

# PATHOGENESIS AND HISTOPATHOLOGY OF VIRAL AIRWAY'S INFECTIONS

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**Abstract:** In the paper the author reviews the several elements of the pathogenesis and histopathology of viral infections of airways. Viral infections are important in morbidity, economic costs as well as the repercussions over the body. The pathogenic factors of virus infections are represented by entering the host body and their attaching to specific receptors on the airways, followed by multiplication and dissemination in the host organism.

**Cuvinte cheie:** căi respiratorii, infecții virale, patogenie, histopatologie

**Rezumat:** În lucrarea de față autorul trece în revistă câteva elemente legate de patogenia și histopatologia infecțiilor virale de căi respiratorii. Infecțiile virale sunt importante prin morbiditate, costuri economice ridicate ca și prin repercursiunile asupra organismului. Factorii de patogenitate a infecțiilor virale sunt reprezentați de pătrunderea în organismul gazdă și atașarea de receptorii specifici de la nivelul căilor respiratorii a virusurilor, urmată de multiplicarea și diseminarea acestora în organismul gazdă.

## INTRODUCTION

Viral infections are important in morbidity, economic costs as well as the repercussions over the body. Respiratory tract infections mainly aims extremes of age (newborn, infant and toddler) affecting immunity and favoring bacterial superinfections and the development of sequelae. The pathogenic factors of virus infections are represented by entering the host body and their attaching to specific receptors on the airways, followed by multiplication and dissemination in the host organism.

Regardless of the histopathologic findings, viruses with respiratory tropism presents 3 features: - ability to avoid the mucociliare barrier and other respiratory defensive means;

- the effect on flora ubiquitous that inhabit the upper respiratory system: respiratory viral infections creates imbalances in bacterial colonization, reduces clearance and phagocytosis mucociliar and increase bacterial adherence to respiratory epithelium;
- the same virus can cause different clinical syndromes, also the same clinical syndrome may be caused by many viruses.

Pathogenesis of viral infections includes irritation of infective process and mechanisms wich leads to the installation of signs and symptoms of infection. Interaction between microorganism and - host depends on the micro-organism's ability to inoculate itself in macroorganism and cause, lesions as well as the neutralization of its defense mechanisms. Thus, in the pathogenesis of viral infection occurs 3 "players": the virus, the macroorganism and the environment. The virulence of the virus is determined by a complex of characters that promote infection, multiplication and initiation of specific lesions on the respiratory tract of the host.

Penetration of infectious agents to the lungs is often about as air-borne microorganisms present in the inspired air particles or microorganisms that form initially localized in the nasopharynx and / or mouth of which is then sucked from the

lungs. In excess of 6 micron particles are retained on the nose and then transported to pharynx and swallowed without exceed bronchiolar. The particles with diameter less than 2 microns can, in turn, extend to the alveolar level.

The nose is the initial portion of the airways, and one of the most richly vascular organ. Working in close unity with other segments of the respiratory tract, it's the first defeding line of the host performing several roles:

- heat and humidify inspired air;
- purifies the air inspired by foreign particles;
- is the first filter of respiratory tract pathogens.

Air cleaning is taking place in chile after surgery nasal vestibule (retain particles > 15  $\mu$ m), the muco-ciliary clearance (retain particles > 10  $\mu$ m) and the cilia (those from the earlier portion of the nasal passages directs foreign particles to the outside, and cilia located posterior to the pharynx mobilizes antigenic particles), thus ensuring two-way drainage.

Layer of mucus that cover respiratory nasal epithelium is double layered: outer layer with a texture of "gel" which retains solid particles and an internal layer "watery". Foreign particles retained by the outer layer of mucus, under the action of rhythmic movement of cilia in nasal passages, watery slides on the internal layer. In case of damage to the mucous barrier, the filter of foreign particles is altered and negatively influences the optimal functioning of other segments of the airways (eg alteration of muco-ciliary system causes sinusitis).

We present further some features of respiratory infections in the respiratory tract depending on the segment concerned.

1. Rhinosinusitis or "common cold" is a self-limiting syndrome. Viruses that determine rhinosinusitis are in 5 families: myxoviruses, paramyxoviruses, adenoviruses, picornaviruses and coronaviruses, the incidence being maximum in winter season. Respiratory viruses invades nasal mucosa and causes stimulation of mucus secretion from epithelial cells containing viral antigen. Viral

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- infection induces vasodilation and edema of respiratory submucosa, with infiltration of mononuclear cells and polymorphonuclear cells respectively. Superficial epithelial cells are exfoliated and eliminated while an increased production of mucus. Inflammatory response within the nasal mucosa occurs in 2-4 days. Structural and functional changes in the ciliary equipment cause altering the clearance of mucus. The body reacts by producing antibodies specific IgA (local) and IgG (serum) thus contributing to limiting viral replication.
- Denudation and edema occurs in tracheobronchial disease with impaired transport of mucus. The resulted bronchoconstriction is due to the hiperreactivity of the hiperreactivity of the reflex arc and the vague nerve with the clinical consequence cough (dry or productive). Also takes place a partial dysfunctionality of the ciliary cell that cause accumulation of secretions with obstruction and secondary atelectasis.
  - Bronchiolitis is characterized by bronchiolar obstruction with edema, mucus and cellular debris. Thus lumen narrowing occurs with disturbing bronchiolar pulmonary ventilation, resulting in emphysema and atelectasis dependent of bronchiolar affected areas. Moderate obstruction allows the passage of air through bronchiolar during inspiration, with reduction of alveolar air during the exhale, with the consequence of emphysema formation ("air trapping"). Instead, bronchiolar severe obstruction blocks the air passage draws leading atelectasis. On the pathophysiological will always result hipoxemia being accompanied by the hypercapnia in the severe forms of disease.
  - Viral pneumonia is characterized by accumulation of mononuclear cells in perivascular space and submucoasa, causing airway obstruction. The disease progresses when the alveolar type II cells lose their structural integrity by reducing the production of surfactants and development hialine membranes followed by infiltration with inflammatory cells and alveolar interstitial space. Have been described 2 "pattern" sites, both changes being in the interstitial lung:
    - In the first pattern, the cilia epithelium become cuboidal or flat epithelium, with loss of cilia, subepithelial tissues and interalveolar walls are infiltrated with mononuclear cells, recesses are filled with inflammatory exudate. Lesions vary in severity in different areas of the lung;
    - In the second pattern, the bronchial tree and recesses have severe and extensive changes. Inclusions appear to intranuclear bronchiolar epithelium and the cells that line the recesses. Necrosis may occur, denudation and epithelial desquamation in bronchiolar and bronchi. Bronchial walls are infiltrated with lymphocytes, macrophages and plasma cells. Alveolar cells are swollen and recesses are hialine membrane thick paste.
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