SOME PATHOLOGICAL ASPECTS OF NATURALLY OCCURRING CHRONIC RESPIRATORY DISEASE

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Symptoms and lesions of naturally occurring chronic respiratory disease in poultry were studied. The disease may be suspected when adult flocks encounter a slowly developing and progressively emaciating respiratory infection without much fatality. The disease can be diagnosed serologically.

Damage was primarily localised to the respiratory tract. The whole air passage was found to contain a variable amount of inflammatory exudate, while the lining mucous membranes were congested, thickened or partially sloughed. Marked thickening of the abdominal air sac-walls, typical beaded reaction of the tracheal lining; with or without linear haemorrhagic streaks, and calcification of the tracheal cartilages were among the more prominent lesions.

Lesions on the serous coverings of the abdominal viscerae suggest that in West Pakistan chronic respiratory disease was complicated with infection of *Escherichia coli* and coliform group of organisms.

INTRODUCTION

Many respiratory diseases of domestic poultry, including the chronic respiratory disease can cause more or less similar symptoms and somewhat comparable gross lesions (Jungherr and Luginbuhl, 1952; Van Roekel, 1966). This makes their differential diagnosis rather difficult. The difficulty is all the more accentuated when in a flock two or more of these infections appear simultaneously or follow each other in quick succession. Furthermore, as the primary causative agent of the disease was extremely pleomorphic, as well as widely distributed in nature (Nelson, 1953), therefore its isolation and identification is usually not easy. As such, the study of its pathology in nature is likely be of material help in its diagnosis.

Serological tests are very valuable in the diagnosis of the disease but histopathology of field cases, in the opinion of Jungherr *et al.* (1953) gave clues both to its primary as well as complicating causative factors. The present study was thus undertaken to look into the general pattern of the disease in the field and thereby record the nature and role of its probable complicating agents in the country.

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MATERIALS AND METHODS

Adult fowls of varying ages and either sex, which for a period of 2-3 weeks had shown severe respiratory symptoms but no mortality, were chosen and their sera subjected to rapid plate agglutination test. The symptoms comprised occasional coughing, sneezing, gasping, jerky movements of head, gargling sounds and a variable amount of nasal and ocular discharge. Feed consumption in them was distinctly below normal and a few advanced cases also showed cyanotic combs and wattles. Twenty-five such birds were carefully selected from three different poultry units of Lyallpur, all of which had been previously vaccinated against the Newcastle disease.

PPLO antigen used in the rapid plate agglutination test of Adler (1954) was of the serial number 16, which was received through the kind courtesy of Dr. Walter H. Martin of the USDA. For ready reference and easy comparison positive sera of known titres, also received from the same source, were run concurrently with the test sera and those of the negative controls.

Sick fowls, which had for over 2 weeks shown the above-mentioned symptoms and whose sera gave agglutination titres of 1 : 640 or higher, along with two obviously healthy and agglutination test negative controls were sacrificed and subjected to post mortem as well as histopathological examination. Selected pieces of the morbid organs and those of the negative controls were fixed in 10 per cent formalin, dehydrated in graded ethyl alcohol, cleared in xylene and embedded in paraffin. Sections of 4 to 6 micron thickness were then cut and stained with Haematoxylene and Eosin stain after Gridley (1960).

RESULTS

Macroscopic Findings

As against the negative controls, the carcases of the diseased fowls were emaciated and anaemic. Their upper respiratory tracts were found to contain variable amounts of mucopurulent (6),* fibrinous (4) or frothy (3) discharge. The nasal mucous membrane was hyperaemic (10) or thickened (7). The more copious tracheal exudate, on the other hand, was either frothy (15) or mucopurulent (5). Tracheal mucosa was thrown into distinct folds (15); sometimes referred to as "Beaded reaction," which also showed linear haemorrhagic streaks (12). Sloughing of the mucous lining (11) was also seen.

Frequent inflammatory changes in the air sacs were quite extensive. The thoracic air sacs, in particular, showed marked thickening of the walls

*Figures in parentheses are actual frequencies of the various lesions among the 25 diseased birds of the present study.

(17) and a variable congestion (8). They contained large quantities of caseous (8), frothy (7), fibrinous (5), or mucopurulent (3) discharge.

Lungs looked mostly normal. Some of the large bronchi, when incised, were, however, found partially plugged with some cheesy material (4).

Abdominal cavities contained small quantities of fibrinous (3) or suppurative (3) exudate and the peritonium showed mild adhesions (6) with the surrounding viscera. The coverings of the heart were slightly thickened (4) and the pericardial sacs were distended (4).

Digestive tracts were all normal and so were the livers and spleens.

Microscopic Lesions

Trachea, lungs, hearts, livers and spleens of both the diseased and the control birds were the only organs studied under the microscope. Inflammatory degenerative changes were, however, restricted to trachea and the lungs while the hearts and livers were only slightly affected.

Apart from focal necrosis (19) and sloughing (11) of the tracheal mucous membrane, there was marked hyperplasia of the lining epithelium (20) which was accompanied with a variable infiltration by the epithelioid cells. Varying degrees of degenerative changes extended deep into the walls of the trachea, involving also the hyaline cartillage which stained darker and at places appeared calcified (13). Photomicrographs of tracheal walls are shown in Fig. 1 (A and B).

Affected lungs showed varying degrees of congestion (6). Focal granulomas (3), scattered over the lung tissue, comprised mainly of large mononuclear cells, reticuloendothelial cells and lymphocytes. Areas of consolidation (6) were accompanied with mononuclear and epithelioid cell infiltration as well as epithelial proliferation. Areas of encapsulated caseation necrosis (5) and atelactasis (3) were also seen Fig. 1 (C and D).

Zones of inflammatory changes (4) observed between the visceral layer of the pericardium and the myocardium, comprised mainly of mononuclear cells, lymphocytes and erythrocytes. However, inflammatory reaction was not observed deep into the cardiac musculature (Fig. 1. E.).

Sections of spleen were all normal but those of the liver (3) showed sporadic mild congestion (Fig. 1 F).

DISCUSSION

As mentioned earlier, the poultry units from which the suspects of the present study were drawn had all been previously vaccinated against the New-

cattle disease. The possibility of complication by other fast spreading and more ravishing respiratory diseases of viral origin was excluded by the fact that severe respiratory symptoms lasted for a period of over two weeks without a single death. Final confirmation of the disease was based upon the rapid plate agglutination test; which according to Osteen (1954) and Gianforte *et al.* (1955) excelled all available serological tests not only in simplicity and practicability but also in specificity and reliability.

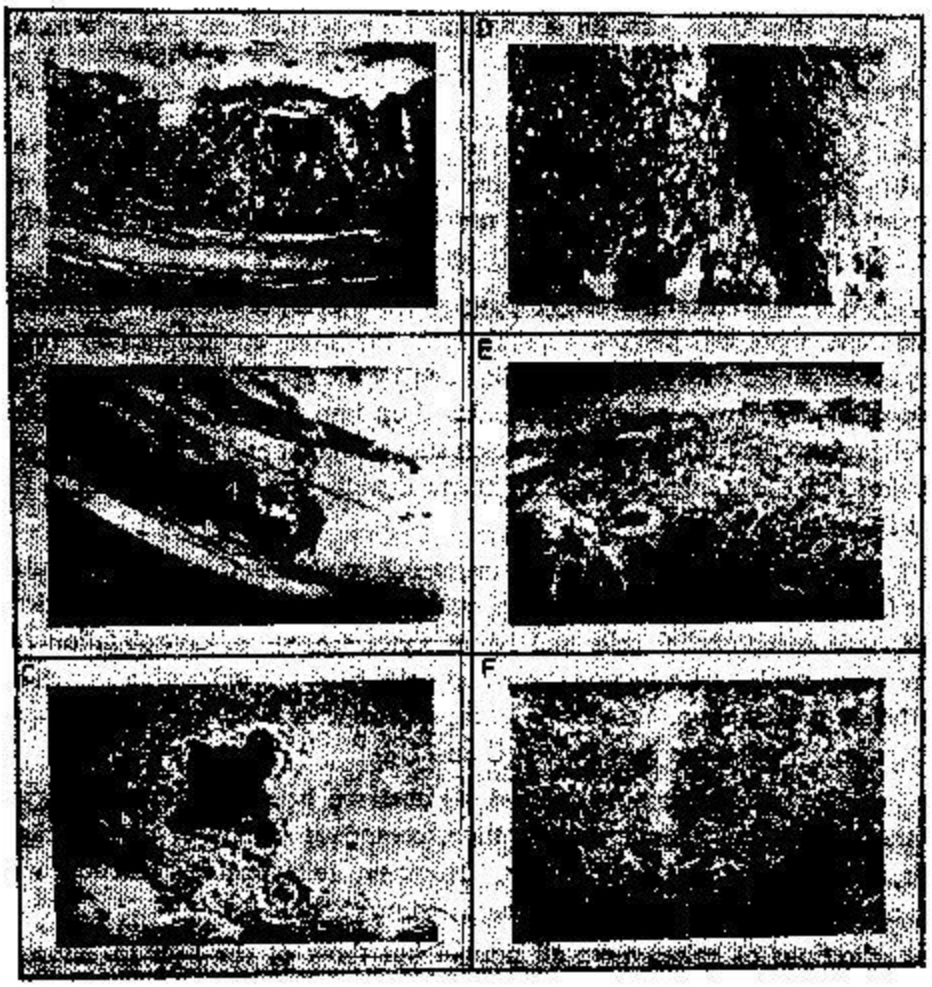

Fig. 1. Photomicrographs of organs affected by chronic respiratory disease: Trachea A (Low power) showing 1. Epithelioid cells. 2. Haemorrhages. 3. Hyperplasia. B (High power) Showing 1. Cellular degeneration. 2. Calcification of hyaline cartillage. 3. Epithelial desquamation. Lungs C (Low power) showing 1. Caseation necrosis. 2. Focal granulomas. 3. Congestion. D showing 1. Atelasis. 2. Congestion. 3. Normal tissue. Heart E (Low power) showing 1. Thickened pericardium. 2. Inflammatory exudate. Liver F (Low power) showing 1. Thickened blood vessels. 2. Congestion.

Symptoms of the disease noticed during the 2-3 weeks' observations are, on the whole, in line with the clinical signs of the disease described by Van Roekel and Olesiuk (1952), Fahey *et al.* (1953), Crawley and Fahey (1954) and Shah (1964). But these symptoms are also not uncommon in other respiratory infections of poultry, *i.e.*, Newcastle disease, infectious laryngotracheatis and infectious bronchitis (Van Roekel and Olesiuk, 1952; Tudor, 1953) or fowl pox, aspergillosis, and fowl cholera (Jungherr and Luginbuhl, 1952). Yet a rise in body temperature, nervous involvement, diarrhoea, pock like lesions on skin were not observed.

The post mortem lesions and the histopathological picture of the morbid tissues was enough to conclude that the organs primarily involved in field cases of the disease were mainly those of the respiratory tract. Thickening, exudation and congestion of the respiratory tract lining, with or without discreet necrotic foci, specially pronounced in the tracheal region, were among the more consistent lesions observed in the affected carcases of the present study. Under the microscope, there was marked hyperplasia, especially of the tracheal epithelium, accompanied with infiltration of epithelioid and reticuloendothelial cells; but giant cells were not always present. Degenerative changes in the tracheal lining involved also the deep-seated cartilage cells. Complete ossification was, however, not seen, although scattered osteoclasts and osteoblasts were noticeable.

Linear haemorrhagic streaks on the tracheal mucosa and the calcification of the tracheal cartilages observed in the sick birds of the present study does not seem to have been observed by the Western workers. If these lesions have some correlation with some secondary invaders, only prevalent in this part of the world, such as *Pseudomonas aerugenosa* (Shah, 1964), is not known. According to Hofstad (1965), presence of submucosal haemorrhages in the tracheal lining were indicative of infectious laryngotracheatis but the intranuclear inclusions, considered characteristic of this disease by Seifried (1931) were not seen. Haemorrhagic spots on the gizzard and proventricular submucosa, the lymphoid patches and follicles of the intestines, typical Newcastle disease lesions, were also not seen.

The hyaline cartillage in the tracheal rings of fowls affected with avian respiratory infections have recently been shown subject to changes leading to their ossification (Garside, 1968). As mentioned earlier, somewhat similar lesion was recorded in 13 of the disease affected carcases of the present study. Whether calcification/ossification was associated with the disease needs to be studied.

Wasserman *et al.* (1954), Fahey (1955), Gross (1956) and Glantz *et al.* (1962) are all of the opinion that fibrinous coating of serous membranes and mild

pericarditis in naturally occurring disease were due to a secondary infection by *Escherichia coli*. Stubbs *et al.* (1955) also reported peritonial adhesions and some pus in the abdominal cavity. According to Gross (1961, 1962) this organism could not readily infect the air sacs unless they were previously invaded by *Mycoplasma gallisepticum* alone or in combination with either infectious bronchitis or Newcastle disease virus. Marked exudation by the pericardium and serosal surfaces of the abdominal viscera have also been ascribed to the secondary infection by the coliform group of organisms (Gray, 1953). Therefore it may be concluded that both these organisms play a complicating role in naturally occurring chronic respiratory disease in West Pakistan.

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