SLEEP APNEA: MANIFESTATION OR CAUSE OF SEIZURES IN CHILDREN? CASE REPORT

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ABSTRACT

Introduction. Sleep apnea (SA) in children is underdiagnosed. Compromising somatic development and alterated quality of life are frequently present. Central sleep apnea (CSA) may be the cause of convulsive episodes in sleep or could be their result. Polysomnography is the gold-standard of SA objective assessment.

Material and methods. We present a 7-year-old boy hospitalized in September 2013 for choking episodes occurred during sleep. The assessment was made by history, clinical examination, laboratory investigations (functional, imaging, biological) and interdisciplinary checkups.

Results. Repeated respiratory tract infection and a history of about 20 days of episodes of choking were established by anamnesis. Were diagnosed: underweight status; chronic rhinitis; tonsillar hypertrophy; obstructive ventilatory dysfunction; mixed sleep apnea with predominant central component; atopic status; convulsions. Imaging explorations revealed normal aspects. Complex hygienic-dietary and medical treatment has led to the disappearance of respiratory disorders during sleep, with normalization of spirometric parameters and nutritional status.

Conclusions. Polygraphic sleep study identified sleep apnea, indicating the predominance of the central component and facilitating etiologic diagnosis. Interdisciplinary management led to favorable evolution of the case.

Keywords: apnea, sleep, convulsion, child

Abbreviations

AHI	apnea-hypopnea index
BMI	body mass index
CNS	central nervous system
CSA	central sleep apnea
EEG	electroencephalogram
ENT	ear, nose and throat
GER	gastroesophageal reflux
OSA	obstructive sleep apnea
MRI	magnetic resonance imaging
SA	sleep apnea
SaO_2	arterial oxygen saturation

INTRODUCTION

Sleep disorders occur in about 30% of children. These can manifest as: sleep apnea, parasomnias (night terrors, enuresis, nocturnal seizures, dream anxiety, bruxism), etc. Sleep apnea (SA) occurs in 2-4% of children, is frequently underdiagnosed and is due to airway obstruction during sleep (obstructive apnea) and CNS's inability to optimal regulate breathing (central apnea). Sleep study (polysomnography) is the gold-standard of SA objective assessment.

CASE PRESENTATION

We present a male patient, aged 7 years, followed up in our department from September 2013. The patient came from a rural area, from a family with low socioeconomic level and limited access to medical services. He presented many upper respiratory tract infections since infancy. The reason for admission was the choking episodes occurred during sleep.The history of disease pleaced the onset of manifestations about three weeks before the presentation in emergency. Episodes occurred between

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23.00-24.00 or 04.00-06.00, with a duration of 10-20 seconds. Nocturnal bruxism is mentioned. For five days the child had runny nose and productive cough, for which he received mucolytic treatment and antihistamine in outpatient.

Physical examination on admission: patient with good status, appropriate appetite, afebrile, with weight deficit (weight = 19 kg, height = 120 cm, BMI = 13.2 kg /m^2, under the 5^{th} percentile for sex and age), respiratory rate = 20/minute; pulse = 88/minute; blood pressure = 105/65 mm Hg; bilateral bulbar conjunctival congestion, throat congestion, hypertrophied tonsils, multiple dental cavities.

Ophthalmological checkup identified acute conjunctivitis and mixed astigmatism. ENT evaluation identified chronic allergic rhinitis and tonsillar hypertrophy of second degree. Following investigations provided normal relations: cardio-pulmonary radiography; abdominal and cardiac ultrasound; electrocardiogram; Holter ECG recording; barite esogastroduodenal passage.

Spirometry revealed moderate obstructive ventilatory dysfunction.

Overnight polygraphy has identified mixed sleep apnea with the predominance of the central component, with apnea-hypopnea index of 3.5/hour, desaturation index 2/hour, average SaO_2 = 98%. (Fig. 1)

EEG performed in basal conditions recorded age appropriate background track. Hyperventilation causes the activation of slow medium voltage delta theta bursts, lasting 0.3-3 seconds, with persistence over 45 seconds after cessation of hyperventilation. (Fig. 2) Neurologic evaluation diagnosed convulsions.

Native brain MRI revealed structurally normal CNS. (Fig. 3)

Psychological assessment identified polymorphic dyslalia, harmonious cognitive and emotional development, proper for chronological age. Were not identified psychopathological aspects.

Biological exploration has highlighted atopic status: hyperimmunoglobulinemia E = 3345 IU/mL, specific antibodies to respiratory allergens (dust mites, dog and cat hair, Cladosporium herbarum) and food (cow's milk proteins, egg, casein, flour wheat, soybean).

With treatment (exclusion diet, antihistamine medication, inhaled fluticasone, leukotriene receptor antagonist, sodium valproate), the outcome was favorable. A single episode of apnea was documented during sleep by day. Duration and intensity of sleep disorders have declined to extinction during hospitalization. At home the patient presented only two brief and mild episodes nightly. He continued the therapy initiated in our service and the multidisciplinary follow up was started (pediatric, otolaryngology, pediatric neurology, etc.) Evaluations conducted during January-May 2014 confirmed the positive evolution of the case. The patient was asymptomatic, with improved nutritional status (weight = 22 kg, height = 122.4 cm, BMI = 14,7kg/m^2 at 26^{th} percentile for sex and age) and normal spirometric values.

DISCUSSION

The diagnosis oriented to establish the etiology of respiratory disorders during sleep in the presented patient discuss the following pathological:

– Sleep apnea
– Nocturnal gastroesophageal reflux
– Seizures in sleep

American Academy of Sleep Medicine has classified the sleep apnea as follows:

– Obstructive apnea
– Central apnea
– Mixed apnea
– Hypopnea (obstructive/central/mixed) (1).

Obstructive sleep apnea (OSA) affects 1-3% of non obese children younger than 8 years. The peak incidence is recorded in preschoolers (3-6 years). (2)

The etiology of OSA in children is complex. As risk factors are mentioned: adeno-tonsillar hypertrophy, atopy, asthma, gastroesophageal reflux. (3) The major components of OSA are episodic hypoxia, intermittent hypercapnia and sleep fragmentation. Obstructive sleep apnea (OSA) is the repeated interruption of breathing during sleep lasting more than 10 seconds or more than 5 to 10 episodes of hypopnea per night. Upper airway obstruction may be partial, prolonged or complete and intermittent. Ventilation during sleep is disturbed. OSA leads to repeated awakenings, impaired sleep quality and pathological events (cardiovascular, metabolic, neuropsychological, growth retardation, etc.) (2,3,4,5). Some common symptoms of OSA in children are: episodes of sleep apnea, frequent respiratory infections and growth failure. (4)

Polysomnography is the gold standard for diagnosis of respiratory disorders during sleep. (3,6) Polygraphic records demonstrate sleep apnea (SA) and its severity. The diagnosis of SA is specified when index apnea-hypopnea (AHI) is greater than 1/hour. Central sleep apnea (CSA) may present with sudden awakening accompanied by shortness of breath, seizures respectively.

[redacted] STARDUST Pg 1 of 1

Patient Name:	[redacted]		
Gender	M	Weight:	19 kg
Birth Date	8/19/2007	Height:	1.2 m
Patient Age	6 years	Body Mass Index:	13.2
Patient ID	5110826352143		
Study Number	4033		
Study Date	10/8/2013 at 10:56:07 PM	Device Serial Number	2000004466
Time in Bed (TIB)	546 minutes	Stardust Type	Stardust II

Dg: mixed moderate sleep apnea syndrome predominantly central, apnea-hypopnea index of 3.5/h, desaturation index 2/h, average saturation 98%. Moderate persistent asthma at onset with allergic component. Recommended Flixotide 2 x 50 mcg/day, Montelukast 5 mg 1 tablet/day, EEG. Control at one month possibly for CPAP titration.

	Code	Index (#hour)	Total Numbers of Events	Mean duration (sec.)	Max duration (sec.)	Events by Position	
						Supine (#)	Non – Supine (#)
Central Apneas	CA	2.1	19	12.9	20	2	17
Obstructive Apneas	OA	0.4	4	11.6	14.5	2	2
Mixed Aneas	MA	0.2	2	15.8	16	0	2
Hypopneas	HY	0.8	7	13.1	15	2	5
Total		3.5	32	13	20		
					Time in Position	19.4	526.6
					AHI in Position	18.6	3

Snoring

Total Snoring Event Flags	2
Snore Flags Index (#/hour)	0.2

Oximetry distribution

< 95% (minutes)	0
< 90% (minutes)	0
< 85% (minutes)	0
< 80% (minutes)	0
< 75% (minutes)	0
< 70% (minutes)	0
< 60% (minutes)	0
< 50% (minutes)	0
Total Dur (min) < 97	2.5
Average (%)	98
Desat Index (#/hour)	2
Desat Max (%)	7
Desat Max dur (sec)	16
Lowest SpO_2 (≥ 2 sec) (%)	83
# Episodes (≥ 5 min) ≤ 88%	0
Longest dur (min) SpO_2 < 88%	0

Heart Rate

Mean HR (BPM)	74.4
# of LHR	10
LHR min (BPM)	53
# of HHR	22
HHR max (BPM)	125

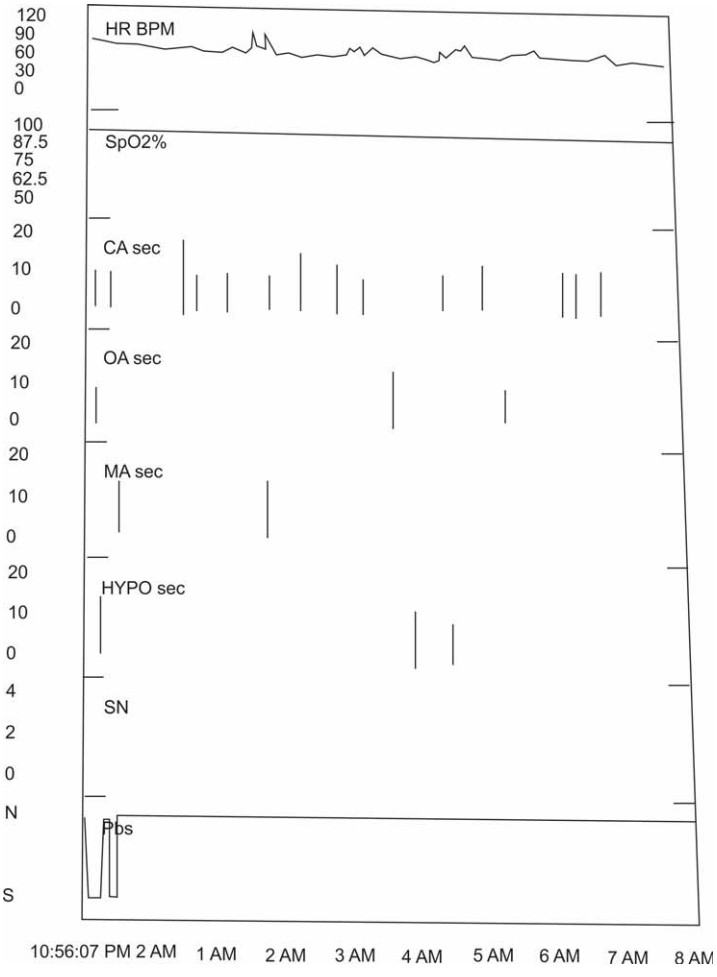

FIGURE 1. *Polygraphic sleep study: Sleep mixed apnea. B.I. male, aged 7 years*

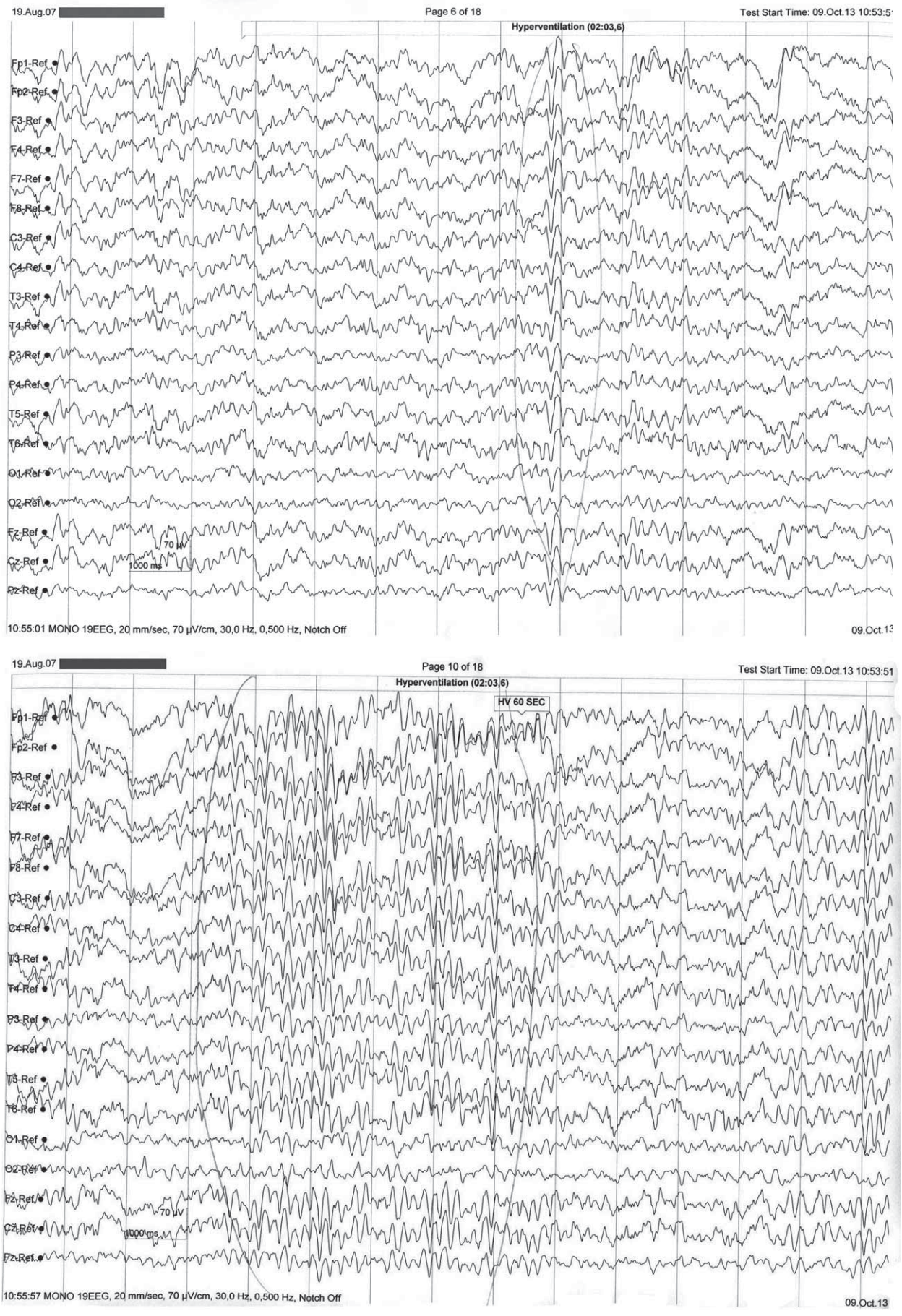

FIGURE 2. *Electroencephalogram: Convulsions. B.I. male, 7 years old*

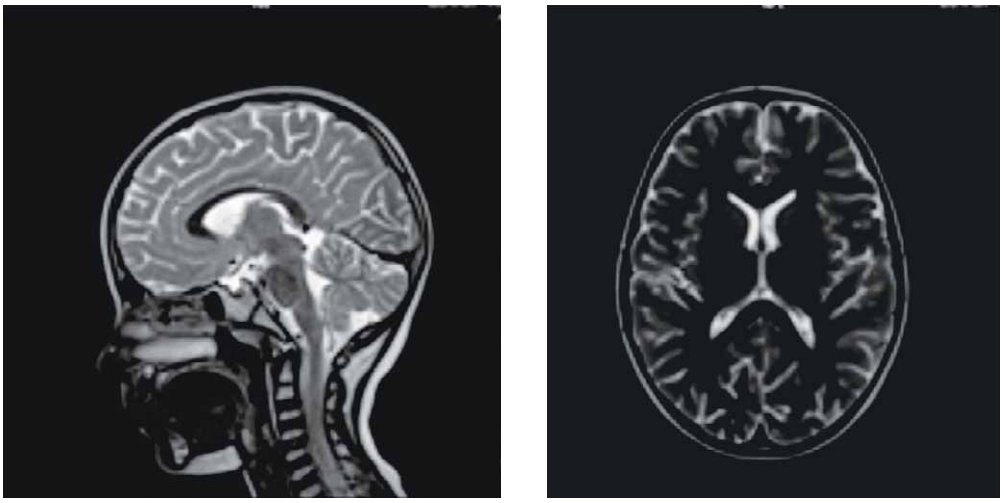

FIGURE 3. *Brain MRI: normal appearance. B.I. male, aged 7 years*

As risk factors are mentioned: male gender; cardiac pathology; CNS disorders (tumors, ischemia). CSA is characterized by inefficient/absent ventilation and compromised gas exchange. CSA and OSA may have common pathogenic mechanisms. (7)

Seizures during sleep may manifest with respiratory disorders (choking). Only 10% of cases of epilepsy have only nocturnal events. Sleep apnea can trigger nocturnal seizure. In sleep epilepsy paroxysms manifest during nocturnal or diurnal sleep, occurring frequently when falling asleep or at awakening. Epilepsy can induce central sleep apnea. 5% of patients with sleep apnea have epilepsy. (8)

In nocturnal manifestations of gastroesophageal reflux (GER) are involved aspiration of gastric content and vagal mediation. (9) Nocturnal episodes of RGE, frequently associated with acid reflux and awakenings, can lead to sleep apnea by spasms of the vocal cords. RGE in children can lead to upper airway edema and atypical clinical features: cough, recurrent and/or persistent wheezing, obstructive sleep apnea. (9,10)

Allergic rhinitis associated with tonsillar hypertrophy leads to upper airway obstruction, as a component of mixed sleep apnea in the presented case.

CONCLUSIONS

Polygraphic sleep study identified sleep apnea, indicating the predominance of the central component and facilitating etiologic diagnosis. Early diagnosis, appropriate treatment and interdisciplinary management led to positive ovolution of the case.

ACKNOWLEDGEMENT

We thank Assoc. Prof. Dr. Mihaicuta Dan Stefan, Dr. Roxana Tudorache and Prof. Dr. Maria Puiu for their valuable contribution to the assessment and monitoring of the case presented.

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