1938

INFECTION OF THE CHICKEN WITH CAPILLARIA COLUMBAE (RUD.)

P. P. Levine
New York State Veterinary College, Ithaca, New York

Follow this and additional works at: http://digitalcommons.unl.edu/jrnlparsitology

Part of the Parasitology Commons

http://digitalcommons.unl.edu/jrnlparsitology/759

This Article is brought to you for free and open access by the Parasitology, Harold W. Manter Laboratory of at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Journal of Parasitology Archives by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.
INFECTION OF THE CHICKEN WITH CAPILLARIA COLUMBAE (RUD.)*

P. P. LEVINE

Department of Pathology and Bacteriology, New York State Veterinary College, Ithaca, New York

There have been several reports of infections of chickens with Capillaria columbae. The first record was by Graybill (1924) who also found it in turkeys. Morgan (1932) reported it in England. Reis, Nobrega and Reis (1936) have cited numerous instances of capillaria infection in the domestic fowl.

Pathogenic effects of C. columbae in chickens have not been reported. Stubbs and Crawley (1922) described a severe chronic proliferative enteritis in two chickens caused by a mixed infection with Capillaria spp. and Heterakis vesicularis. A diphtheritic follicular enteritis in one chicken caused by Capillaria spp. was described by Graham et al. (1929). They found a marked dilation of the intestine anterior to the caeca and a histo-pathological study showed necrosis and sloughing of the epithelium. On the other hand the pathogenicity of Trichosoma tenuissimum (Dies.) (=C. columbae) for pigeons was reported by Eber (1917) and Schlegel (1918). These workers observed that the infected pigeons showed extreme emaciation and finally died. A severe catarrhal enteritis was found on post-mortem examination.

Graybill (1924) found that the ova of C. columbae when incubated in shallow layers of saline solution at 22° to 25° C. were embryonated after 7 days while another culture embryonated in 6 days at 24° to 25° C. He did no feeding experiments. The direct life cycle of C. retusa (Cram not Raillet) was experimentally demonstrated by Cram (1932). However, after a restudy of her specimens, Cram (1937) is convinced she was working with C. columbae and not C. retusa. Cram incubated the ova at 30° C. and found that when they were examined 42 days later they contained fully developed embryos. The embryonated ova were fed to a chicken and a quail and capillaria were recovered from the intestines of these birds 19 and 22 days later, respectively.

A satisfactory vermicide has not been reported for capillaria. Carbon-tetrachloride and tetrachlorethylene have been tried by Cram (1932) and by Rietz (1927) with inconstant results.

Received for publication, March 27, 1937.

*The writer is indebted to Dr. E. Cram, of the National Institute of Health, Washington, D. C.; Dr. B. Schwartz and Dr. E. Wehr, of the Zoological Division, Bureau of Animal Industry, Washington, D. C., for their aid in identifying the parasites submitted to them on various occasions. The writer also wishes to acknowledge his thanks to Mr. W. Bell for his help in making parasite counts in the anthelmintic studies.
Since *C. columbae* has been found on numerous occasions in the intestines of chickens sent to this laboratory, a study of this parasite was undertaken. The life cycle and its pathogenicity in particular were studied. In addition some experiments were done to test the efficiency of two vermicides against it.

**METHODS**

The birds used in these experiments were Barred Plymouth Rocks, Rhode Island Reds and Single Comb White Leghorns. These chickens were artificially hatched, brooded, and kept in batteries or individual cages with wire screen floors. Adventitious infection with *C. columbae* was not encountered during the course of this investigation.

To produce heavy infections with *C. columbae* the technique described by Levine (1936a) was used. In this method fly larvae consume the putrescible material in feces passed by parasitized chickens. The fecal residue containing the unharmed capillaria ova is moistened and incubated until embryonation takes place. This material is then mixed with the mash and fed to the birds. It was found that when chicken manure was used as a source of infective material coccidia as well as capillaria were being transmitted. Fortunately a number of pigeons which were being kept at the laboratory were found to be naturally infected with *C. columbae*. Consequently pigeon manure was used as a source of infective material. In spite of the fact that the pigeons were passing coccidal oocysts transmission of coccidiosis to the chickens did not result. This corroborates the results of Nieschulz (1925), cited by Becker (1934), who was able, with pigeon coccidia, to produce only a mild and transient infection in 2 out of 23 chickens.

Worm counts were done by scraping the mucosa of the entire intestine into a dish of warm water and examining it in small portions under a wide field binocular microscope. The procedure was facilitated by adding to each portion a few drops of 20% NaOH solution to dissolve the mucus.

Statistical analyses of the weight records were done according to the method of Fisher (1934) for paired data. The probability table used in determining the significance of mean differences was Student’s table for ‘t’ as modified by Livermore (1934). Mean differences were considered significant if the odds were 19:1 or greater.

**EXPERIMENTAL**

**Life Cycle Studies**

Ova of *C. columbae* procured from female worms which had been removed from the intestines of chickens, were placed in shallow layers of distilled water and incubated at 30°C. Embryonation was completed
on the 6th or 7th day, at which time microscopic examination revealed the fully formed coiled and moving embryo within the egg-shell. The embryonation time agrees with Graybill's results (1924) although he incubated the ova at lower temperatures. Suspensions of the embryonated ova were pipetted directly into the crop of a large number of parasite-free chickens ranging in age from 28 to 66 days. Periodic examinations of the feces from these birds were done. Fertile non-embryonated ova did not appear in the feces until 21 days after the infective feeding. The birds were autopsied from 36 to 294 days after feeding the embryonated ova. In every case mature specimens of *C. columbae* were found in the intestinal mucosa.

In one experiment 8 birds, after being starved for 24 hours, were fed embryonated ova mixed with the feed. One bird was autopsied on each of the following 6 days and on the 10th and 13th day after the infective feeding, respectively. Microscopic examination of fresh scrapings from the mucosa of the small intestine showed active larvae 24 hours after the infective feeding. That these larvae had already started to penetrate the intestinal epithelium was demonstrated in stained tissue sections of the intestine. As the parasites got older they increased in size and were found partially imbedded directly beneath the epithelium of the villi. Occasionally some worms were found deep in the mucosa beneath the epithelium of the intestinal glands but generally the villi harbored most of the worms. Although in heavy infections *C. columbae* were found in all portions of the intestine including the duodenum and caeca, generally the parasites were most commonly encountered in the mucosa of the lower two-thirds of the small intestine.

*Pathogenicity of C. columbae for Chickens*

Twenty-six Rhode Island Red chickens, 8 weeks old, were divided into two groups so that each bird in one group was matched with a bird of the same sex and weight in the second group. This was done to insure a similar distribution in both groups thereby facilitating the statistical analysis of the weight records which were made at regular intervals. Each group was kept in a separate compartment of a holding battery with wire screen floors. Feed and water were before the birds at all times. Infection in group 1 was effected by mixing with the mash pigeon manure which contained embryonated *C. columbae* ova prepared as already described. The birds ate the contaminated feed readily and generally consumed all of it within two hours. Regular feed was then placed before them as usual. Infective material was fed on September 15, 16, 17, 19, 24, 29, October 5 and 9. No attempt was made to count the number of ova fed.

Clinical Symptoms: The first symptoms were noted 12 days after the first infective feeding. In the feces of the infected group were found
numerous pinkish colored shreds of material which had a soft, moist, somewhat rubbery texture. Microscopic examination of wet preparations of this material showed it to be composed of strings of mucus, necrosed epithelial cells and numerous erythrocytes, granulocytes and lymphocytes. In one instance there was observed a long strip of epithelial cells still joined together and retaining the shape of a villus. Entwined in this material were large numbers of active immature C. columbae apparently of different ages since there were marked differences in the sizes of these worms. During the next four days the elimination of epithelium and inflammatory exudate was greatly increased which caused the feces to become quite fluid. The infected birds exhibited a depressed attitude and stood huddled together with wings and tails drooping and feathers ruffled. During the following two weeks the character of the feces gradually returned to normal and the majority of the infected chickens regained their normal appearance. Four of the birds, however, lost weight steadily, became extremely emaciated and weakened, and either died or were destroyed 24, 25, 41 and 44 days, respectively, after the first infective feeding. Nervous symptoms were not noticed in the parasitized birds.

Examination of the weight records in Table I shows that during the period up to and including the ninth day after the first infective feeding there were no significant differences between the mean weights of the control and the infected birds. On the 15th day after the first infective feeding the mean weight of the infected group was 31 pounds less than that of the controls. It should be noted that this weight loss occurred at about the time that the first clinical symptoms were observed. When this weight difference was analyzed statistically it was found to be highly significant, for each parasitized bird had lost weight during that week while the controls had made normal gains. During the following three weeks the weight differences were substantial and significant statistically. Although the mean weight difference between the control and the infected groups at the end of the last week of the experiment was the largest recorded, that difference was found to be not significant. The weights of two emaciated birds in the parasitized group served to lower the mean weight substantially but the other birds in that group had made substantial gains and weighed about the same as the controls. In other words, the general trend in the parasitized group was towards recovery.

Four days after the last weight records were taken all the birds were autopsied and the worms counted. In no case did the number of parasites found in any single bird exceed 14. On the other hand, the worm counts of the birds that had died or that were destroyed as a result of the infection ranged from 1555 to 4734. The very great disparity between the number of parasites in the dead or dying birds and in the sur-
LEVINE—INFECTION OF CHICKEN WITH CAPILLARIA

vivors, coupled with the observation that large numbers of parasites were being eliminated in the feces during the acute stage of the infection, suggests that “self cure” must be credited with a rôle in the survival from this infection. Undoubtedly the resistance of each individual bird largely determines to what degree this “self cure” can operate.

**Table 1.**—Effect of infection with Capillaria columbae on weight (in pounds) of eight-week-old chickens. Infective material fed Sept. 15, 16, 17, 24, 29, Oct. 5 and 9.

<table>
<thead>
<tr>
<th>Date</th>
<th>Sept. 1</th>
<th>17</th>
<th>24</th>
<th>30</th>
<th>Oct. 6</th>
<th>12</th>
<th>19</th>
<th>26</th>
</tr>
</thead>
<tbody>
<tr>
<td>Days after first infection</td>
<td>-14</td>
<td>2</td>
<td>9</td>
<td>15</td>
<td>21</td>
<td>27</td>
<td>34</td>
<td>41</td>
</tr>
<tr>
<td>Mean weight infected birds Group 1</td>
<td>1.1</td>
<td>1.9</td>
<td>2.2</td>
<td>2.0</td>
<td>2.2</td>
<td>2.2*</td>
<td>2.5*</td>
<td>2.7*</td>
</tr>
<tr>
<td>Mean weight control birds Group 2</td>
<td>1.1</td>
<td>1.9</td>
<td>2.2</td>
<td>2.3</td>
<td>2.6</td>
<td>2.6*</td>
<td>2.9*</td>
<td>3.2*</td>
</tr>
<tr>
<td>Mean difference</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>.3 ± .05</td>
<td>.4 ± .08</td>
<td>.4 ± .10</td>
<td>.4 ± .13</td>
<td>.5 ± .23</td>
</tr>
<tr>
<td>Significance of mean differences</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

* 11 birds only; all other mean weights of infected and controls, are on 13 birds.

Lesions: Birds infected with a few worms showed no changes in the intestine. Heavy infections caused a moderate thickening of the intestinal mucosa with reddened areas varying from pinhead hemorrhagic spots to diffuse hyperemia of large portions of the mucosa. In some instances very little or no exudate was present while in others the mucosa was covered with large amounts of catarrhal exudate ranging in color from opaque white to a translucent salmon pink. In one trial several of the birds showed a severe necrotic enteritis. Examination of the necrosed epithelium disclosed not only enormous numbers of immature capillaria but also a few coccidia. Since heavy infections with *C. columbae* alone never produced such lesions, the coccidia together with the capillaria were undoubtedly responsible for the extensive necrosis. On a number of other occasions the intestines of parasitized birds showed no abnormal changes in spite of the fact that the number of worms ranged from 1500 to 5000. This lack of gross inflammation in spite of a heavy infection may be so misleading that one may easily overlook the presence of these parasites.

The microscopic changes, for the most part, corroborated the gross findings. There was a dilation of the capillaries in the villi with an increased mucus production by the goblet cells in some cases. The parasites were found beneath the epithelium of the villi and in some instances the villi were pierced from one side to the other. In a few cases the parasites were found to have penetrated to the muscularis mucosa.

Where the parasites were cut longitudinally, it was found that the epithelium immediately above the worm either was in the process of
being shed or was completely detached from the villus. In most cases although the epithelium above the worm was still continuous the cells themselves were necrosed, atrophic and formed a very thin membrane. These findings explain the presence of epithelial cells in the discharges of parasitized birds. Aside from capillary congestion there was little local reaction around the worms themselves. This corroborates our findings in those cases where extremely heavy parasitisms were encountered in apparently normal intestines. There can be no doubt that the damage done by *C. columbae* results in part from the desquamation of the intestinal epithelium. This may be due either to the mechanical action of the worms or to the action of enzymes produced by the parasites in their migration through the tissues, or to both. Where large areas of mucosa have been denuded of epithelium, secondary bacterial infection could very easily gain headway and produce a necrotic enteritis.

**THE EFFICIENCY OF TOBACCO DUST FOR THE PREVENTION OF INFECTION**

The feeding of tobacco dust in the mash from the day of hatching has been shown to be ineffective for the prevention of infection with *Ascaridia lineata* in chickens (Levine 1936b). Similar experiments were done to test the efficiency of tobacco dust for the prevention of *C. columbae* infection. Fifteen Rhode Island Red chickens, 3 months old, were fed a mash containing 2% tobacco dust (nicotine content 1.75%) by weight. This mash was kept constantly before the birds throughout the duration of the experiment. Two days after being started on this mash 607 grams of pigeon manure containing embryonated *C. columbae* ova were fed to the entire group over a period of ten days. Post-mortem examination of the birds at various times after the infective feedings had terminated showed that the parasites not only invaded the intestinal epithelium but that they grew to maturity. That absolute prevention of the parasitism was unsuccessful cannot be doubted. However, since quantitative studies including a control group were not done, it cannot be stated with certainty whether or not some degree of protection was conferred.

**TREATMENT WITH CARBON-TETRACHLORIDE AND TETRACHLORETHYLENE**

Another series of experiments were done with individual birds infected with *C. columbae*. These birds were placed in separate cages, starved for 12 to 18 hours after which the anthelmintic was introduced directly into the crop. Feed was placed before the birds 1 to 3 hours after the drug was administered. All the manure passed for 48 hours following the treatment was collected in pans holding enough 1% formalin solution to cover it. At the end of this time the birds were destroyed and counts were made of the worms in the intestines and in the
manure. The efficiency of the drug was then calculated in the usual manner.

Five birds (average weight 2.5 pounds) were dosed with 1, 2, 3, 4, and 5 cc carbon-tetrachloride, respectively. This experiment was twice repeated. The results were quite erratic. The efficiency ranged from 0% (7 birds) to 39% (1 bird). There was no correlation between the efficiency of the drug and the amount administered.

In another experiment each of two birds was dosed with 2 and 3 cc tetrachlorethylene, respectively. This experiment was also twice repeated. The efficiency in 5 birds was 0% and in one was 2%.

Apparently neither of the drugs used was effective in removing the parasites.

DISCUSSION

The loss of weight, extreme emaciation and death of chickens heavily parasitized experimentally with *C. columbae* as recorded in this paper confirms the reports by various workers on the pathogenicity of capillaria for chickens. The gross lesions in the pure infections resembled those described by Eber (1917) and Schlegel (1918) in pigeons. Desquamation of the intestinal epithelium in cases of *C. columbae* infection apparently is similar to that of the esophageal and crop epithelium in pheasants parasitized with *C. annulata*, described by Graham (1935). The fact that this desquamation and catarrhal enteritis are the factors responsible for the elimination of a tremendous number of parasites is another example of the protective action of an inflammatory reaction. This “self cure” is quite marked under experimental conditions when heavy infecting doses are fed over a short period of time and when reinfection is prevented. Whether or not natural exposures with continual reinfection over a long period of time would elicit the same response on the part of the host cannot be answered at this time. Proliferative necrotic enteritis as described by Stubbs and Crawley (1922) and Graham *et al.* (1929) was not produced in our experimental birds except in a few cases where coccidiosis was a complicating factor.

The fact that pigeons may readily transmit the infection to chickens has not been taken into account by other workers in discussing the prevention and control of this parasitism. A small number of parasitized pigeons with easy access to any portions of chicken yards or ranges may easily be a constant source of infection for a flock of chickens. An obvious control measure, therefore, would be to confine all barnyard pigeons on poultry farms.

1 Wehr (1937, *J. Parasitol.* 23: 573) in a recent abstract report on *C. columbae* in pigeons confirms these observations, as well as several points on the life history recorded in the present article.
Our results of the treatment of *C. columbae* infection with carbon-tetrachloride and tetrachlorethylene are at variance with those of Rietz (1927). It is possible that had the medication been repeated as Rietz had done, an increase in efficiency might have resulted.

**SUMMARY**

The direct life cycle of *Capillaria columbae* (Rud.) was confirmed. Complete embryonation of the ova takes place in shallow layers of distilled water at 30° C. within 6 to 7 days. Twenty-four hours after the ingestion of the ova by chickens, the larvae can be found penetrating the intestinal mucosa. The worms mature in 21 days at which time fertile non-embryonated ova are found in the feces of the parasitized birds.

A severe infection with *C. columbae* was experimentally produced in chickens. The clinical symptoms and weight records of the parasitized birds showed that this parasite was pathogenic for fowls and heavy infections caused loss of weight, emaciation, and death. The lesions consisted of a catarrhal enteritis with desquamation of the intestinal epithelium.

The comparative ease with which parasitized pigeons may transmit *C. columbae* to chickens suggests the necessity for the elimination of pigeons in any control program.

The attempt to prevent infection by feeding mash, in which was mixed 2% tobacco dust by weight, was unsuccessful. Individual treatment of birds with single doses of carbon-tetrachloride and tetrachlorethylene likewise was not successful.

**BIBLIOGRAPHY**


Nieschulz, O. 1925 Arch. f. Protistenk. 1: 479.


