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PATHOLOGICAL AND AETIOLOGICAL STUDIES ON GRANULOMATOUS DERMATITIS IN RACING PIGEONS IN ASSIUT GOVERNORATE

(With 10 Figures)

By
SANAA, A. H. EL-SHAMY, SHAHERA, M.R. ABDEL HASEEB and AZHAR, M. ABDEL AZIZ

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دراسة على باثولوجية وسبب تورمات التهابية في جلد الحمام الزاجل في محافظه أسيوط

سناء عبده حسن الشامي ، شهيرة محمد رشاد عبد الحسيب ، أزهار محمد عبد العزيز

أجرى هذا البحث على بعض الحمام الزاجل بمحافظه أسيوط.تم جمع تسعه حمامات مصابه بالالتهابات الجلدية الناتجة عن العدوى بالميكروب القولوني (E. Coli) وقد مثلت هذه العينات نسبه ٥ر٤% من حمام المزرعة (٢٠٠ حمامه). وقد أظهرت الدراسة أن نسبه الفقس في بيض حمام المزرعة قد انخفض من ٩٠% إلي ٣٣% مع ظهور هزال واضح على الطيور والتهابات في العين كما أمكن مشاهده تورمات جلديه على جلد الحمام المصاب. وبأجراء الفحص الميكروسكوبي لطبقه جلد الحمام المصاب وجد زيادة واضحة في سمك الطبقة الخارجية للقشرة (Epidermis) مع ترسيب لمادة الكيراتين في بعض الأحيان وكذلك زيادة في سمك طبقه ما تحت القشرة (Dermis) مع وجود التهابات في الانسجة الدهنية. وقد أظهر الفحص الميكروسكوبي لعينات الكبد للحمام المصاب وجود التهابات مزمنة مماثلة لتلك التي وجدت في الجلد في ثلاث حالات فقط.

SUMMARY

In Assuit Governorate, E. coli dermatitis was obseved in 9 (4.5%) out of a flock of 200 birds. The flock showed emaciation, conjunctivitis and

decreased hatchability rate. Histopathological examination revealed hyperkeratosis, parakeratosis, local chronic inflammatory nodular lesions in the dermis, panniculitis and cellulitis. Similar granulomatous lesions were seen in the three livers of these cases.

Key Words: *Racing pigeons, Granulomatous dermatitis*

INTRODUCTION

E.coli infection is widely spread in mammals and avian species and resulting in high economic losses (Gross, 1991). In avian species, serositis; omphalitis; salpingitis; conjunctivitis; hepatitis and enteritis were reported by Frommer et al.. (1990). Pigeons are of economic importance as meat production and racing. Recently racing pigeons are breed in adjacent to avian species, hense many avian diseases are seen in pigeons and cause high economic losses. Skin and feather problems are common in all avian species (Pass, 1989 and Riddal, 1996). In broilers, lesions of the skin and subcutis cause economic losses degradation, rejection and condamenation of carcasses (Morris, 1991).

In pigeons, as in other avian species, dermatitis may be of infectious or non-infectious causes. Infectious causes include viruses, bacteria, fungi and parasites. Viral dermatitis is seen in avian influenza, avian pox, Corona virus, Herps virus infection and Newcastle disease (Acland et al., 1984; Pass and Perry, 1984).

Bacterial dermatitis is described in association with E. coli, Pasteurella multocida, Staphylococci and Colstridial infections (Glunder 1990; May et al., 1982; Frazier et al., 1984; Cheville et al., 1988; Pass, 1989; Messier et al., 1993; Raidal, 1995 and Barnes and Gross, 1997).

Mycotic dermatitis is seen in aspergillosis and Candida albicans infection (Beemer et al., 1970; Richard et al., 1980; Okoye and Okeke, 1986 and Chute and Richard, 1997).

Dermatitis also occurs in ectoparastic infestations as in Cnedocropts species (Pass and Sue, 1983 and Okoye and Tkeme (1990).

Non-infectious dermatitis is stated by Frigg and Torhorst (1980). The dermatitis reaction may be acute, subacute or chronic with changes in the epidermis and dermis. The reaction may include one or more of the following features: Acanthosis, hyperkeratosis, parakeratosis, dyskeratosis, vasculitis, panniculitis, fibrosis, cellulitis and or

granulomatous reaction (Jubb et al., 1985 and Carlton and McGavin 1995).

The purpose of this study is to investigate the possible cause of feild cases of pigeon dermatitis and to describe the clinical symptoms, gross pathological and histopathological alterations in skin and internal organs of these pigeons.

MATERIAL and METHODS

Bacteriological examination:

Samples from affected areas of the skin were taken under sterile condition for bacteriological examination. Samples were inoculated in nutrient broth for 18-24 hours at 37°C, then subcultured on blood agar MacConkey and eosin methylene blue agar for 24 hours at 37°C. Pure colonics were picked up and stained by Gram's stain. Further identification of microorganisms was carried out according to Cruickshank et al., (1980) and Collins and Lyne (1991):

1- Colonial morphology: color, shape, size, odor and pigment production.
2- Morphology of microorganisms.
3- Biochemical reaction, Methyl red (M.R.), Voges-Proskauer (V.P), indole, citrate utilization, ureas and lactose fermentation.

Histopathological examination:

Specimens from the affected nine pigeons were taken from skin, liver, heart, lungs and kidnies, fixed in 10% neutral buffered formalin and processed routinely for light microscopy. Four micron thick praffin sections from each tissue were stained with hematoxylin and eosin. Additional sections, were stained with periodic acid- Sciff (PAS), Giemsa stain, Gram's stain and Ziehl-Neelsen's stain, Banchroft and Stevens (1982).

RESULTS

Bacteriological findings:

The suspected colonies were Gram negative bacilli, smooth glassy and translucent rose pink in color on MacConkey's media.

On blood agar, some strains were surrounded by haemolysis. The organism appeared motile, formed gas from glucose fermentation, ferment lactose, indole, M.R. (+) ve, V.P., citrate (-) ve and urease-ve.

Histopathologiacl findings:

The nine examined pigeons showed skin lesions generally seen at the posterior ventral region, caudal back, around cloaca and occasionally on the thigh. The skin at these areas was focally thickened and unfeathered. Such lesions were irregular in shape, raised above the skin surface, ranged from 0.5-1.5 cm in diameter and were multiple up to three nodule-like growths in the same area.

The lesions were usually bilaterally situated. The colour of the skin nodules was either faint yellowish brown or dark brown and covered with superficial scab. In some cases the nodular lesions could be difficulty enucleated. In one case the lesions were ulcerated, haemorhagic and exudes purulent-like exudate. The latter was thick, viscous and of dirty yellow colour. All the nodular lesions were rather firm in consistency and on cut section haemorrhagic caseated exudate of variable amounts could be seen. The subcutaneous tissue was frequently involved, edematous with sero-sanguinous fluid and the underlying muscles revealed peticheal haemorrhages.

Three pigeons showed small erosions at the junctions of the conjunctiva and skin of the eyelid. The erosions were covered with thick fibrinopurulent exudate. On examination of the viscera, lesions similar to those seen in the skin could be detected only in the liver.

On microscopical examination, two cases showed necrosis and ulceration of the epidermal cells on which bacterial clusters could be seen. The other specimens showed intact epidermal tissue with multiple focally thickened areas in which both of polyheadral cells and granular cells showed hyperplastic changes. The hyperplasia of the superficial layer was raised above the epidermal surface and associated either with deposition of excessive acidophilic kearatin layers, hyperkeratosis (Fig.1) or with retention of keratohyaline within the cells, parakeratosis.

On examination of the dermal and subcutaneous tissues, multiple granulomas, diffuse cellulitis, and panniculitis were consistently observed. The granulomas appeared as chronic local inflammatory cellular reaction with caseous centres (Fig. 2). Due to the consistent, cellular death and subsequent caseous necrosis, these granulomas could be considered as high turnover granalamas. The caseaus necrosis

appeared as granular structureless acidophilic debris. The later was surrounded by a cellular zone which differed in thickness according to the size of the granuloma. It consisted mainly of mononuclear lymphocytes, plasma cells, macrophages, epithelioid, giant cells and some heterophils (Fig. 3). Most of the macrophages appeared large vacuolated cells with peripherally situated nucleus (Fig. 4). The foreign body multinucleated giant cells intermingled between the epithelioid and macrophage cells. The nuclear arrangement within the cytoplasm, either took central location or had peripheral situation or involved the whole cell cytoplasm (Fig. 5). Some of these multinucleated giant cells undergone hyalinosis and appeared as highly acidophilic irregular structures, others showed phagocytized bacillary-like debris within their cytoplasm. In such cells the nuclei arranged as horse-shoe shape taking an longhan's like shape (Fig. 6). Mostly the granulomas were surrounded with connective tissue capsule or trials for encapsulation by proliferation of fibroblasts and fibrocytes. Many large granulomas showed secondary or even teritary daughter small granulomas in their walls.

The dermis, subcutaneous tissue and even in-between the muscle fibers of the muscular layer, diffuse infiltration with inflammatory cells mainly heterophils, lymphocytes and macrophages was seen. In addition erythrocytic extravasation and angiopathic lesions were observed. In the all the examined sections this cellulits was distinctly observed.

The subcutaneous adipose tissue demonstrated features of panniculits in which the interlobar and interlobular tissues revealed signs of acute inflammatory reaction. Hyperaemia and ecchymotic extravasations were occasionally seen. Heterophilic infiltration was also demonstrated. The adipose tissue cells revealed acidophilic intracellular crystalline like debris (Fig. 7). In addition the vascular ramifications in the dermis revealed angiopathic changes these changes included endothelial dystrophic and hyperplastic changes, the media of the arterioles showed vacuolated smooth muscle cells. The perivascular areas revealed inflammatory oedema.

On examination of liver sections diffuse dystropic changes were seen. These changes were mostly in the form of proteinous dystrophy and fatty change (Fig. 8). Many hepatocytes revealed coagulated, condensed acidophilic cytoplasmic granules. In some sections, focal mononuclear cell infiltrations were seen (Fig. 9). The intrahepatic

vascular branches revealed endothelial degenerative changes and smooth muscle cell vacuolation (Fig. 10).

DISCUSSION

In our findings hyperkeratotic, parakeratotic dermatitis were seen above pigeon skin nodular lesions from which E. coli was isolated. Similar findings were described by Randall et al., (1984). Such changes could be related either to the E. coli toxin or other mechanical causes. Litter or cages may play the role of superficial epidermal irritation and even local infection with the E. coli or other agents. Similar conditions were reported in broilers in staphyloccal and E. coli infections (Pass, 1989) and in quails (Raidal, 1995).

In the present investation, the gross granulomatous lesions were mostly in the unfeathered areas probably as predliction sites of E. coli dermatitis or even the exposed sites for wound and predisposing factors for infection. Similar sites were reported in dermatitis of broiler and turkeys (Gonders and Barness, 1987 and Glunder, 1990).

Microscopically, local inflammatory cellular reaction with caseous centers were seen. The wall consisted of mononuclear lymphocytes, plasma cells, macrophages, epitheliod cell, giant cells and fibroblasts. These granulomas were described as high turnover granulomas (Rubin and Farber, 1994). This type of granulomas was described in mycobacterium infection, fungi, helminths and their ova and many organisms that replicate intracellularly. In this study, necrosis of the inflammatory cells especially macrophages is probably due to hypersensitivity to E. coli or its toxins as well as the cytotoxic factors released from the synsethized lymphocytes.

Subcutaneous cellulitis was seen as diffuse inflammatory reaction between the granulomatous lesions. The reaction consisted of hyperaemic blood vessels with angiopathic alterations and inflammatory cellular reaction involving heterophils, lymphocytes and macrophage.

Escherichia Coli was similarly isolated from avian cellulitis and also induced the disease experimentally in broilers (Peighambar et al., 1995). The outhors described in association accumulation of lymphatic fluid in the abdominal cavity and considered it as a possible biological predisposing factor for cellulitis. In present study, no signs of ascitis could be detected. In our opinion, ascitis may be a result of cellulitis if

occurs in the abdominal area or adjacent to the coelomic cavity. The presence of cellulitis could be assumed to the escaped aetiological agent through macrophages or due to the diffusion of its toxins to the neighbouring sites.

In this paper, degenerative and inflammatory changes were seen in the blood vessels of dermis and subcutis. These changes were similarly described in E. coli infections (Carlton and MacGavein, 1995). These changes could be attributed to the aetiologic agent and/or its toxins. Acute inflammatory reaction in the interlobular and interloblar adipose tissues could be also attributed to the toxins of the organism.

Hepatopathic alterations included dystrophic changes, focal mononuclear cell infiltration and degenerative changes in the intrahepatic vascular ramifications. These changes could be attributed to the systemic distrubution of the bacterial toxins. The dystrophic changes in hepatocytes may be also attributed to the vascular degenerative changes.

The gross occular lesions described was also reported in relation to E. coli. infections (Cheville et al., 1988). An experimental work is planned and will be published later.

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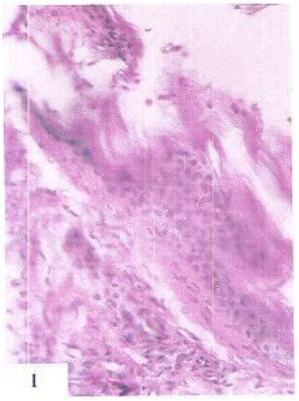
LIST OF FIGURES



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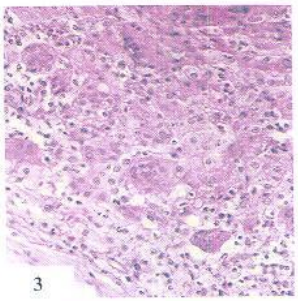

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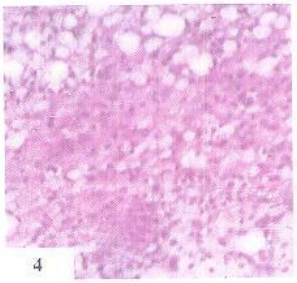

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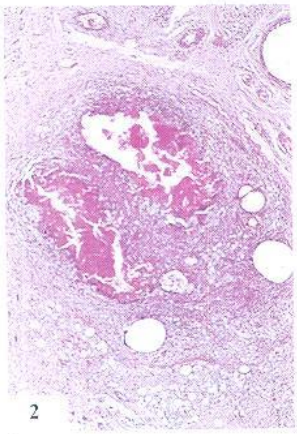

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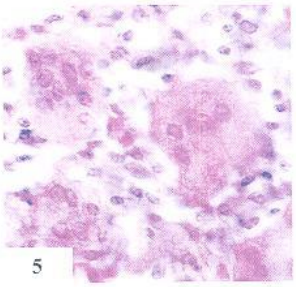

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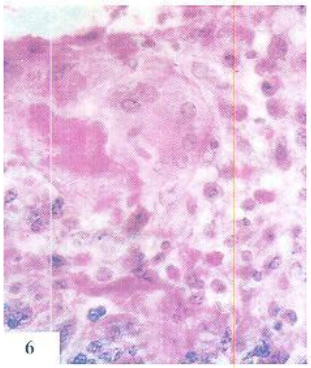

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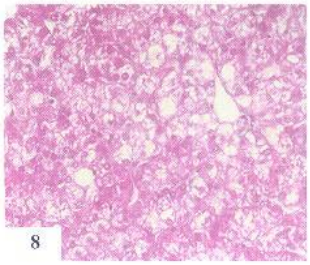

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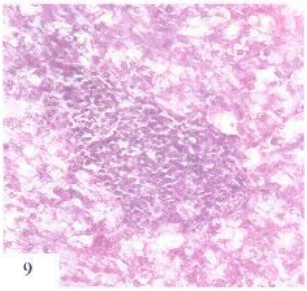

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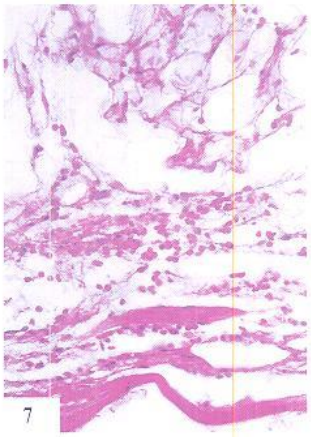

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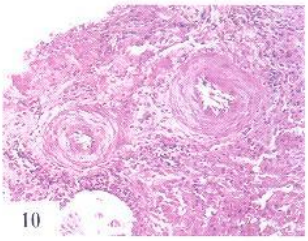

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