4069

Pleural Reactions to Primary and Secondary Infection with Tubercle Bacilli.

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It has been shown[1] that the pleura of a guinea pig, previously sensitized to tubercle bacilli, reacts differently from the pleura of a non-sensitized pig. Paterson obtained a reaction on injecting varying amounts of from 0.001 gm. to 0.00001 gm. of tubercle bacilli in sensitized animals, and no reaction in non-sensitized animals unless the dose was 0.002 gm. or more.

If tuberculous pleural effusion develops in the human chest after days or weeks of febrile reactions, the patient usually gets well or improves, the fluid is absorbed, and thickening of the pleura takes place, unless the lung is too actively involved. The object of our study has been to determine whether tuberculous pleurisy with effusion is essentially a protective mechanism and how it is produced.

Thirty adult guinea pigs averaging 650 gm. were tested with O. T. intracutaneously and were found to be negative. Fifteen of these pigs received 0.1 mgm. RI (Trudeau) of a relatively avirulent strain of human tubercle bacilli subcutaneously into the right groin. Three weeks later they gave a positive tuberculin reaction. Fifteen of the animals were left untreated; 68 days later the 15 sensitized and the 15 untreated pigs received intrapleurally 0.001 mgm. of a more virulent strain of bacilli H-305 in 1 cc. saline. The 30 pigs were then tested as follows:

(1) Five of the sensitized and 5 of the non-sensitized pigs were tapped and aspirated at 24-hour intervals for a period of 5 consecutive days, and cellular studies made of the fluids.

(2) The second set of 5 sensitized and 5 non-sensitized pigs were killed at the same time intervals as above, and studies were made of the fluids, thickening, adhesions, and the dissemination.

(3) The third set of 5 sensitized and 5 non-sensitized pigs were permitted to live in order to study the length of life and difference in distribution between the aspirated and non-aspirated animals.

Following reinfection fluid was obtainable in small amounts varying from 0.3 to 2 cc. in nearly all the sensitized animals during the first few days. None of the controls showed an immediate exu-

[1] Paterson, Robert C., *Am. Rev. of Tuberculosis*, 1917, i, 353.

date but some of them developed fluid weeks or months after inoculation. This latter finding may be explained by endogenous reinfection. Cellular studies were made on the fluids obtained. In the sensitized animals there was an immediate response of fluid containing large amounts of polymorphonuclear leucocytes varying from 80% to 92% in the first 24 hours, with a gradual replacement of the polymorphonuclear leucocytes by clasmatocytes. On the 4th and 5th days the clasmatocyte count varied from 72% to 100%.

In order to determine the type of cells that may be found in the non-sensitized animals (controls), where no immediate exudate is present, 0.1 cc. of broth was used to wash out the pleural cavity. From the first to the 5th day after reinfection the predominating cells in the washed cavities were clasmatocytes varying from 96% to 100% as is true in normal animals (Gay and Morrison).[2] The white count varied from 1,750 to 12,750 cells per cu. mm. Whether there is an increase in total clasmatocytes and white cells in these infected controls over the normal animals is still under investigation. Adhesions and thickening of the visceral, parietal, and diaphragmatic pleura averaged 70% in the sensitized animals within the first 5 days, whereas no adhesions, thickening, or fluid were found in any of the non-sensitized animals even with an intrapleural dose of 5 mgm. of H-305.

Intrapleural injections of small doses of tuberculin gave no pleural reaction in either sensitized or non-sensitized animals. Emulsions of dead streptococci gave no reaction in sensitized or non-sensitized animals, but the injection of 0.1 mgm. RI or 0.001 mgm. H-305 gave a positive reaction in 17 out of 20 sensitized animals. Thickening and adhesions were encountered in all the sensitized animals that were permitted to live on. The degree of thickening varied from fine adhesions to complete obliteration of the pleural cavity, with marked adherence of lung to the chest wall. Thickening and adhesions were also found in 20% of the non-sensitized animals weeks or months after infection.

In 40% of sensitized animals tubercle bacilli were found in the exudate 1 to 5 days after reinfection. In the non-sensitized animals no tubercle bacilli were found when 0.001 mgm. H-305 was used as the dosage. When 5 mgm. were used 40% of the broth washings from the non-sensitized animals were found positive for tubercle bacilli.

The difference in the length of life of the sensitized and non-sensitized animals has not as yet been determined.

[2] Gay, F. P., and Morrison, L. F., *J. Am. Med. Assn.*, 1923, lxxx, 1298.

Conclusions: To obtain an immediate pleural reaction by means of tubercle bacilli, previous sensitization appears necessary.

Tuberculous pleurisy in guinea pigs would appear to be a reinfection as is believed to be the case in man.

The reaction of the sensitized pleura on reinfection with tubercle bacilli is another illustration of the Koch phenomenon, and offers advantages for the study of the protective significance of this reaction.

4070

Philippine Fowl Disease.

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What is believed to be a new disease of fowls recently appeared in the Philippines. The epizootic began in September, 1927, and proved to be highly contagious. The first case of the disease appeared in Manila and rapidly spread over an area having a radius of about 50 miles with Manila as the center of the infection. By February 1, 1928, it has been estimated that at least 50,000 fowls have succumbed as a result of this disease. Both males and females are affected and fowls of all ages are susceptible. The disease has been prevalent in chickens, and a few cases have been noted in ducks and geese.

Symptoms: The onset of the disease is sudden. Chickens at first show an indisposition to move about, preferring to sit or stand quietly in a secluded spot. Very early in the course of the disease there is a diarrhea which gradually improves if the fowl is to recover. Gasping for air associated with jerking movements of the head downward and backward is a characteristic symptom. This is caused by large quantities of tenacious mucus which obstructs the posterior nares and pharynx. Many fowls die of suffocation early in the course of the disease. Excessive thirst is noted in many cases and the crop is often filled with large quantities of water and mucus as well as foul smelling gas in some instances.

Occasionally there is a bloody diarrhea but this is not a common symptom. The fowls usually die within 1 to 7 days following the

* This preliminary report has been prepared by Dr. McKinley and will appear in this form in his forthcoming review of the filterable virus diseases.