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دراسة عن وجود مرض سرطاني في الخلايا الليمفاوية
في الحمام

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من بين أفراد قطيع من الحمام مكونه من 20 حمام وجد زوج من الحمام تحتوي كبدة على نمو سرطاني بنزين من 3035 جم. تمت دراسة الطبيعة الباثولوجية لهذا النمو، حيث وجد أنه من الاحتمال أن يكون نوع من أنواع سرطان الخلايا الليمفاوية.
A naturally occurring lymphoproliferative neoplastic disease resembling reticuloendotheliosis was observed in one flock of Egyptian pigeon (Assiut governorate). The neoplastic mass were detected mainly in the liver. Histopathologically revealed pleomorphic proliferative infiltration with dark staining large cells, lymphocytes of different sizes, reticulum like cells, as well as mature and immature mononuclear cells. Histochemical studies revealed intracytoplasmic inclusion bodies. Based on the histopathological and histochemical studies, this lymphoproliferative neoplastic disease may be reticuloendotheliosis.

INTRODUCTION

Reticuloendotheliosis is a neoplastic disease caused by ancorna virus affecting several avian species (WITTER, 1977). The causative agent of this disease was firstly isolated from tissue of turkey with lesions resembling those of lymphoid leukosis. ROBINSON and TWEHAUS (1974). Some authors stated that reticuloendotheliosis is a true neoplas (THEILEN, et al., 1966, CAMPBELL et al., 1971, and LINNA & THOMPSON, 1974), however other authors stated that the lesion contain non-neoplastic presumably reactive cells (WITTER, et al., 1970) or are frankly necrobiotic (PURCHASE, et al., 1973).

A lymphoproliferative disease (reticuloendotheliosis) had been described as a naturally occurring neoplasam among three flocks of Japanese Quail in Mexico SCHAT, et al. (1975). In the available literature no reports exist on the natural occurrence of leukemia lymphoproliferative disease or reticuloendotheliosis among pigeon and especially in Egypt. We now report the natural occurrence of a lymphoproliferative neoplastic disease resembling reticuloendotheliosis in one flock of Egyptian pigeon in Assiut governorate.

MATERIAL and METHODS

A breeder having 30 pairs of mature pigeons in Assiut Governorate observed, that one pair had lost appetite, emaciated roughed feather and the abdomen was greatly enlarged where the birds unable to stand. Clinical examination of all the flock (60 birds) revealed no abnormality except these clinical signs in this one pair. The clinically affected birds was sacrificed. In these birds a tumour mass was found in the liver weighing 300 and 325 gm, where the liver tissue appeared as a remnant at the periphery of tumour mass. Five birds from the flock were sacrificed randomly and having no gross abnormality. Multiple cut sections were done and small pieces of the tumour tissue including the liver as well as the other

organ were taken and fixed in 10% neutral buffer formaline and Carnoy's fluid. The fixed tissues were embedded in paraffin wax. Section in thickness of 4 - 6 M were done and stained with hematoxylin and eosin, PAS and feulgen reaction.

RESULTS

Gross pathology:

The tumour masses which were found in the liver of both birds were grayish white in colour, hard in consistancy and resembled the lymphoid tissue weighing 300 and 325 gm. The hepatic tissue appeared as a remnants at the periphery of the tumour with pale yellow colouration. The intestine and spleen were congested. The lungs were pale in colour and studded with a dark black coloured foci.

Histopathology:

The tumour consists of masses of cells. These masses were irregular in shape and of different size. They were encircled with fine connective tissue stroma (Fig. 1). The tumour cell have two arrangement either accumulated in masses (previously described) or found infiltrating the hepatic lobules (Fig. 2). The neoplastic cells were highly pleomorphic. According to the size and micromorphology of the nucleus three types of cells could be distinguished. Large dark staining highly pleomorphic cells with diameter 7.5 - 12.5 M. The nucleus of this cells was darkly stained, hyperchromatic, had oval, rounded, quaderangular, triangular or elongated shape. The cytoplasm of this cells was abundant and slightly basophilic (Fig. 3). Medium sized cells with a diameter ranged from 7 - 9 u, this cells had open vesicular nucleus, which contained one or two nucleolus. The cytoplasm was less abundant than the proceeding cell and having a basophilic character (Fig. 4). The third type of cells were the lymphoid cells which were widely distributed in the neoplastic mass. Few amount of large reticular cells was observed. Also mitotic figers in the neoplastic cells were observed in 3 - 4% of this cells.

The large dark neoplastic cells showed intracytoplasmic inclusion bodies. This inclusions were periodic acid and feulgen reaction positive (Fig. 5). The liver cells showed dystrophic changes and some of them were filled with yellow greenish pigment (bile pigment). Kuffer cell activation and lymphoid cell infiltration of the portal tract was observed. Some liver lobules were atrophied and the others were completely disappeared and replaced by neoplastic tissue.

The changes in the other organs spleen, intestine, kidneys and heart were slight congestion with necrobiotic and strophic changes. The lung showed focal areas of anthracosis.

Histopathological examination of the organs belonging to the clinically normal birds revealed no pathological changes.

DISCUSSION

A naturally occurring lymphoproliferative neoplastic disease was found in one flock of Egyptian pigeon in Assiut governorate. The tumour like lesions were detected in the liver and project into the abdominal and thoracic cavity. The neoplastic tissue closely resembled the lymphoid tissue. Histopathological examination revealed that the tumour consists of masses of neoplastic cells surrounded by fine connective tissue stroma. Four types of cells could
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be seen. Highly pleomorphic dark staining large cells, cells with open vesicular nucleus, lymphoid cells of different sizes and reticular like cells. These above mentioned gross and histopathological changes along with the necrobiotic changes observed in the liver cells are in harmony with the lesion of reticuloendotheliosis described by WITTER, (1977) in turkey and SCHAT, et al. (1975) in Japanese Quail in Mexico. The lesion observed in our material was primarily neoplastic, however necrobiotic changes could be considered as secondary due to the effect of the neoplastic tissue. The kupffer cell activation as well as the lymphoid cell proliferation in some parts of the liver could be considered as an immune mechanism.

The etiology of the avian lymphoproliferative neoplastic disease can not be established on gross and histopathology alone. Three groups of oncogenic viruses, Marek's disease herps virus 6MDHV, reticuloendotheliosis virus (REV) and Avian leukemia virus (ALV) have been studied in detailed in chickens (SCHAT, et al. 1975). In our material the presence of intracytoplasmic inclusion bodies in the neoplastic cells indicating the viral etiology of the neoplastic disease, at least one of the above mentioned group may be incriminated as a causative agent.

The gross appearance of the tumour were greatly resemble those of Marek's disease and avian leukosis. The difference between reticuloendotheliosis, Marek's disease and avian leukosis could be carried out to some extent on histological bases. The absence of pathological lesions in the nerves together with the presence of large dark staining cell and fine connective tissue stroma are atypical for Marek's disease lesions (SCHAT, et al. 1975). The pleomorphism of the tumour cells in our observation differs from the uniform type of lesions described by WIGHT (1963) and LÖLÍGER and SCHUBERT (1967), which they found to be equivalent to leukosis in chickens. Moreover the presence of lymphoreticular cell in the neoplastic lesions should be of considerable diagnostic value since such cells are not typical of either lymphoid leukosis or Marek's disease.

REFERENCES


Fig. (1): Showed fine c.t. stroma
H.E. Stain (25 x).

Fig. (2): Showed tumour cells infiltrating the liver cells.
H.E. Stain (25 x).

Fig. (3): Showed large staining cells. H.E. Stain (40 x).
**Fig. (4):** Showed medium sized cells H.. Stain (40 x).

**Fig. (5):** Showed intracytoplasmic inclusion bodies
Folgen reaction (100 x).