic stroke has not been systematically investigated. In patients with acute ischemic and hemorrhagic stroke, the presence of oxygen desaturation, detected by continuous pulse oximetry, is associated with increased age, higher National Institute of Health Stroke Scale (NIHSS) score,[12,13] and the presence of dysphagia.[11] An association of dysphagia, dysarthria, and severe OSA (AHI > 30) has been reported in a cohort of Japanese patients with intraparenchymal brain hemorrhage.[14]

The aims of the present study were (1) to evaluate the prevalence of OSA and dysphagia in patients with acute, first-ever, ischemic stroke; (2) to investigate the clinical correlates of these conditions; and (3) to verify the hypothesis that these conditions are associated with each other.

Materials and Methods

Patients

We analyzed a cohort of 140 consecutive patients admitted to the stroke unit of the Policlinico Agostino Gemelli, Catholic University, Rome, Italy. Patients were enrolled between February 2013 and July 2014. Inclusion criteria were both genders, age older than 18 years, a definite clinical diagnosis of ischemic stroke with onset of clinical symptoms within the previous 72 hours, and a NIHSS[12,13] greater than 1 at the time of the study, when clinical assessment of dysphagia and polygraphic evaluation of OSA were performed. Stroke was defined as any acute focal cerebral event with symptoms lasting at least 24 hours, as confirmed by neuroimaging evidence (computed tomography [CT] or magnetic resonance imaging [MRI]) of brain ischemia. Exclusion criteria were transient ischemic attack, absence of neuroimaging (CT or MRI) evidence of brain ischemia, cerebral venous thrombosis, cerebral hemorrhage, history of previous stroke, presence of concomitant neurologic diseases, previous history of dysphagia, OSA patients in treatment with positive airway pressure ventilation, extreme severity of clinical conditions requiring intubation, and intensive care unit treatment. The extension of the ischemic lesion was measured by a quantitative score based on CT scans, the Alberta Stroke Programme Early CT Score (ASPECTS).[15] The onset of symptoms of stroke was carefully assessed by questioning the patient and any witness to ascertain whether it was noted on waking or when already awake. In the former case, the event was classified as "wake-up stroke."[7] All patients or caregiver gave written informed consent before enrollment. The study was performed in agreement with the Declaration of Helsinki and was approved by the Ethics Committees of the Catholic University of Rome.

Clinical Information Collected

For each patient, the following data were recorded: age, gender, body mass index (BMI), neck circumference, atrial fibrillation (AF) (documented by electrocardiography), history of hypertension (systolic blood pressure ≥ 135 mm Hg or diastolic blood pressure ≥ 85 mm Hg), diabetes (fasting serum glucose > 127 mg/mL), hypercholesterolemia (total cholesterol ≥ 190 mg/dL), and chronic therapies in use at the moment of the index event.[16-18] BMI and neck circumference are clinical predictors of OSA.[19,20] Neurological deficit was measured at the time of the cardiorespiratory sleep study, using the NIHSS, by examiners trained and certified in the use of this scale. Vascular syndromes were classified on the basis of the clinical presentation at the onset of symptoms, according to the Oxfordshire Community Stroke Project,[21] as lacunar syndrome (LACS), posterior circulation