DISORDERS OF THE UPPER AND LOWER RESPIRATORY TRACT UPON EXPOSURE TO OCCUPATIONAL RISK FACTORS

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Abstract: *The hereby article displays the aspects of the upper respiratory tract pathology determined by the prolonged exposure to various occupational hazards an highlights the importance of an efficient collaboration between the ENT physician and the occupational medicine specialist. Early diagnosis of respiratory tract disorders and their identification in the clinical systemic context of the occupational hazards contributes to the implementation of prevention measures and treatments capable to increase the quality of life.*

INTRODUCTION

In the process of industrialization and especially in the evolution and expansion of technological processes, various chemical substances have appeared that increased the toxic potential affecting the entire human organism and contributed to the exposure of the respiratory system to different occupational hazards with unwanted repercussions on the airways.

In this context, one can take into consideration the following chronic disorders: rhinitis, sinusitis, pharyngitis, laryngitis, bronchitis and other effects such as anosmia, ulceration and trophic nasal septal perforation.

The airways are easily penetrated by gases, vapors and toxic dusts, thus being the most exposed to danger, mainly because: the contact surface with the toxic substances is very large, approximately 100 m^2 in deep breathing; the pulmonary capillary network is almost 2000 km long; the alveolar capillary membrane thickness is of approximately 0.8u; the toxin enters rapidly and directly into the general circulation and it can spread throughout the entire organism in almost 23 seconds.

The action of gases and vapors in the respiratory system depends on two factors: solubility in water and lipids, and the chemical reactivity of the toxin. Hydrosolubility favors the infiltration at the level of upper airway, while liposolubility aims at the lower airway and alveoli, by infiltrating the lipoprotein cellular membrane. Hydrosolubility facilitates the absorption within the bloodstream of mostly inert gases and vapors.

The distribution ratio between the arterial blood and alveolar air depends on the degree of hydrosolubility, according to the Henderson-Haggard law. Highly reactive, hydro soluble (irritating, acids and bases) substances affect especially the airways without influencing blood saturation. Besides hydrosolubility and reactivity, the toxic particles can also be subject to pinocytosis.

The dynamics of the toxins that penetrate the organism through the airway is also influenced by factors as the size of the individual, the intensity of pulmonary ventilation (increased in case of physical effort), the condition of pulmonary circulation.(1,2)

Chronic rhinitis occurs in the chronic intoxication with chrome, nickel, gases and irritating vapors, together with their toxic compounds. Patients have nasal obstruction which causes the nasal voice, breathing through mouth and sometimes the snoring during sleep, especially if lying in decubitus position. Most of the time, the nasal obstruction occurs alternatively in each nasal fossa, so that when the patient is lying in lateral decubitus, the obstruction installs in the declive fossa.

The discharge of the nasal secretions in the rhinopharynx determines the patient to make effort in removing debris from within the nose, action that can develop into a permanent repetitive behavior accompanied by difficult dry swallowing.

Patients also may complain from hyposmia or even anosmia, headache, fatigue, restless sleep, and various disorders of aero digestive tract.

The clinical examination reveals the presence of filamentous mucous especially in the lower floor of the nasal fossa, situated between the turbinates and the septum. In some cases, mucopurulent secretions may appear covering the inferior turbinate or the floor of the nasal cavity. The pituitary mucosa is hyperemic, sometimes purplish, edematous or pale.(2,3,4)

The symptomatology of the chronic hypertrophic rhinitis is characterized by difficult nasal breathing, with alternation from one fossa to another during the day, condition known as bilateral hypertrophic chronic rhinitis. The nasal obstruction becomes permanent and it is accompanied by rhinolalia, hypoacusis, and reflexive headache of nasal origin, epiphora, conjunctivitis, dacryocystitis, and fatigue.

The hypertrophy of the turbinates and especially of the inferior one is the most common localization. Breathing becomes difficult and accompanied by closed rhinolalia, especially during the exhale, as the patient has the feeling of a retronasal obstruction. The surface of the turbinates is muriform or cauliflower-like, thus obstructing the concha.

Chronic rhinitis is characterized by symptoms of respiratory discomfort. Although the lumen of the nasal fossa is widened, the patient has the sensation of a dry pituitary mucosa and rhinogenic headache. The mucosa of the nasal fossa is pale, dry and occasionally covered with mucopurulent mucus or small crusts.

Local symptoms in maxillary chronic sinusitis consist in nasal obstruction caused by congestion and thickening of the

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mucosa, pus, and polypoid neoformations, purulent rhinorrhea, cacosmia, sensation of intranasal or perinasal fullness, and spontaneous pain. Patients breathe through their mouth, and experience cough and dryness of throat.(2,4)

Purulent discharges are visible within the middle meatus of the nasal fossa. Sometimes polyp like formations can be observed, as well as the hypertrophy of the inferior turbinate and the head of the middle turbinate, the edema of the mucosa within the middle meatus with an aspect of duplication.

Among the general symptoms, the patient often complains about headaches when bending the head, when coughing, sneezing or nose blowing, and also about craniocervical and facial neuralgia.(3)

The symptoms of the frontal chronic sinusitis are the following: frontal orbital pain with paroxysms localized in the superolateral internal orbit, with radiation in the supraorbital region, temporal region and the hemicranias on the specific part, and diffuse chronic congestion of ocular conjunctiva.

The clinical picture of the posterior chronic sinusitis depends on the presence or absence of sinus and nasal communication, which allows the differentiation between open or closed ethmoid sphenoid sinusitis.(4)

The open chronic ethmoid sphenoiditis is characterized by intense retro-ocular headache radiating in the vertex, back of the neck and mastoids, sometimes associated with dizziness cause by position changes.

There also appears diffuse congestion at the level of superior meatus, on the end of the middle turbinate and on the ceiling of the cavum.

The closed ethmoid sphenoiditis is characterized by ocular and neurological disorders. Patients complain about maxillary neuralgia with increased suborbital, dental or various unspecific pains, accompanied by burning sensations, tingling, tearing, nasal hydrorrhea, and sneezing.(3,4,5)

Ulceration and perforation of the nasal septum is common in chronic intoxication with chrome, nickel, arsenic and their toxic compounds. The clinical picture in the initial stage is discreet and it may go unnoticed, except for the dryness and local discomfort due to crusts on the margins of the ulceration. Their elimination causes mild epistaxis or secondary infections that lead to the increase of the perforation.

The anterior rhinoscopy reveals the oval or round shaped lesion situated in the anterior inferior part of the cartilaginous septum. In the initial phase, the lesion in characterized by the presence of erosion in the pituitary membrane; ulceration gradually follows, that exposes the cartilage and becomes covered with sanguine crusts that bleed when detached. At first, the margins of the lesion are unhealed and slightly bleeding, covered with hematic crusts and mucus. They progressively become smooth and scarred. Ulcerations caused by chrome intoxication are deep with high margins.(3,1,5)

Chronic laryngitis occurs in the intoxication with nickel and its toxic compounds, gases, irritating vapors. Specific functional symptoms are dominant in the clinical picture, such as chronic dysphonia and coughing. Vocal cords appear pinkish-pale in the chronic catarrhal laryngitis.

In the chronic hypertrophic laryngitis, the vocal cords are thickened and have the aspect of pachydermia monocorditis.

In another form of hypertrophic corditis, the lesions are merging at the level of vocal apophysis. Atrophic laryngitis shows crusts that cause suffocation bouts and burning sensations. In these cases, the vocal cord mucosa is dry and reddish.(3)

At the level of the pharynx, chrome, arsenic and their toxic compounds, together with gases and irritating vapors cause the diffuse congestion of mucosa and sometimes of the uvula.

The patient often complains about burning sensations or dryness and discomfort on deglutition. The dryness sensation is caused by the decreased amount of saliva.

Sometimes, the erythema is accompanied by discreet edema of uvula, tongue and jugal mucosa that can be observed in the prints of teeth on the margins of the tongue. Arsenic exposure proved to cause vesicular eruptions.

In the lower part of the air waves, chronic intoxications with cadmium and its compounds, chrome and its compounds, phenols, sulfuric acid and hydrochloric acid can trigger chronic bronchitis. At first, patients experience coughing most commonly in the morning, when one does their bronchial toilet and gradually the coughing will occur during the day and in the evening. The disease is usually manifested by progressive dyspnea, prolonged exhale with forced elimination of vital capacity, numerous diffuse sibilant rales and bilateral wheezing (obstruction of the large bronchi), fine sibilant rales in exhale and dry crackles in the first part of inhaling or exhaling (obstruction of the small bronchi), and signs of hyperinflation (diminished breathing, hypersonority, and distant vesicular murmur).(3,4,5)

Occupational bronchial asthma may occur due to exposure to a series of chemical products, vegetal substances or substances of human or animal nature.

The most common chemical substances related to the occurrence of occupational bronchial asthma are chlorine and its compounds, formalin, phosphorus and its compounds, cadmium and its compounds, mercury and its compounds, nickel and its compounds, organophosphorus substances, and so on.(5)

In the asthmatic crisis, the patients experience anxiety and severe dyspnea caused by the edema of the bronchial mucosa, mucus plugs and the resistance of the smooth bronchial muscles. The asthmatic crisis can vary in duration and intensity, from mild coughing episodes that last for a few minutes, to bouts of dyspnea with wheezing that can go on for hours and can stop spontaneously or by means of bronchodilator medication. In between seizures, the patient is in good condition, with no respiratory distress, and he or she can function normally.(1,4)

The clinical examination of patients with asthmatic crisis will reveal typical elements such as: shortness of breath, respiratory frequency of 20-30 breaths per minute, and coughing.

Thoracic examination will expose the use of the accessory muscles during the breathing process, a hyperinflated thorax with relatively limited respiratory ampliation and prolonged exhaling.

The percussion method will show diffuse hypersonority, while the auscultation during a prolonged exhaling will reveal musical sibilant rales, inspiratory or expiratory, which are often noticed by the patients themselves and which cause the characteristic aspect of whistling breathing.(4)

During the asthma attack, the patients are afebrile, tachycardic (an average of 90-100 beats/minute) and with no significant TA variations.

The termination of an asthmatic crisis is progressive, after a few tens of minutes, usually after the administration of bronchodilator medication, or rarely enough spontaneously. Dyspnea decreases gradually, the wheezing becomes less perceptible, and the cough becomes productive. The phlegm is mucous, often in the form of plugs, and it contains Curshmann spirals, eosinophils, and crystals.(1,2)

Pulmonary carcinoma occurs as a result of exposure to a multitude of occupational factors: asbestos, asphalt,

benzopireny, cadmium and compounds, hexavalent chromium, nickel, lead, ionizing radiations, mineral oils, and so on.(5)

In the case of pulmonary carcinoma, the clinical manifestations are of great diversity in relation to the anatomo-clinical forms and the stage of the disease.

By the time the pulmonary symptomatology – extra pulmonary or general – appears, the disease is already in an advanced stage, with a highly probable tumor development in the next years.

Cough is usually the initial symptom, more exactly a persistent drug resistant cough with mucous phlegm. In the late stages, the cough is irritating, persistent and often caused by a pleural effusion or a bronchial or mediastinal compression.

Frequent hemoptysis in small amounts is a suggestive form of pulmonary carcinoma. Almost always the small hemoptysis results from the ulceration of the bronchial mucosa, while the most significant hemoptysis is the result of bronchial artery erosion.(2,4)

Dyspnea is an unspecific symptom and also a tardive manifestation. It may be the result of an obstruction or compression of large bronchia or of a pleural or mediastinal compression syndrome.

Localized wheezing, accompanied or not by paroxysmal coughing and which does not disappear after the coughing, could be a sign of a recent bronchial obstruction, often of tumor origin.

Respiratory symptoms are usually associated with localized chest pain, dysphonia, asthenia, anorexia, feverishness, which are typical signs of advances neoplasia.(1,4)

The respiratory manifestations are the most frequent, but unfortunately are unspecific and misleading.

CONCLUSIONS

The relationship between the ENT physician and the occupational medicine specialist has a crucial role in the early detection of occupational hazards with clinical manifestation in otorhinolaryngology, as well as in the process of establishing a diagnosis that will reflect the associated ENT pathology.

Various toxic pathogen agents caused by exposure to different occupational hazards penetrate the human organism through the respiratory system.

Of primary importance in maintaining the homeostasis of respiratory tract is the integrity of respiratory mucosa. Any disorder that might occur at this level, in the context of exposure to toxic substances, will eventually lead to the above mentioned pathology, which triggers the decrease of the quality of life of the affected individuals.

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