



Complications of anesthesia during electroconvulsive therapy due to undiagnosed obstructive sleep apnea: A case-study



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ABSTRACT

This is a case description of two patients with bipolar affective disorder, who presented complications, possibly due to underlying, undiagnosed obstructive sleep apnea syndrome (OSAS), during anesthesia for electroconvulsive therapy (ECT).

The first patient, just after receiving the second ECT, developed tachypnea with spasm of the upper airways and severe oxygen desaturation. He was intubated and transferred to the medical intensive care unit where he was extubated 15 h later. The second patient, just after the eighth ECT, developed tachycardia and severe hypertension. He was transferred to the recovery room where he received oxygen therapy via nasal cannula and amlodipine. Both patients in the diagnostic polysomnographic tests which followed revealed a moderate to high apnea – hypopnea index (AHI) and distortion of sleep architecture. These cases highlight the need to assess for OSAS patients who receive ECT, especially if they exhibit perianesthesia complications.

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1. Introduction

OSAS is a common, chronic condition characterized by repeated episodes of complete or partial obstruction of the upper airway during sleep, associated with increased respiratory efforts, intermittent arterial oxygen desaturation, systemic and pulmonary arterial blood pressure alterations and sleep fragmentation [1].

The HypnoLaus study reported a high prevalence of moderate-to-severe sleep-disordered breathing (≥ 15 events per hour), of 23,4% in women and 49,7% in men, in a population-based sample using full polysomnography, [2]. Another similar study from Brazil also reported a high prevalence of sleep-disordered breathing (apnoea-hypopnoea index > 5 events per hour) of 46,6% in men and 30,5% in women [3]. Few studies examine the prevalence of OSAS in individuals with clinically diagnosed psychiatric disorders [4]. According to a recent review, it appears that there is an elevated prevalence of the disease in psychiatric patients with major

depressive disorder (MDD) and posttraumatic stress disorder (PTSD) [4]. OSAS is associated with greater mortality, as well as significant adverse cardiovascular, gastrointestinal, genitourinary, endocrine, psychiatric, and social consequences [1]. Moreover, patients with OSAS are particularly vulnerable during anesthesia and sedation [5].

Electroconvulsive therapy is generally considered a safe procedure, in which electricity is used to create a seizure in a patient who has received general anesthesia. Although there is no survey examining the prevalence of OSAS among patients receiving anesthesia for ECT, failure to recognize and manage the risk for OSAS in patients receiving this treatment might expose them to anesthetic difficulties [6]. As spontaneous breath returns, a sleep apneic patient can express difficulty in supporting ventilation. Oxygen saturation can rapidly deteriorate due to the supine position and the diminished pulmonary reserve, particularly in obese patients.

In this paper, we describe two cases with cardiopulmonary complications during the ECT procedure; in both patients an underlying OSAS was diagnosed.

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2. Patients and methods

2.1. Patient 1

The first patient was a 52 year old man with a Body Mass Index (BMI) 28.4kg/m², suffering from bipolar affective disorder (BPAD). He had no other significant, known medical history. Because of a depressive episode, he was hospitalized at the 1st Department of Psychiatry of Eginition Hospital. He was under treatment with mirtazapine, quetiapine, venlafaxine, olanzapine, lorazepam and he was planned to follow a series of 12 ECTs.

The patient received intravenous diazepam and propofol, as anesthetic drugs, and immediately after the 2nd ECT was completed, while still in the period of semiconsciousness, he presented paradoxical motion of his chest and the upper airway collapsed. Because of respiratory failure - arterial blood gases, pH: 7.49, PaO₂: 69 mmHg, PaCO₂: 30 mmHg, HCO₃⁻: 22mmol/l, while breathing oxygen 10lt/min via a nasal cannula - he was intubated and transferred to the intensive care unit (ICU). After 15 hours in the ICU, the patient was medically stabilized and transferred back to the psychiatry unit. Hematologic and biochemical examinations were normal. Both, a cardiologic evaluation, including echocardiography and stress test and a pulmonary evaluation, including chest X-ray and spirometry, were unremarkable.

As the patient was overweight and reported snoring during sleep, he underwent standard full-night polysomnography performed in a quiet, sound and light proof room, with stable humidity and temperature. Polysomnographic findings - according to standard scoring criteria [7] - were as follows: time in bed 392min, sleep latency (SL) 13.2min, total sleep time (TST) 357min, sleep efficiency (SE) 91.1%; stage 1, 2min (7%), stage 2, 336min (94.1%) and rapid eye movement sleep (REM), 14min (3.9%) No slow wave sleep (SWS) was detected. The absolute number of respiratory events during sleep was 444 (73 central, 288 obstructive and 48mixed apneas, and 35 hypopneas) and the apnea hypopnea index (AHI) was 74.6 (severe OSAS), with a mean SaO₂89.4%, a minimum SaO₂72% and percentage of sleep time with SaO₂<90% (t < 90%) 48.8% (Fig. 1). Positive Airways Pressure (PAP) titration was carried out on a second night, normalizing the AHI at an inspiratory pressure (IPAP)17cmH₂O and an expiratory pressure (EPAP) 11cmH₂O (bi-level mode, BiPAP).

2.2. Patient 2

The second patient was a 55 year old man with a Body Mass Index (BMI) 31.5kg/m², suffering also from BPAD and OCD. He had no other significant, known medical history. Because of a depressive episode and obsessive thoughts, he was hospitalized at the 1st Department of Psychiatry of Eginition Hospital, under venlafaxine, olanzapine, sertraline, alprazolam and a plan to receive 10 ECTs.

The patient received intravenous diazepam and propofol, as anesthetic drugs, and 15min later, when the 8th ECT was completed, he developed severe hypertension (210/120 mmHg) and tachycardia (130 beats per minute) with mild desaturation. He was admitted to the recovery room and received oxygen (3lt/min via a nasal cannula) and amlodipine. After 1.5 hours in the recovery room that the patient was medically stabilized, he transferred back to the psychiatry unit. Hematologic and biochemical examinations were normal. A cardiologic evaluation, including echocardiography, stress test and 24-h ambulatory blood pressure monitoring revealed mild nocturnal hypertension. The pulmonary evaluation, including chest X-ray and spirometry, was normal.

As the patient was obese and had nocturnal hypertension, he underwent standard polysomnography performed in a quiet, sound and light proof room, with stable humidity and temperature.

Polysomnographic findings - according to standard scoring criteria [7] - were as follows: time in bed 195.9min, SL 14.2min, TST 140.5min, SE 91.1%; stage 1, 11.5min (8.2%) and stage 2, 129min (91.8%). No SWS and REM sleep were detected. The absolute number of respiratory events during sleep was 61 (2 central and 2 obstructive apneas, and 57 hypopneas) and the AHI was 26 (moderate OSAS), with a mean SaO₂95.4% and a minimum SaO₂ <90% (Fig. 2). Positive Airways Pressure (PAP) titration was carried out on a second night, normalizing the AHI at a range of pressures 5-10cmH₂O (auto mode, auto-CPAP).

3. Discussion

OSAS is being increasingly recognized as an important cause of medical morbidity and mortality. Polysomnography is the gold standard to diagnose sleep-disordered breathing [1].

Recent studies in general unselected populations, based on polysomnography data, assess a very high prevalence of the disease, ranging from 46,6 to 49,7% in men and 23,4 to 30,5% in women [2,3]. Although it is a relatively common sleep disorder, up to 80% of individuals with moderate to severe OSAS may remain undiagnosed and, more alarmingly, untreated [8]. Apneas and hypopneas occur during sleep and most patients are unaware of these events. A high degree of suspicion is mandatory to detect OSAS in patients with risk factors, symptoms, and typical physical findings.

People with serious mental illness (SMI), particularly MDD, have a high prevalence of OSAS, ranging from 13.9 to 42.4% in different clinical studies [9]. Higher frequencies of OSAS were seen in MDD (36.3%), than in BPAD (24.5%) and schizophrenia (15.4%) [9]. It is mandatory to recognize OSAS in patients with BPAD because disruption of the sleep-wake cycle caused by OSAS could possibly lead to mood instability and relapse of BPAD [10]. Furthermore, the treatments for BPAD (benzodiazepines and antipsychotics) may trigger respiratory failure in patients with an underlying, undiagnosed OSAS [4,5,10]. Unfortunately, existing data are characterized by considerable heterogeneity of the study population and a high risk of bias.

Although ECT procedure is quite safe, there is a need for sedation. Failure to diagnose and treat OSAS in ECT patients may expose them in anesthetic complications. Propofol affects breathing and upper airway (UA) patency by direct inhibition of the dilator muscle activity [11]. Also, supine position with decreased pulmonary reserve and loss of consciousness with reduced arousal during ECT, further increase the risk of UA collapse. In addition, benzodiazepines relax muscular tone, causing an increased risk of upper airway obstruction, and suppress the ventilatory response to hypercapnia and may even reduce the hypoxic response [4,5]. Taken together, all these parameters can potentially lead to apnea in an already unstable UA.

We believe that, regarding our first patient, a serious, sustained UA collapse was the reason of his inability to breathe.

The second patient seems to have had an episode of Negative Pressure Pulmonary Edema (NUPPE), which develops when a patient struggles to breathe against an obstructed airway during an UA collapse. In that case, very negative intrathoracic pressures are generated, which, can potentially lead to NUPPE or post-obstructive pulmonary edema, with severe hypertension, tachycardia and oxygen desaturation [12]. Resolution of this pulmonary edema is usually rapid, because alveolar fluid clearance mechanisms are intact.

Given that all other examination were unremarkable, we strongly hypothesize that in both our patients, the underlying pathophysiological mechanism for the serious complications during ECT was undiagnosed and untreated OSAS.

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