Sialodacryoadenitis Virus Infection, Upper Respiratory Tract, Rat

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Synonyms. Rat submaxillary gland virus infection; SDAV infection.

Gross Appearance

Gross lesions are usually extrarespiratory and limited to mixed or serous salivary glands, exorbital glands, Harderian glands, periglandular connective tissue, cervical lymph nodes, and thymus. The submaxillary and parotid salivary glands are enlarged, pale, and edematous. Intermandibular and cervical connective tissue is gelatinous due to periglandular edema. This edema restricts the venous return in the neck, resulting in distention of the great veins entering the thoracic inlet. Exorbital glands are occasionally enlarged. Harderian glands are swollen and flecked with yellow-gray spots. These foci must be distinguished from normal brown-red mottling of the Harderian gland imparted by its normal content of porphyrin pigment. The cervical lymph nodes are enlarged and the thymus is atrophic. In these cases, ocular lesions may include corneal opacity, corneal ulcers, pannus, hypopyon, hyphema, and megaloglobus (Innes and Stanton 1961; Jacoby et al. 1975, 1979).

Microscopic Features

Respiratory lesions are primarily restricted to the upper respiratory tract. They precede inflammatory changes in the exocrine tissues of the head. Over the course of approximately 5 days, beginning on the 2nd day of infection, there is spreading necrosis of respiratory epithelium in the nasal cavity accompanied by congestion, edema, and mixed inflammatory infiltrate of the lamina propria (Figs. 120 and 121). The epithelial lining of the turbinates is most severely affected; olfactory epithelium is usually spared. Some meatuses are

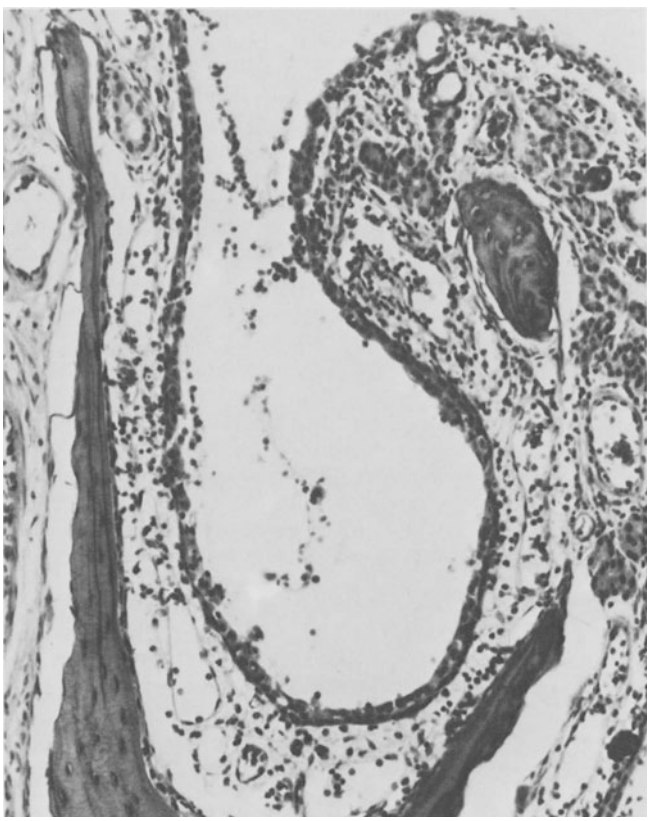

Fig. 120. Ventral turbinate and lateral wall of nasal cavity in rat experimentally infected with sialodacryoadenitis virus. Note exudative inflammation of the mucosa with gaps in epithelial integrity. H and E, × 240 (reduced by 15%)

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covered by exudate composed of necrotic epithelium, neutrophils, and mucus. Despite a tropism of this virus for serous or mixed salivary glands, the serous mucosal glands of the nasopharynx sustain relatively mild injury. Necrotic ducts and acini within mucosal glands do occur, however, and afford some specificity to the lesion. There is qualitatively similar inflammation in the trachea, but changes are milder and less uniform than in the nasopharynx. Upper respiratory lesions are resolved by the end of the 2nd week of infection. Lung changes are confined to mild hyperplasia within peribronchial lymphoid nodules (Jacoby et al. 1975, 1979).

Severe inflammatory changes occur within mixed or serous salivary glands, exorbital glands, and Harderian glands. Description of these changes is beyond the scope of this volume. The reader is referred to several excellent studies of sequential changes in these tissues (Innes and Stanton 1961; Jacoby et al. 1975, 1979).

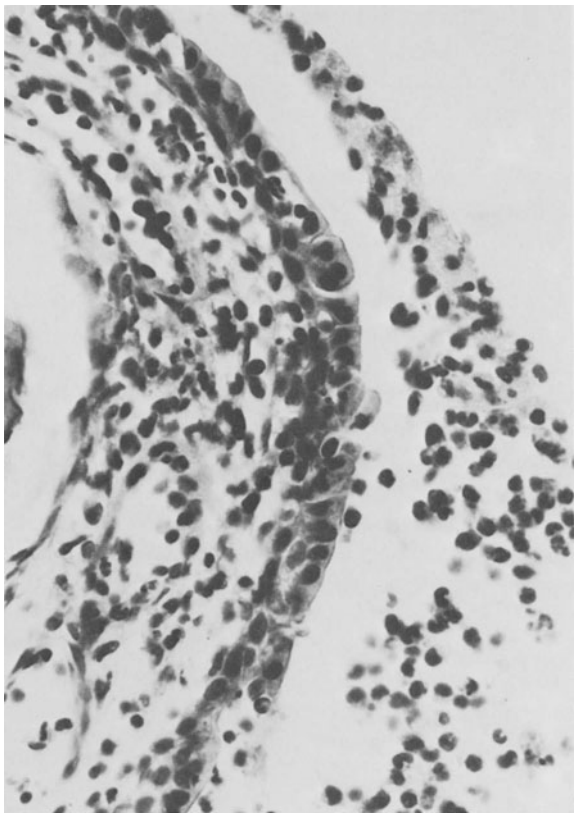

Fig. 121. Ventral turbinate of a rat experimentally infected with sialodacryoadenitis virus. Much of the epithelium is necrotic and desquamated. Some leukocytes are present in the lumen. H and E, × 740 (reduced by 15%)

Ultrastructure

We have found no report of the ultrastructural features of respiratory tract lesions caused by SDAV, but these features have been studied in infected submaxillary gland epithelium (Jonas et al. 1969). Infected epithelial cells have focally dilated cisternae of endoplasmic reticulum and cytoplasmic vesicles which contain spherical dense or hollow cores, 60–70 nm in diameter, surrounded by an envelope 80–120 nm in diameter. The characteristic corona, seen in negatively stained preparations, is not seen by transmission ultramicroscopy. Morphologically, sialodacryoadenitis virus is indistinguishable from Parker's rat coronavirus.

Differential Diagnosis

Upper respiratory tract lesions must be distinguished from those caused by Parker's rat coronavirus, Sendai virus, pneumonia virus, *Mycoplasma pulmonis,* and pathogenic bacteria. Pneumonic changes, which frequently accompany rhinitis caused by Parker's rat coronavirus, Sendai virus, and pneumonia virus, have not been reported in SDAV infection. A careful histopathological examination of the exocrine tissues of the head is usually sufficient to enable one to provisionally diagnose SDAV infection, but rhinotracheitis can precede changes in these tissues.

Biologic Features

Natural History. Sialodacryoadenitis virus causes acute limited infections; there is no evidence for a carrier state. The virus is highly contagious and is transmitted by aerosol, direct contact, and fomites. There are two patterns of infection. Enzootic infections occur primarily in breeding colonies, where sucklings are passively immune, adults are actively immune, and weanlings are a continuous source of susceptible individuals due to waning passive immunity. It is therefore weanlings that generally exhibit clinical signs. Explosive epizootics occur in nonimmune colonies with the highest morbidity in weanlings. Signs are usually transient and consist of intermandibular and cervical edema, swelling of submaxillary glands, sneezing, nasal and ocular discharges which are often red-tinged due to a high content of porphyrin, photophobia, and keratoconjunctivitis and its sequelae. Some complications of keratoconjunctivitis, such as glaucoma and phthisis, cause permanent disfigurement (Jacoby et al.

1979). Subclinical infections are common. Extensive host range studies have not been done but SDAV can experimentally infect mice by the respiratory route (Bhatt et al. 1977).

Pathogenesis. Sialodacryoadenitis virus is epitheliotropic, with replication limited to the respiratory tract and certain exocrine tissues of the head and neck. It replicates at all levels of the respiratory tract but produces disease primarily in the upper respiratory tract, where the highest titers are achieved. Virus is excreted for 7 days, after which it is cleared and neutralizing and complement-fixing antibodies appear in the serum (Jacoby et al. 1975, 1979).

Etiology. Sialodacryoadenitis virus (Coronaviridae) is a pleomorphic, enveloped RNA virus with plump. pedunculated surface projections (corona). It is approximately 114 nm in diameter. The virus replicates intracytoplasmically and virions are formed in cytoplasmic vesicles and endoplasmic reticulum (Jacoby et al. 1979). The virus is closely related antigenically to Parker's rat coronavirus (Bhatt et al. 1972).

Frequency. Coronavirus infections are common in commercial and institutional rat colonies (Jacoby et al. 1979; Parker et al. 1970). Because of the close antigenic relationship of SDAV to Parker's rat coronavirus, seroconversion to both viruses occurs in SDAV-infected rats. It is therefore difficult to confirm SDAV infection by serology alone (Bhatt et al. 1972; Jacoby et al. 1979).

Comparison with Other Species

Coronaviruses are ubiquitous in humans, animals, and birds (Bohl 1981). Although coronaviruses cause respiratory infections in chickens, humans, and rats, SDAV (and to a limited degree Parker's rat coronavirus) is the only coronarvirus that replicates and produces disease in salivary, exorbital, and Harderian glands.

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