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Clinical case of treatment of obstructive sleep apnea/hypopnea syndrome in patient with bronchial asthma

key words: *apnea, bronchial asthma, sleep disorders, OSAHS*

Humans spend one third of their life in sleep. The daytime activity pattern of a person, as well as his/her mental and somatic health depend on the quality of sleep, its duration and possible disturbances.

The sleep respiratory impairments constitute a quite widespread pathology which, unfortunately, has not been studied much up until now. Since many diverse types of these impairments can be found among the population, to control them effectively it is necessary to improve the processes of diagnostics, treatment and prevention as they are found in modern medicine.

The respiratory impairments during sleep constitute a group of pathological conditions which pose an important and socially significant problem to the modern medicine. One of the variants of the problem, that occurs most often, is the Obstructive Sleep Apnoea – Hypopnoea Syndrome (hereinafter referred to as OSAHS). OSAHS is defined as a combination of an excessive daytime sleepiness and the respiratory impairments during sleep that are caused by the intermittent recurring episodes of the upper airways collapse. Apnoea constitute a complete falling of the upper airways with the 10 second long (or longer) periods during which the ventilation comes to a halt. Hypopnoea is a partial constriction of the upper airways' lumen with the 10 second long (or longer) periods during which the ventilation is reduced to 50 (or more) per cent of its normal capacity. The apnoea – hypopnoea episodes are considered obstructive if they do not inhibit the respiratory efforts. If no respiratory efforts are present, the apnoea – hypopnoea episodes are considered to be «central» [3, 4].

Based on the data of Wisconsin Sleep Cohort Study (2003), the prevalence of OSAHS among the general population is about 10–12%, and among the middle-aged persons (30–60 y.o.) the syndrome affects up to 24% of population. More than 40% of patients with OSAHS have concurrent pathology: ischemic heart disease, arterial hypertension, chronic obstructive pulmonary disease, bronchial asthma [11].

Patients with OSAHS experience the lowering of the throat muscle tone during their sleep which leads to the narrowing of the upper airways' lumen, to the airways partial or complete collapse and to the pulmonary ventilation arrest. Hypoxemia and hypercapnia result in the compensatory activation of the sympathoadrenal system and in the formation of intensified respiratory efforts aimed at the restoration of the airways' patency. These moments lead to the patient's transition to a more superficial sleep phase or to a complete awakening; after ventilation is restored, the process of blood reoxygenation takes place. Next the patient falls asleep again, muscular hypotonia sets in, the pathological circle closes. These phenomena can occur hundreds of times during the night which results in the derangement of the normal sleep structure, development of the daytime sleepiness, as well as the circulatory and hemodynamic disturbances, the oxidative stress and the systemic inflammatory response syndrome [2, 5].

OSAHS considerably lowers the life expectancy and the quality of life of the patients. Excessive daytime sleepiness, cognitive and neurotic disturbances, night snoring and sexual disturbances cause the patients to experience individual and social conflicts. The OSAHS patients constitute the high risk group as far as the traffic' and industrial traumatism are concerned [13].

OSAHS is characterized by the following risk factors: anatomical and functional disturbances (namely, a reduced caliber of upper airways), genetic predisposition, smoking,

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obesity, male sex, female sex in postmenopause, advanced age [9].

The somatic consequences of OSAHS are of the comparable significance; they are as follows: the cardiovascular (arterial hypertension, ischemic heart disease, circulatory insufficiency, rhythm disturbances, strokes) and severe metabolic disturbances (resistance to insulin and leptin, development of diabetes mellitus (type II), obesity) [2. 6].

Combination of OSAHS and bronchial asthma in one patient reciprocally complicates the course of these nosologies. The frequency of OSAHS in cases of bronchial asthma amounts to around 8–20%. Even a healthy person experiences the reduced ventilation and respiratory stimuli' perceptibility during sleep, but in a bronchial asthma patient these phenomena can lead to dramatic consequences: hypoxemia, hypercapnia, pulmonary hypertension and a high risk of nighttime death. The severity of the combined pathology is conditioned by the impossibility of reaching an adequate re-saturation level after apnoea in the lungs of a bronchial asthma patient, as compared to healthy lungs, as well as by an insufficient ventilatory response due to the weakness of the muscles of respiration and the respiratory drive depression [1, 10].

The method of polysomnography is the commonly recognized OSAHS diagnostics standard; this method provides for the simultaneous registration of the patient's parameters of sleep, breathing pattern, parameters of cardiac activity and of the blood oxygenation, as well as of the lower limb movements.

The polysomnography is conducted in specially equipped sleep laboratories with a laboratory assistant constantly present; the laboratory assistant controls the technical adequacy of the study and the patient's behavior, with the subsequent interpretation of the results by physicians trained in sleep medicine [14].

OSAHS should be defined as the condition during which Apnoea–Hypopnoea Index ≥ 15 episodes per hour, or when at the value of Apnoea–Hypopnoea Index varying from 5 to 14 episodes per hour the daytime sleepiness, the consciousness' or mood impairments, the history of arterial hypertension, ischemic heart disease or acute cerebrovascular disease have been documented [12].

The non-invasive positive pressure ventilation is the most effective method of OSAHS treatment. Based on the conclusions of the American Academy of Sleep Medicine, the positive airway pressure (PAP) is the treatment of choice for all OSAHS forms and should be offered to every OSAHS patient [7, 8].

In the department of diagnostic, therapy and clinical pharmacology of lung diseases State organization «National institute of Phthisiology and Pulmonology named after F.G. Yanovsky AMS of Ukraine successfully works polysomnography laboratory, which is equipped not only diagnostic devices, but the latest modifications CPAP- devices (continious positive airway pressure) and BiPAP- (bilevel positive airway pressure) therapy. Based on laboratory diagnosis is made and treatment selection for patients with SOAHS, including the combination of his asthma, COPD, hypoventilation syndrome.

Here we present a clinical case based on our own observations (in the process of work the State budget funds have been used).

Patient L., 1949 BC. admitted to the hospital complaining of significant daytime sleepiness and fatigue, headache, disturbed night's sleep with frequent awakenings, dry mouth upon awakening, dry cough, shortness of breath at rest, aggravated by exertion. There were also short of falling asleep while performing monotonous work and driving (past three years, do not drive). Relatives worried sick about her in the presence of loud snoring and pauses in breathing during sleep. On a scale Epworth Sleepiness Scale daytime sleepiness patient was assessed as severe (24 points) at a rate of up to 10 points. He considers himself a patient for 8 years, when the snoring, sleep disturbance, fatigue and daytime sleepiness. Also, the patient complained of asthma attacks 1–2 times a week during the day and 2–3 times a month at night, morning stiffness in the chest (asthma control test (ACT) – 21 points), episodes of high blood pressure and pain in the area heart.

The patient non-smoker and never-smoker. From the history of suffering from asthma for 27 years. Diagnosis: bronchial asthma, persistent, III severity, partially controlled. Comorbidity: hypertension Renoparenhimna II (2) stage 3 degrees. Hypertensive heart. Ventricular fibrillation extrasystolic. CH II A (2) c, III (3) functional class. NYHA with preserved left ventricular systolic function. 4. CKD Risk: polycystic kidney disease II (2) Art. CRI I. Pulmonary heart. Pulmonary hypertension secondary.

Accepts basic therapy of asthma: Seretide 50/500 micrograms 2 times a day to stop asthma symptoms salbutamol 2 inspiration is stated when needed. Regarding comorbidity patient gets treatment – valsakor 320 mg/day, tryfas 5 mg/day, preductal 75 mg/day, amlodipine 10 mg/day, koriol 12.5 mg 2 times a day, veroshpiron 50 mg 1 time a day, atoris 10 mg per day.

Objectively: general state of moderate severity. Anthropometric data indicate a high risk of developing SOAHS: height – 165 cm, weight – 115 kg, body mass index – 42 kg/cm2, the circumference of the neck – 43 cm, heart rate – 87 per min., BH – 18 per min., AT 150/90 mm.

On radiographs – chronic bronchitis symptoms, ECG – sinus rhythm, signs right and left heart overload.

According to research on spirographic curve «flow-volume» forced expiratory volume and total bodyplethysmography on the machine «Master Screen PFT» company «Cardinal Health» (Germany) FEV1 was 44.5%, reversibility of bronchial obstruction – 13.2%.

Conducted determine the strength of respiratory muscles (PImax and PEmax) and neyrorespiratornoho drive (P0,1), on the unit «MasterScreen-PFT» company «Cardinal Health». Decreased strength of respiratory muscles both insufficiently (PImax (38%); PEmax (45%)), a slight increase neurorespiratory drive (246%), which confirms the severity of the disease.

Identified mixed (obstructive-restrictive) disturbances in respiratory function associated not only with bronchial obstruction, but with obesity.

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Even in the absence of primary lung pathology the obesity, especially abdominal type, has profound pathophysiological effects on the respiratory system, disrupting the ventilation function, respiratory mechanics, strength and endurance of the respiratory muscles, gas exchange, control breathing, exercise tolerance, leading eventually to respiratory failure. The direct impact of obesity on the physiology of breathing – a mass increase and decrease compliance of the walls of the chest with fat deposits around the edges, and the consequent difficulty in getting chest on inspiration. The deposition of fat in the mediastinum limits the mobility of the lungs. When excess fat deposits in the abdomen develops diaphragm dysfunction, which is the imbalance ratio length / tension of muscle fibers due to hyperextension, limiting diaphragm excursion. The accumulation of fat in the airways gives the ability to maintain normal cartilage rings airway clearance.

Obesity reduced the lung volumes, especially expiratory reserve volume and functional reserve capacity, which play an important role in maintaining patency of the distal airways. By reducing the amount of closing reserve volume below is the collapse of the alveoli and microatelectasis development. Thus, obesity combined two options disorders of respiratory function – this restriction (reducing lung volumes) and obstruction (narrowing of the distal airways).

Along with violation of compliance chest reduced elasticity of the lung tissue by increasing the blood supply vessels of the lungs, increased airway resistance, collapse of distal airways.

To overcome the rigidity of the chest and airway resistance consumes additional energy, increasing the work of breathing, growing fatigue and weakness of the respiratory muscles. This creates an imbalance between the requirements of the respiratory muscles and its productivity, which leads to a feeling of breathlessness.

With obesity formed a special pattern of frequent and shallow breathing. This increases the proportion of ventilation «dead space» in minute volume ventilation reduces the effectiveness of alveolar ventilation.

Obesity profoundly violates the mechanical properties of breathing in asthma. Reducing the excursion of the diaphragm and respiratory volume reduces the ability to maintain airway passage, leading to increased contractility of the smooth muscle of the bronchi and increased bronchial hyperreactivity because of narrow airways and bronhoprotektivnoho no effect on deep breathing [6].

The patient was conducted daily monitoring of ECG (EC-2H, Labtech), blood pressure (ABMP04, Meditech). The values of blood pressure averaged 190/99 mm Hg. Art., the maximum reported BP 233/131 mm Hg. Art., elevated levels of daily index (DI - 35%) at a rate of 10–20% (characterizes the level of blood pressure reduction at night), the index time (Hldx – 85%) at a rate of 15–20% (indicates the number of blood pressure measurements exceeding normal levels). When Holter recorded lower overall heart rate variability (SDNN – 50 ms), at a rate 96–162ms, indicating increased activity of the sympathetic division of the autonomic nervous system (ANS), especially at night, the presence of supraventricular beats (CE) – 1,364 per night, at a rate to 100 beats ventricular (st) – 125 per day, at a rate of 100. Average heart rate was – for 83 minutes.

In the biochemical analysis of blood – increased levels of total cholesterol to 6.2 mmol/L (normal 5.2 mmol/L), triglycerides up to 2.4 mmol/L (normal 1.8 mmol/l), and other general settings blood and urine – no pathology.

The patient performed night polysomnography study (PSG), the results of which revealed significant changes of UGS: the index of apnea/hypopnea (IAH) – 68.3/h, 22.2 apnea index/h average night saturation of 57.0%, 39.0% minimum saturation index desaturatsiyi 100.0/h. The patient exhibited diagnosis – SOAHS, grave severity.

Given the severe degree of sleep disorders and the presence of comorbidities, patient auxiliary ventilation recommended therapy with BiPAP.

The first night BiPAP treatment was performed in a laboratory to determine the level of sleep pressure and therapeutic monitoring the effectiveness and tolerability of treatment [15].

After the first night with the medical device PAP patient noticed a good sleep, feeling good holiday. According to the results of the polysomnography while sleeping with the device found ViPAP improve sleep disturbed options: normalization index apnea / hypopnea (IAH) to 0.7/h, apnea index to 0/h mean night saturation to 72.0%, the minimum saturation 60%, to 28.3 index desaturation 28.3/h. So this mode of ventilation support considered adequate for the patient. The patient for 10 sessions of treatment is BiPAP.

After 10 days the patient's condition improved significantly, decreased fatigue, decreased daytime sleepiness, episodes of sleep during the day, snoring, headaches. Decreased dyspnea, cough disappeared, and dry mouth. The general condition of the patient is satisfactory. HR for 74 min., BP 140/85 mm Hg. Art., RR19 in min. On a scale Epworth Sleepiness Scale level of daytime sleepiness in a patient decreased from 24 points to 11 points.

The evolution of the PSG continued to improve after 10 sessions of treatment whis BiPAP (table 1).

Compared with the results of the first diagnostic PSG showed a reduction in respiratory distress index to 0.7/h, apnea index to 0.3/h, desaturation index to 7.2% overnight.

The patient is recommended to keep a basic asthma medication and spending PAP-therapy on an ongoing basis.

After 10 sessions of therapy BiPAP positive trend observed symptoms of asthma: the number of asthma attacks, decreased cough (ACT 17 points).

The evolution of the PFT before and after treatment presented in Table. 2. There was a general decrease in airway resistance (Rtot) from 270.1% to 78.4%, residual volume (RV) from 145.9% to 121.9%, increase in total lung capacity (VC) from 47.6% to 90.9% increase in FEV1 of 44.5% to 62.3% and FVC from 49.6% to 68.4% inspiratory capacity (IC) from 44.4% to 71.2% and the flow rate at the level of small bronchi (FEF 25) from 28,8 to 66,6, (FEF 50) of 28,6 to 62,0 (FEF 75) from 44.3 to 76.1.

When conducting repeated daily monitoring of blood pressure and ECG average daily blood pressure decreased, and reached 163/88 mm Hg. Maximum BP recorded a day – 180/95 mmHg. Art. and average heart rate

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Table 1
Dinamic of PSG results before and the end of treament

Sings	Before treatment	After the 1th night treatment	After the 10 days treatment
Desaturation index, h	100.0	28.3	7.2
SaO2 Mean,%	57,0	72,0	86,0
SaO2 Minimum,%	39,0	71,0	80,2
Basal Heart Rate, /min	92	88	79
Desorder of breath, /h	68,3	0,3	0,7
Apnea Index, /h	22,2	0,0	0,3
Hypopnea index, /h	46,1	0,0	0,7
Total apnea, /night			
-obstructive	82	10	8
- mixt	40	9	2
- central	53	11	2

Table 2
Dinamic of PFT results before and the end of treament

Sings	Before treatment		After the 10 days treatment	
	Absolute rate	% of due	Absolute rate	% of due
R tot, kPa*s/L	1,01	270,1	0,24	78,4
IC, L	0,98	44,4	1,58	71,2
ITGV, L	3,40	121,3	3,56	126,9
RV, L	2,96	145,9	2,48	121,9
TLC, L	4,39	83,8	5,19	99,3
VC MAX, L	1,42	47,6	2,72	90,9
FEV_1, L	1,08	44,5	1,32	62,3
FVC, L	1,42	49,6	1,72	68,4
FEV_1/ FVC,%		75,74		76,7
FEF 25, L/s	0,37	28,8	0,86	66,6
FEF 50, L/s	1,05	28,6	2,28	62,0
FEF 75, L/s	2,39	44,3	4,11	76,1
PEF, L/s	3,73	60,3	5,51	89,2

was 73 beats/min. Has also decreased levels (DI - 18%), Hldx – 66%, SDNN – 80 ms, SE – 550/day, st – 22/day, indicating a decrease in blood pressure, lower blood pressure at night, improve the function of the autonomic nervous system (increased activity of the parasympathetic ANS love night), reduction of arrhythmia. An objective examination revealed no pathology.

Given the need for continuous BiPAP therapy patient invited to lengthy observation at the Institute. It was recommended to advise the patient 1 month after initiation of therapy and thereafter consult it every year.

The purpose of consultations after 1 month of treatment is to control treatment effectiveness and patient adherence to therapy. Treatment positively with good patient adherence to therapy, no unwanted displays of therapy, no complaints (snoring, daytime sleepiness), improving the patient's quality of life. To evaluate the success of therapy anamnesis find out with an emphasis on SOAHS symptoms, sleepiness assessed on a scale Epworth, analyze information chip electronic medical equipment, and if necessary, carry PSG. If unsatisfactory evaluation the correction of therapy provided. If a patient complains of daytime sleepiness and has other clinical symptoms SOAHS and counter treatment duration indicates insufficient average length of treatment overnight (<5 hours), the therapeutic strategy should be aimed at improving patient adherence to CPAP therapy. If in this situation the meter indicates a high average duration of treatment (> 6 hours), it is necessary to control to test PSG, whether established therapeutic regime CPAP therapy and, if necessary, its correction.

After a month of treatment the patient had not complaints of snoring and excessive daytime sleepiness, sleepiness on a scale Epworth was 7 points. There was a decrease in body weight of 7 kg per month. Undesirable manifestations therapy is not noted, was not the need to conduct PSG. The patient used the device BiPAP therapy each night, on average 5–6 hours a night. Treatment regarded as successful and the patient was invited to annual surveillance visits.

Table 3
Dinamic of arterial pressure and ECG results before and the end of treament

Sings	Before treatment	After the 10 days treatment
	% of due	% of due
AP middle	190/99	163/88
AP max	233/131	180/95
DI%	-35	-18
Hldx% per night	85	66
SDNN ms	50	80
ШЕ per day	125	22
CE per day	1364	550

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Annual consultations up procedure performed after 1 month of therapy, and monitoring changes in body weight, blood pressure. Also, specify the presence of proper maintenance of medical equipment and accessories. While maintaining persistent excess body weight patient recommend more aggressive mode to reduce body weight. When weight loss browsing mode CPAP therapy.

Thus, the BiPAP therapy creates the basis for the complete normalization of the sleep respiratory impairments caused by the upper airways collapse and relieves the bronchial asthma course, improves the functional indicators and lowers the level of cardiovascular morbidity and the risk of life-threatening arrhythmias. Most of the chronic pathological states require the use of constant background therapies, so does the OSAHS treatment, too, which foresees the use of CPAP therapy devices (or their modifications) every night (or almost every night – at least three times a week) accompanied by a periodical doctor advice.

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