

## CASE REPORTS

### Acute lacunar infarct in an obese adolescent with obstructive sleep apnea

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Obstructive sleep apnea is associated with an increased risk of acute ischemic cerebrovascular accidents in adults. However, this association has not been well established in the pediatric population. We report a case of acute right internal capsule lacunar infarct manifesting as left-sided hemiplegia in an adolescent with severe obesity. Severe untreated obstructive sleep apnea was identified during the hospital stay. Positive airway pressure therapy, weight loss, and tonsillectomy eventually normalized sleep-disordered breathing. The patient recovered completely without any residual neurological deficits with multidisciplinary care. Lacunar infarcts due to hypertension-related cerebral vasculopathy are a well-known cause of stroke in adults. Untreated obstructive sleep apnea may cause hypertension and potentially predispose even the pediatric population to develop lacunar infarcts. Polysomnography needs to be considered as a part of the diagnostic algorithm in pediatric patients with stroke.

**Keywords:** pediatric obesity, ischemic stroke, obstructive sleep apnea

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#### INTRODUCTION

Obstructive sleep apnea (OSA) is known to be associated with potentially life-threatening conditions, including acute coronary syndromes and ischemic cerebrovascular accident (CVA) in adults.<sup>1</sup> Approximately 70% of adults with ischemic strokes were identified to have untreated OSA.<sup>1</sup> The association between untreated OSA and ischemic CVA is well established in adults, but it is not well investigated in the pediatric population.

Ischemic stroke in children is not as common as in adults.<sup>2</sup> The common etiologies for pediatric ischemic stroke are congenital heart disease, prothrombotic conditions, sickle cell disease, and arteriopathies.<sup>2</sup> Obesity is a known risk factor for OSA.<sup>3</sup> The National Health and Nutrition Examination Survey reported that one-third of children in the United States were either overweight or obese, with disproportionately higher rates in African American and Hispanic children.<sup>4</sup> The epidemic of pediatric obesity has amplified the incidence of weight-related OSA in children.<sup>5</sup> Furthermore, obesity is an established, independent risk factor for stroke in adults.<sup>3</sup> In this context, we report a case of an adolescent with severe obesity and untreated OSA who presented with an acute ischemic infarct in the right internal capsule.

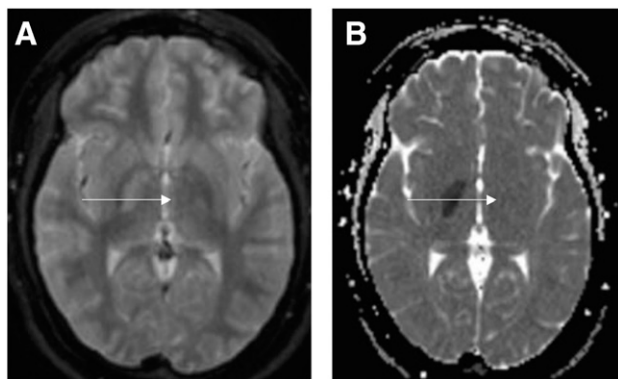
#### REPORT OF CASE

A 16-year-old White female was brought to the emergency department by her mother for a wake-up stroke (patient going to sleep without neurological deficits and waking up with stroke symptoms) manifested by left-sided hemiplegia involving the face, arm, and leg. The National Institutes of Health Stroke Scale at presentation was 10 (facial palsy 2, left arm 4, left leg 3, and dysarthria 1). National Institutes of Health Stroke Scale scores range from 0–42, and a score of 10 at baseline is considered a

moderately severe stroke.<sup>6</sup> The patient had class III obesity (375 lbs, body mass index 62.1 kg/m<sup>2</sup>, 99.8th percentile, z score 2.9) at the time of her initial presentation. Magnetic resonance imaging of the brain demonstrated a lacunar infarct in the posterior limb of the internal capsule on the right side, as shown in **Figure 1**. Computed tomography angiography of the head and neck were negative for flow-limiting stenosis or vasculopathic pattern. Extensive laboratory work, including coagulation and autoimmune panel, were negative. Aside from obesity, past medical history included well-managed intermittent asthma and irregular menses. Prior physician visits earlier in the year did not find abnormally elevated blood pressures. However, the patient's mother did report loud snoring and frequently witnessed apnea, especially with the patient in the supine position. She does not have a history of sickle cell disease. There was no evidence of elevated low-density lipoprotein cholesterol (78 mg/dL). Echocardiogram and electrocardiogram were negative for structural and conduction abnormalities.

The patient was noted to have intermittent hypoxemia while asleep during her hospital stay. The initial split-night study demonstrated severe OSA with an obstructive apnea-hypopnea index (OAH) of 172 events/h (0 events/h central, 3 events/h mixed) and severe nocturnal hypoxemia (77.2 minutes or 58.4% of sleep time with oxyhemoglobin saturation < 90%, oxyhemoglobin saturation nadir of 60%) as illustrated in **Table 1**. Transcutaneous CO<sub>2</sub> values were within normal limits. Positive airway pressure (PAP) titration and positional therapy normalized OAH (1 event/h) at a bilevel PAP setting of 19/12 cm H<sub>2</sub>O (**Figure 2**). She continued bilevel PAP of 19/12 cm H<sub>2</sub>O. After 2 months of inpatient rehabilitation and PAP therapy with > 95% adherence (defined as more than 4 hours of use per night), the patient improved with partial resolution of her motor deficits, and she was discharged home. The patient was subsequently managed by a multidisciplinary team, including sleep

**Figure 1**—MRI of the brain without contrast showing restricted diffusion lesion in the posterior limb of the internal capsule on the right side.



(A) Hyperintense lesion on DWI. (B) Lesion with corresponding hypointensity on ADC sequence. ADC = apparent diffusion coefficient, DWI = diffusion weighted imaging, MRI = magnetic resonance imaging.

medicine, neurology, rehabilitation medicine, and a structured weight management program.

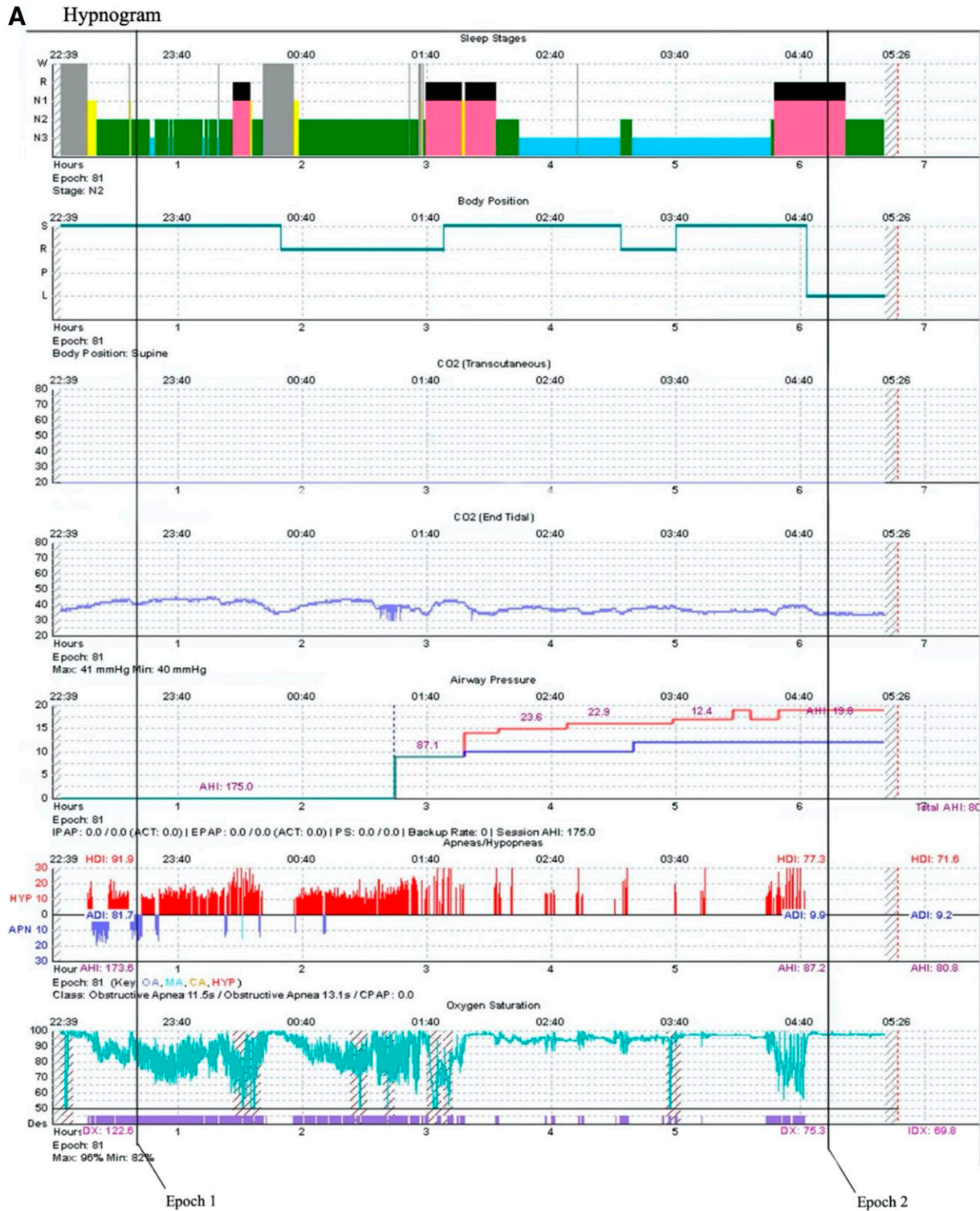
The patient was noted to have 3–4+ tonsils, occupying roughly 75% of the pharyngeal space. At the time of acute presentation, the patient was deemed not to be an ideal candidate for surgery (both adenotonsillectomy [T&A] and bariatric surgery) due to an increased risk of perioperative complications. The patient was enrolled in the medical weight management program. She lost 93 pounds over a span of 1.2 years with diet and exercise. Her body mass index dropped from 62.1 kg/m<sup>2</sup> to 47.83 kg/m<sup>2</sup>. Left-sided weakness had resolved completely by this point. A repeat polysomnogram after weight loss revealed severe OAH (43 events/h, central apnea-hypopnea index of 0 events/h) but improved oxygenation (2.8% of sleep time with oxyhemoglobin saturation below 90%, oxyhemoglobin saturation nadir of 74%). As mentioned in **Table 1**, the response to PAP therapy (residual OAH of 0.4 events/h with a bilevel PAP setting of 14/10 cm H<sub>2</sub>O) and nocturnal hypoxemia improved significantly.

**Table 1**—Serial polysomnograms demonstrating OSA improvement with weight loss and T&A.

	PSG Number 1	PSG Number 2	PSG Number 3
Time from stroke presentation, d	29	325	962
Type of study	Split-night PSG	Split-night PSG	Split-night PSG
Interim interventions	N/A	Weight loss	T&A
Weight (lbs)	367	300	291
BMI (kg/m <sup>2</sup> )	62.1	48.4	48.4
BMI percentile, z score	99.74, 2.80	99.51, 2.59	99.29, 2.45
Key PSG parameters			
TST (min) for baseline portion	132	182	213
% stage N1/N2/N3 sleep	2.9/43.4/33.1	7.6/68.8/10.0	3.7/63.0/17.1
% REM sleep	20.7	13.6	16.1
Sleep efficiency	91.9%	86.9%	88.4%
% supine	63.3	0	5.9
Total AHI	175	40	5.6
REM sleep AHI	120	83	20
Supine AHI	170	NA	24
% obstructive/mixed/central	98.2/1.8/0.0	100/0/0	100/0/0
Arousal index	51.5	20.4	11.8
Oxyhemoglobin nadir	60%	74%	90%
% time SpO <sub>2</sub> < 90%	58.4%	2.8%	0%
% etCO <sub>2</sub> > 50 mm Hg	0%	0%	0%
PAP parameters			
Best bilevel PAP pressure	19/12	14/10	10/5
AHI at best PAP pressure	1	0.4	2.0
Oxygen nadir	73%	94%	93%
Arousal index	11.7	5.6	2.0
% tcPCO <sub>2</sub> > 50 mm Hg	0%	0%	0%

Of note for PSG number 1, while the patient was on optimum bilevel PAP, AHI was not normalized in the supine position; however, AHI was normalized in the lateral recumbent position. As such, the patient was recommended for positional therapy in addition to bilevel PAP until the following studies. AHI = apnea-hypopnea index, BMI = body-mass index, etCO<sub>2</sub> = end-tidal carbon dioxide, OSA = obstructive sleep apnea, PAP = positive airway pressure, PSG = polysomnography, REM = rapid eye movement, SpO<sub>2</sub> = oxyhemoglobin saturation, T&A = adenotonsillectomy, tcPCO<sub>2</sub> = transcutaneous monitoring of carbon dioxide partial pressure, TST = total sleep time.

Figure 2—Hypnogram of the initial split-night study and representative epochs.

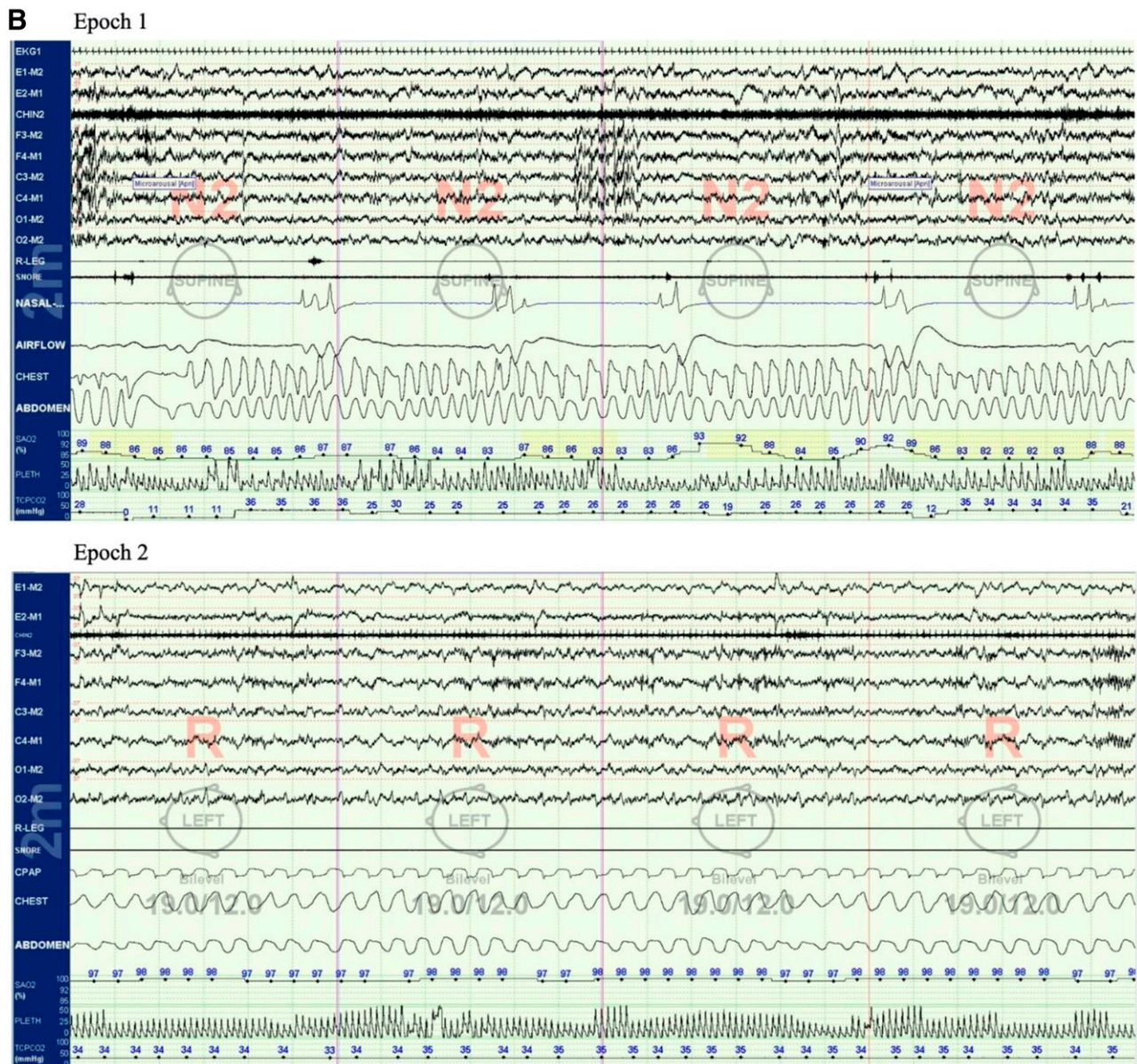


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**Figure 2**—Hypnogram of the initial split-night study and representative epochs. (continued)



**(A)** Hypnogram of the initial split night study showing severe OSA and severe nocturnal hypoxemia. **(B)** Two representative epochs of (epoch 1) the severe OSA and (epoch 2) PAP and positional therapy that normalized AHI at a bilevel PAP setting of 19/12 cm of H<sub>2</sub>O. ADI = apnea disturbance index, AHI = apnea-hypopnea index, APN = apnea, CA = central apnea, CPAP = continuous positive airway pressure, DX = desaturation time, EPAP = expiratory positive airway pressure, act, HDI = hypopnea disturbance index, HYP = hypopnea, IDX = desaturation index, IPAP = inspiratory positive airway pressure, MA = mixed apnea, OA/OSA = obstructive sleep apnea, PAP = positive airway pressure, PS = pressure support.

The patient had a T&A 2.5 years after the stroke. A third split-night study showed remarkable improvement, as shown in **Table 1** (baseline OAH of 5.6 events/h and residual OAH of 2 events/h with bilevel PAP 10/5 cm H<sub>2</sub>O). She had not gained significant weight since her previous polysomnogram 2 years previously. PAP therapy abolished snoring and improved daytime sleepiness. Weight loss, T&A, rehabilitation, and PAP therapy facilitated complete recovery from the stroke and OSA-related symptoms. She is currently a full-time college student and continuing with weight loss medical management.

### DISCUSSION

Our case report emphasizes the possible association between obesity, OSA, and CVA in the pediatric population. It also highlights the positive impact of weight loss and OSA treatment on stroke recovery.

Untreated OSA is associated with increased risk of ischemic CVA, increased stroke-related mortality, and poor long-term neurological recovery in adults.<sup>7</sup> Although the causal effect of untreated OSA on CVA cannot be entirely ascertained, several

plausible mechanisms support this hypothesis in adults.<sup>1,7,9</sup> Hypertension due to increased sympathetic activity, increased inflammatory markers, platelet dysfunction, endothelial disruption, and altered cerebral hemodynamics are some of the postulated mechanisms.<sup>1,8,9</sup> Hypertension remains the major and modifiable risk factor for stroke, probably irrespective of age group. In the pediatric population, OSA severity has been associated with hypertension, likely due to increased catecholamine levels.<sup>9</sup> Hypertension is known to cause lipohyalinosis of small penetrating vessels in the brain and is a strong risk factor for lacunar infarcts.<sup>9</sup> Except for obesity, our patient did not have any other identifiable risk factors for stroke typically seen in the pediatric population. Even though the patient did not have a formal diagnosis of hypertension, it is reasonable to assume that she had untreated hypertension—perhaps due to untreated OSA and obesity—before the stroke. The evidence of lacunar infarct further supports this hypothesis. This case report emphasizes the possibility of a multidimensional relationship between stroke, OSA, and obesity in the pediatric population.

Metabolic syndrome, obesity, and weight gain correlate with OSA.<sup>1,3</sup> Surgical modalities such as gastric bypass or laparoscopic sleeve gastrectomy result in greater weight loss and are more effective at normalizing sleep-disordered breathing compared with dietary restriction, physical activity, and psychological support.<sup>9,10</sup> A longitudinal population-based study predicted a 26% (95% confidence interval, 18%–34%) reduction in the apnea-hypopnea index with 10% weight loss.<sup>11</sup> Without surgical management, our patient was able to achieve a significant weight loss of 93 lbs, and the management of obesity with behavioral and nutritional modifications improved OSA in our patient.

T&A is often indicated as a first-line treatment option in children with sleep-disordered breathing.<sup>9</sup> Obese children who underwent T&A have been shown to have a significant decrease in OSA severity but to a lesser degree than nonobese children.<sup>9</sup> As expected in our patient's case, T&A helped further reduce the severity of OSA. Improved daytime sleepiness due to PAP therapy may enhance participation in rehabilitation and eventually result in enhanced neurologic recovery after a stroke. Treatment of OSA with PAP was shown to improve hypoxemia, hypertension, and outcomes in adults with ischemic stroke.<sup>12</sup> Polysomnography deserves a unique place in the diagnostic algorithm in pediatric patients with ischemic stroke, especially in obese children. PAP therapy should be a part of the treatment plan as it may assist in neurologic recovery.

## ABBREVIATIONS

CVA, cerebrovascular accident  
 OAH, obstructive apnea-hypopnea index  
 OSA, obstructive sleep apnea  
 PAP, positive airway pressure  
 T&A, adenotonsillectomy

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## SUBMISSION & CORRESPONDENCE INFORMATION

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## DISCLOSURE STATEMENT

All authors have seen and approved the manuscript. The authors report no conflicts of interest.