

# ROLE OF CONDITIONALLY PATHOGENIC MICROORGANISMS IN THE DEVELOPMENT OF EXPERIMENTAL DESTRUCTIVE PNEUMONIA

B. B. Zolotovskii, I. K. Prostov,  
and T. G. Litvinenko

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The incidence of acute suppurative destructive pneumonias (ASDP) among children is rising year by year and, despite some progress made in their treatment, mortality still remains high [1, 4]. Insufficient attention has been paid in the literature to the etiology and pathogenesis of this disease. The onset of ASDP is attributed mainly to the action of a pathogenic staphylococcus, as is reflected in another name for the disease — staphylococcal destruction of the lungs, and treatment is being developed along these lines. A model of ASDP, closely resembling the disease in children in its pathogenesis and morphological picture, has been the subject of a few communications [2, 3, 5, 6].

The object of this investigation was to study the possibility of development of ASDP under the influence of a conditionally pathogenic microflora and to study the role of changes in immunologic status in the appearance of inflammatory-destructive foci in the lungs.

## EXPERIMENTAL METHOD

Experiments were carried out on 32 mongrel puppies of both sexes, aged 3-4 months and weighing 4-5 kg. Immunodepression was induced by the scheme developed for a model of destruction of the lungs, with a pathogenic staphylococcus [2], by giving two intravenous injections of thiophosphamide, with an interval of 48 h between injections, in a dose of 0.4 mg/kg body weight. Under general anesthesia (pentobarbital 15 mg/kg) the animals were infected 7 days after the second injection of thiophosphamide with a suspension of microorganisms together with Freund's complete adjuvant, which was injected into the lower lobe bronchi of the right or left lung through a thin catheter. The mixture consisted of 3.0 ml of a 24-h culture of the organism, containing 10 billion bacterial cells in 1.0 ml.

The animals were divided into four groups with eight puppies in each group. The animals of group 1 were infected with a culture of *Proteus*, those of group 2 with a culture of *Bacillus pyocyaneus*, those of group 3 with a culture of *Escherichia coli*, and the puppies of group 4 served as the control, and received only the adjuvant. Roentgenograms of the chest were taken 7 days after infection, and on the 8th-12th day the animals which survived were killed. At autopsy, the macroscopic changes in the lungs and pleura were assessed, after which an enzyme histochemical study was made of the pathologically changed lung tissue: Paraffin sections were stained with hematoxylin and eosin, and frozen sections were stained by Gomori's method for alkaline and acid phosphatases and by Padykula and German's method for adenosinetriphosphatase (ATPase).

## EXPERIMENTAL RESULTS

The state of all the experimental animals progressively worsened after infection: They grew thin, became adynamic and lethargic, and they refused to eat. Two puppies died in each of groups 1 and 2, and three puppies in group 3. In the control group the state of the animals showed no significant change and none of the puppies died. Early death of the experimental animals was evidently caused by generalization of infection.

Chest roentgenograms of the infected animals revealed opacities of different intensities and size, and of homogeneous or nonhomogeneous character, in the lung field, with sharp or blurred outlines. The opacities

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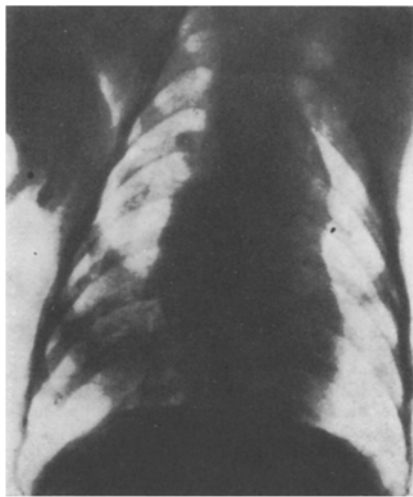


Fig. 1

Fig. 1. X-ray photograph of the thorax of a puppy with acute suppurative destructive pneumonia in the right lung.

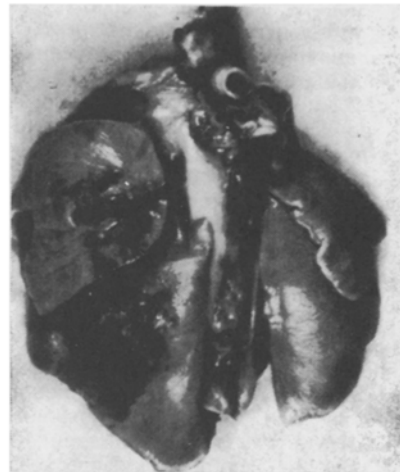


Fig. 2

Fig. 2. Pathological specimen of puppy's lung with foci of acute suppurative destructive pneumonia.

were located mainly in the lower lobes at the site of injection of the organisms, but in some cases they were also observed in the upper lobes. The changes in the roentgenograms were identical in animals of all experimental groups, i.e., microorganisms of different species caused a process in the lungs with an identical roentgenologic picture (Fig. 1).

At autopsy fine deposits were present on the visceral pleura in the region of the affected lobes in the form of greyish films and threads; the pleura itself was rough and dull and its surface was nodular, so that the subpleural pathological foci could easily be detected. The foci varied in size from 0.5 to 2-3 cm in diameter, and in a few cases they occupied the whole lobe. Macroscopically, the destructive foci produced by different microorganisms differed only a little from each other; they were clearly demarcated from the surrounding lung tissue, firm in consistency, and in most cases they were grayish in color, less frequently dark red. On section the affected tissue was airless, with many destructive foci, from the surface of which a purulent exudate escaped (Fig. 2). No such changes were found in the lungs or pleura of puppies of the control group.

The histological investigation showed that the blood vessels of the lungs in the experimental animals were dilated and congested, the fibers forming their walls were separated, and the walls themselves were edematous (Fig. 3a). The vascular endothelium was vacuolated, and desquamated in some places. Thrombus formation took place in many veins and arteries, especially small ones, and perivascular infiltration by polymorphs was observed. Capillaries in the interalveolar septa were congested and dilated and their endothelium had high alkaline phosphatase and ATPase activity; reflecting an increase in their permeability (Fig. 3b). Changes in bronchi of medium caliber were minimal. Considerable disturbances were found in the small bronchi (Fig. 3c, d). The alveoli, interalveolar septa and foci of atelectasis were diffusely infiltrated with polymorphs and macrophages, with high nonspecific phosphatase activity; (Fig. 3d, f).

The most conspicuous feature in the lungs was thus the hemodynamic disorders, characterized by thrombus formation and by severe changes in the vessel walls. The foci of inflammation differed in activity, with predominance of desquamative processes in some areas and of acute suppurative inflammation with the formation of small abscesses in others.

These experiments showed that acute suppurative-necrotic changes in the lungs arise when the immunologic resistance of the patient is depressed. It will be noted that ASDP develops not only through the action of a pathogenic staphylococcus, but also through the action of conditional pathogens and, in some cases, of

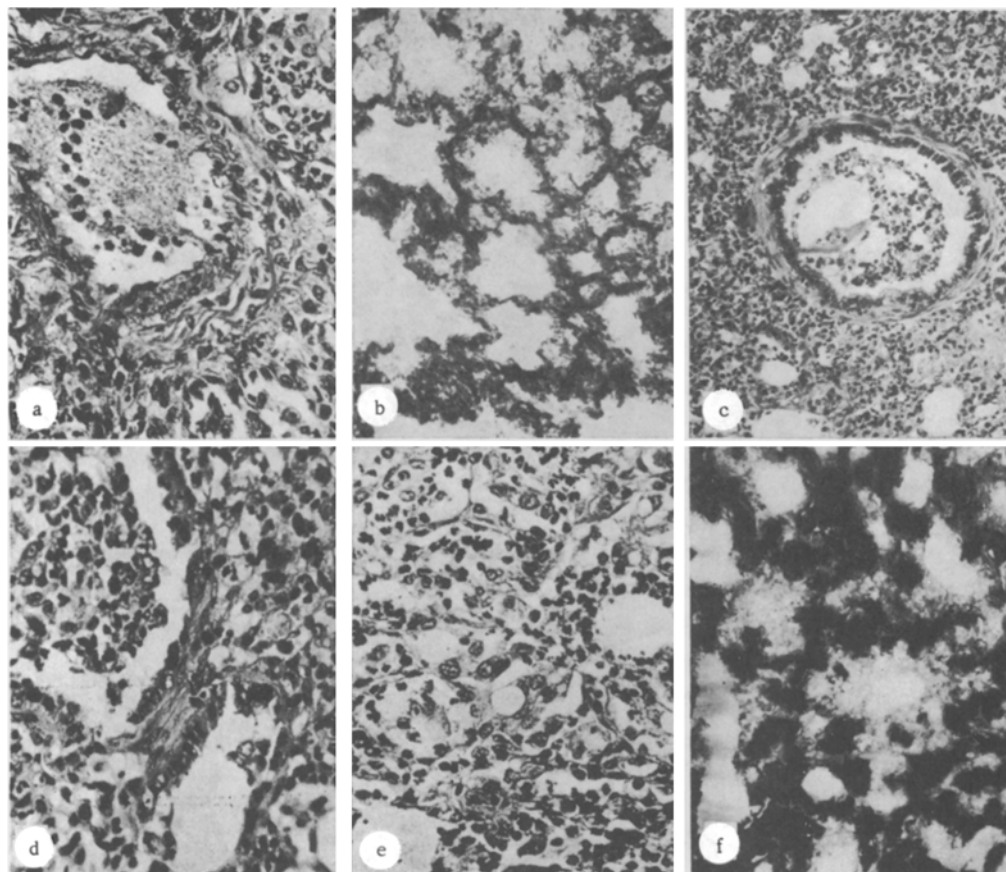


Fig. 3. Changes in lungs in experimental bacterial destructive pneumonia: a) deposition of fibrin in lumen of vessel, thrombus formation, edema and separation of fibers in walls of blood vessel, vacuolation of endothelium; b) increase in ATPase activity in capillary endothelium of interalveolar septa; c) dilatation of small bronchus, smoothing of folds of mucosa, peribronchial inflammation, polymorphs in lumen of bronchus, together with desquamated epithelium and mucus; d) desquamation of bronchial epithelium with focal denudation of basement membrane; e) diffuse polymorph infiltration of alveoli; f) sharp increase in alkaline phosphatase activity in leukocytes and macrophages. a, b, e) *B. pyocyaneus*; c) *Proteus*; d, f) *E. coli*; a, c-e) hematoxylin and eosin; b) Padykula and German's method; f) Gomori's reaction; a, d-f) magnification 400; b, c) 160.

saprophytic microorganisms such as *Proteus*, *E. coli*, and *B. pyocyaneus*. The disturbance of the microcirculation and destruction of the small bronchi play an important role in the pathogenesis of the destructive foci in the lungs.

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