AD NUMBER

AD842475

NEW LIMITATION CHANGE

TO

Approved for public release, distribution unlimited

FROM

Distribution authorized to U.S. Gov't. agencies and their contractors; Administrative/Operational Use; SEP 1968. Other requests shall be referred to Commanding Officer, Fort Detrick, Attn: SMUFD-AE-T, Frederick, MD 21701.

AUTHORITY

Biological Defense Research Lab ltr dtd 13 Sep 1971

TRANSLATION NO. 116

DATE: Sept 1968

DDC AVAILABILITY NOTICE

This document is subject to special export controls and each transmittal to foreign governments or foreign nationals may be made only with prior approval of Commanding Officer, Fort Detrick, ATTN: SMUFD-AE-T, Frederick, Md. 21701.

NOV 6 1968

DEPARTMENT OF THE ARMY
Fort Detrick
Frederick, Maryland

5

HISTOLOGICAL & PATHOGENIC STUDIES OF EXPERIMENTAL PULMONARY ANTHRAX

BY H. VELU, P. GAVAUDAN AND P. SOULIE'.

We have studied in the various previous papers (1) under which condition it was possible to definitely establish with mice whose lungs contained 1-500 anthrax spores though inactive by themselves, yet they were fixed by inhalation or by ingestion to fatal pulmonary anthrax by means of a dose chlorine incapable of provoking anything but a little congestion & edema. The histological examination of the lungs has revealed the existance of pulmonary anthrax accompanied by & probably preceded by peritracheal & peribronchial anthrax in some instances.

Pulmonary anthrax is characterized by a massive pollution with bacteria in the walls of the alveoli; it was not possible until now to pin point the cause of the germination of spores. The bacteria penetrated all the alveolas walls and the capillarics lodging themselves between the histocytes of the covering, causing an edema of the walls & of the congestion. It resulted in a mobilization of the histocytes of the covering which ended at times in a saturation of the alveoli. The leucocyte reaction is inconsistent in its intensity. The alveolas walls is finally destroyed & in extreme cases one can even observe the disappearance of the alveolas structure of the lung.

The intra-alveolas histocytes don't offer the particular characteristics & never any phenomena of phagocytosis of the bacteria nor were they lifted to their level; they could be multinuclear like a simple irritation & the seat of the nuclear division, adjoining important bacterial lesions.

The anthrax bacteria grow in diffuse colonies or in connected chains long enough to cover the alveolas wall in question. It is rare or non-existant

in the intact alveoli where it is formed by very dense clusters of embedded bacterial matter. It is demonstrated at times by a certain polymorphism whose irregular variation is demonstrated in the colony. Hence it offers a particular aspect through its large size and its embossed colony which gives it a mycelioid appearance.

The alveoli generally resembles the principal place of bacterin multiplication under the influence of chlorine. Meanwhile the infection could be initiated, as proven by the existence of peritracheal & peribronchial sources in other points. The subjects sacrificed only a few hours after the inhalation of chlorine where one could find proof of existence bacterial signs, during a process & trained apportionment across the tracheal epithelium & the skin which could very well represent the portal of entry of B. anthracis.

It is impossible to histologically distinguish a lung of a mouse dead of pulmonary anthrax very rapidly which died in 48-72 hours after it inhaled chlorine from a lung of another mouse which succumbed later within 6 days, of anthrax septicemia subsequent to a subcutaneous inoculation of a small fatal dose of spores. In both cases the concentration of spores in the pulmonary sources is identical & the pneumonia has the same symptoms. One observes the same edema, the same alveolor congestion, the same histological reaction which is connected with a primary lesion with a local germination of spores under the influence of a dose of chlorine in capable of prvoking similar disorders or a secondary final lesion, through a blocking of the lung following the methods studied by Binot & Ch. Jaulmes (2) in regard to other bacteriological studies.

Hence no difference could be established between the lungs of the animals which had the anthrax subcutaneously injected or where they were initiated

through chemical issolation of the bronchi and the alveoli of the test animals carriers of the spores which entered the lungs by respiratory or digestive means.

The equal importance exists in the local receptivity not only chosen & limited to the subcutaneous method as assumed by Besredka but prevalent, simply due to a modification of the surface of the skin by the traumatism, the injection the lepilation as conducted by Besredkas' experiments; here, by chlorine inhalation into the lung.

In other words, the same as for tuberculosis as mentioned by E. Arnold (3), the anthrax infection is not merely governed by the number of spores introduced into the organism but also by causes which permit these spores to multiply in any of the tissues. The industrial prophylactic of pulmonary anthrax must bear in mind this conclusion & to try to bar the workmen not only from contagion, thru infected dust, but also protect him from the usual causes of diverse irritations permitting a multiplication of the invading germs in an organism where they could not otherwise develop.

(1) Bull. de l'Acad. med., 1941, t. 125, p. 159; ibid., 1943, t. 127, p. 175. C.R. de la Soc. de biol., 1941, t. 134, p. 1354; ibid, 1942, t. 136, p. 371; ibid., 1943, t. 137, pp. 159, 160 et 224.

(2) Presse medicale, 1938, 24 aout, p. 1281

(3) Presse medicale, 1943, n 23, p. 324.