Investigation of pulmonary tumors in Equidae

Naglaa M. A. Al kalamawey
Researcher- Pathology Department- Animal Health Research Institute

Abstract
Respiratory system health represents one of the most important factors for equidae which represent hard working animals. This study investigated the different types of pulmonary tumors in equidae. Lungs of seventy animals including: (52 out of 70) donkeys and (18 out of 70) mules had been collected from slaughtered equidae in Giza Zoo. Tissue samples were collected randomly from lungs with special care for abnormal lesions. The results showed close incidence of adenocarcinoma, pulmonary blastoma and bronchial gland adenoma occurrence in equidae. .

Key words: *bronchial gland adenoma , equidae, adenocarcinoma.*

Introduction

In equine, tumors of the respiratory tract occur infrequently **(Scarratt *et al*., 1998)**. The most frequently reported primary pulmonary tumor, in equine, is the granular cell tumor **(Anderson *et al*., 1992).**

Non-small cell lung carcinomas (Non-SCLC) account for almost 80% of lung cancers, of which 40% were adenocarcinomas **(Peng *et al*., 2011)**.

In 1981 WHO classified lung adenocarcinomas into the following four subtypes: acinar, papillary, bronchioloalveolar, and solid with mucin formation **(Sobin and Yesner, 1981)**. In the 1999 WHO/ International Association for Study of Lung Cancer (WHO/IASLC) classification, noticed that the majority of pulmonary adenocarcinomas showed a mixture of the four major subtypes mentioned above and it was proposed that such tumors can be classified as adenocarcinoma, mixed subtype.

The acinar variant is the most common form and is defined by the WHO as having, "a predominance of glandular structures, i.e., acini and tubules with or without papillary or solid areas. The better differentiated tumors form orderly glands lined by tall columnar cells with a regular array of nuclei.

Papillary adenocarcinomas are recognized as having a predominance of the papillary structures. Papillary architecture begins with the protrusion of cells into the gland lumen. Generally the better differentiated papillary variants show a core of fibrous connective tissue which is covered by a single layer of uniform cuboidal to columnar cells. Bronchioalveolar carcinomas are defined as an adenocarcinoma in which cylindrical tumor cells grow upon the walls of pre-existing alveoli **(Travis *et al*., 1999)**.

Solid carcinomas with mucus formation are recognized as, poorly differentiated adenocarcinomas lacking acini, tubules and papillae but with mucin containing vacuoles within many tumor cells **(Kumaki *et al*., 2001)**. Another pulmonary tumor is pulmonaryblastoma. Pulmonary blastomas [PB] are rare malignancies, comprising 0.5% of all primary lung malignancies. Well-differentiated fetal type adenocarcinomas are one of three morphologic varieties of pulmonary blastomas. These lesions are composed of immature epithelial and/or mesenchymal components and a resemblance to fetal lung has been noted **(Koss *et al*., 1991)**. Pleuropulmonary blastoma (PPB) and pulmonary blastoma (PB) constitute two rare, but clinically and pathologically, distinct pulmonary tumors characterized by embryonic structure. PB is an aggressive biphasic tumor formed by mixed malignant epithelial and mesenchymal elements that mimic embryonic structure **(Ashworth, 1983)**. These tumors are described in humans **(Koss *et al*., 1991)**, and in adult cattle (**Kelly *et al*., 1994)**.Also a single-case reported exist at 620-day-old rat **(Chen and Frame, 1991)**, at 4-year-old Lhasa Apso dog **(Watson *et al*., 1993)** and in adult horse **(Perez-Ecija *et al*., 2009)**, in addition to a rec-

ord of one case of PleuropulmonaryBlastoma in an equine fetus by **Woolford *et al*. (2010)**.
This study aimed to record the different types of pulmonary cancer in equidae.

Material and Methods

Tissue samples from lungs of seventy animals' including52 donkeys' and18 mules had been collected from slaughtered animals in Giza Zoo and kept in 10% formalin. Formalin – fixed tissues were dehydrated in alcohol, cleared in xylene and embedded in paraffin wax. Sections of 4- 5 µm thick were deparaffinized in xylene and rehydrated in graded alcohols and water then were stained with hematoxylin and eosin (H&E) according to **Bancroft *et al*. (1996)**.

Results

The examined cases of tumors could be classified into:

I. Benign tumor of epithelial origin:

Bronchial mucous gland adenoma (Goblet cell adenoma) was detected in [4] cases of total 70 investigated cases (5.7 %).

The most postmortem character of that type was excess mucous in airways. These tumors were formed of mucous – containing acini of tubulopapillary structures, lined by single layer of cuboidal to columnar epithelial cells with a pale and foamy cytoplasm and characterized by the predominance mucous goblet cell differentiation of the neoplastic epithelium (Fig.1). Frond-like structures of connective tissue core lined by multi layers of well differentiated goblet cells were also detected (Fig. 2), while other areas showed stratification of neoplastic cells with ill- differentiated tumor cells (Fig.3). The tumor was well demarcated but has no capsule.

II- Malignant tumors of epithelial origin:

Adenocarcinomas grossly present with the "three P's" - peripheral, pigmented and puckered. Commonly lesions were found near the pleural surface (peripheral) which is retracted (puckered) over the neoplasm. The cut surface is often white to pale gray with black anthracotic pigment and glistens in cases with mucin production. Desmoplastic reactions are often associated with adenocarcinomas and give the tumor a firm fibrous consistency. In some cases adenocarcinoma was associated with cavitary lesions.

Preneoplastic lesions in form of atypical adenomatous hyperplasia of epithelial element with adjacent collapsed alveoli (Fig.4: a & b).

The following classifications depend on the most predominant tumor growth pattern.

A. Papillary Adenocarcinoma: This pattern of tumor was recorded in [4] cases of total 70 cases with percentage of 5.7 %. Lesions were characterized by peripheral location of an adenomatous lesion showed predominance of anastomsing network of papillary structures. The better differentiated papillary variants composed of central, slightly vascularized connective tissue core, covered with one or more layers of large ciliated columnar or cuboid epithelial cells (Fig.5) with (Fig.6) or without (Fig.7) mucous production covered by a single layer of uniform cuboidal to columnar cells. Some cases showed papillary protrusion (Fig.8).

B. Pulmonary Blastomas (Acinar adenocarcinoma):

i. Blastoma: well-differentiated fetal adenocarcinoma (WDFA) was recorded in [2] cases (2.85 %). This type was in most cases located periphery and associated with pleural scarring. The images contained acinar adenocarcinoma/ well-differentiated fetal type adenocarcinoma exhibiting tubuloacinar growth with a predominance of glandular formation. Different stages had been recorded. Some areas showed nodular aggregation of large cuboidal neoplastic cells given the feature of small compact adenomatous nodular lesion (Fig.9), other areas showed lumen formation with canalization of the nodular structure given an acinar appearance (Fig.10).

ii. Classic Biphasic pulmonary Blastoma [CBPB): One case (1.4 %). In this pattern, neoplastic tissue was comprised of solid areas, loosely arranged blastematous mesenchyme, fenestrated by multiple variable-diameter cystic lumina, formed by trabeculae of loose mesenchyme and lined by a single layer of non-ciliated cuboidal or attenuated epithelium (Fig.

11).
Cavity formations of different sizes were detected in association with some adenocarcinomatous lesions. The cavity in most cases had lining epithelia (Fig.12).

Discussion

Lung cancer is the leading cause of cancer death worldwide. Both principal factors known to cause lung cancer, cigarette smoke and asbestos, induce pulmonary inflammation, and pulmonary inflammation has recently been implicated in several murine models of lung cancer **(Dougan *et al*., 2011)**.
The genetic background of tumor cells in lung cancer is not homogenous. In terms of the cancer stem cell hypothesis, cancers arise from stem cells that have accumulated oncogenic mutations for transformation **(Kim *et al*., 2005)**. Sporadic and step-by-step accumulations of mutations in cancer stem cells can generate a variety of tumor clones with a distinct genetic background **(Ogata *et al*., 2011)**.
The peripheral lung progenitor cell (Clara, type 2, or other specified cell) is considered the origin of atypical adenomatous hyperplasia (AAH) and Bronchioloalveolar adenocarcinoma (BAC) **(Travis *et al*., 2004)**.
According to the 1999 WHO/IASLC classification, atypical adenomatous hyperplasia (AAH) was added to the group of preinvasive lesions, because it is thought to be a precursor of adenocarcinoma **(Travis, *et al*.; 1999)**. Atypical adenomatous hyperplasia is a millimeter-sized nodular lesion, in which the alveoli and respiratory bronchioles are lined by slightly atypical pneumocytes **(Suzuki *et al*., 1997)**. AAH is distinguished from nonmucinous BAC in that it has less cytological atypia, cellular crowding, and overlapping of nuclei, as well as less prominent nucleoli, and smaller cell size **(Travis, 2001)**. This explains the various degrees of cytologic atypia and crowding with subsequent adjacent collapsed alveoli detected in our examined cases which manifested primary stages of cancer development.
Bronchioloalveolar adenocarcinoma (BAC) arising in the peripheral lung is the prototype of human lung adenocarcinoma. It is generally accepted that BAC, at least in part, develops from its precursor atypical adenomatous hyperplasia (AAH) in the fashion of the adenomacarcinoma sequence **(Kitamura, *et al*.; 1999)**.These facts in agreement with peripheral location of neoplasicadenocarcinomatous lesions in our study.
During tumorigenesis the original tissue specific pyruvate kinase (PK), one of the key glycolytic enzymes in mammalian tissues, (type L, R, and M1) is replaced by PKM2 **(Peng *et al*., 2011)**. This change could help tumor cells to produce lactic acid through glycolysis, rather than to produce energy through mitochondrial oxidative phosphorylation, and help tumor cells to survive in low glucose and low oxygen environments and facilitate tumor invasion (**Christofk *et al*., 2008).** Moreover, overexpression of PKM2 was found to be correlated with a low degree of differentiation and the severity of epithelial dysplasia **(Peng *et al*., 2011)**.
Significance central scarring in our investigated adenocarcinoma cases has been debated for several decades. Initially, it was thought that adenocarcinomas arose in association with preexisting scars, and the concept of scar carcinoma was advanced **(Bakris *et al*., 1983)**.
Scaring and collagen formation in our examined adenocarcinomas was favored by the concept that the scar in most lung adenocarcinomas was caused by tumor invasion **(El- Torky *et al*., 1985)** which grow invasively and promote accumulation of extracellular matrix through increased expression of Tissue Inhibitors Matrix metalloproteinases 2 (TIMPs) by the tumor cells **(Kumaki *et al*., 2001)**.
The mechanisms proposed for this scarring include vascular **(Kolin 1988)** or airway occlusion by tumor resulting in alveolar collapse and a dense fibrosing scar **(Kung *et al*., 1985)**, which were detected in the most of our detected cases.
The detected fibrous connective tissue core in the cases of papillary adenocarcinoma cases showed the well-differentiated nature of this subtype **(Kumaki *et al*., 2001)**.
Most of adenocarcinoma detected cases were

associated with subpleural scars which could be due to a variety of causes, including old infarcts, healed pneumonitis or granulomas, or trauma, meanwhile stratification and loss of uniformity in some cases was associated with a loss of differentiation **(Kumaki *et al*., 2001)**.
The relationship between adenocarcinomas and their inflammatory microenvironment revealed a dual role for the immune system in tumor development. Consistent with a role for chronic inflammation in the onset of these adenocarcinomas, the inflammatory cytokine IL-6 acted as an autocrine growth factor in this system, and restoration of immune function reduced lung cancer incidence **(Dougan *et al*., 2011)**. In human lung cancer cells, activation of EGFR signaling can induce production of the inflammatory cytokine IL-6 **(Gao *et al*., 2007)**. These findings suggest a model whereby cytokine deficiency leads to oncogenic inflammation that combines with defective antitumor immunity to promote lung tumor formation **(Herbst *et al*., 2008)**. Tumors in this system were suggested to arise through a combination of tumor-promotinginflammation and failure of spontaneous antitumor immunity **(Dougan *et al*., 2011)**.

Epidermal growth factor receptor (EGFR) tyrosine kinase inhibition is an active strategy in non-small cell lung cancer (NSCLC) **(Fukuoka *et al*., 2003)**. EGFR mutation has been recognized as a crucial step in the transformation of alveolar epithelial cells. A recent report also suggested that the activating mutation of the EGFR gene occurs as an early event during carcinogenesis of lung cancer **(Tang *et al*., 2005)**.
Regarding to another investigated pulmonmary tumor, pulmonary blastoma which is a rare tumor, and was originally termed 'embryoma' by **Barnard in 1952**. The term 'blastoma' was introduced in **1961** by **Spencer** to reflect that the tumor arises from pulmonary blastema in a manner similar to other tumors developing from fetal tissues. Subsequently, pulmonary blastomas have been further subdivided by **Koss *et al*. (1996)** into three categories: Classic biphasic pulmonary blastoma (CBPB), well-differentiated fetal adenocarcinoma (WDFA) and pleuropulmonary blastoma (PPB) of childhood.
Both WDFA and CBPB types were recorded in our investigation and exhibited the microscpoic features which had been reported as pleuropulmonary blastoma by **Woolford *et al*. (2010)**. The stages of tumor canalization in our WDFA investigated cases revealed neoplastic cells –nests formation, nest canalization as previously described by **Spencer (1961)** and end with development of acinar adenocarcinoma (WDFA– type). On the other hand CBPB lesions were formed of comprised solid areas of loose mesenchyme, fenestrated by small ducts or large cystic areas lined by cuboidal epithelium as described by **Woolford *et al*. (2010).**
Pulmonary blastoma is a carcinosarcoma in which the sarcomatous element is composed of an undifferentiated embryonic type of connective tissue and the epithelial element, arranged in a tubular pattern, is embedded **(Barson *et al*., 1968)**.
The histopathological features of our investigated cases could be explained by **Spencer (1961)** who considered that the pulmonary blastoma arose from primitive mesenchymal cells of the pulmonary blastema will be differentiated along two different pathways. On the other hand they were thought to form the stromal elements of the tumour consisting of fibroblastic and myoblastic elements, or they formed nests of cells which became canalized and constituted the epithelial element of the tumour. **Willis (1960)** stated that adult mesenchymal tissue may contain undifferentiated cells which sometimes revert to the embryonic state. Their multiplication may then result in the genesis of both epithelial and mesodermal elements. Carcinosarcoma was defined by **Willis (1960)** as either simultaneous malignant neoplasia in epithelial tissue and the surrounding mesodermal stroma, or a sarcomatous change occurring secondarily in the stroma of a carcinoma. A carcinosarcoma arises therefore from mature cells in two separate germ layers and differs from a blastoma which has its origin in

a pluripotential cell of a single germ layer. **Barson (1968)** feel it would be more correct to regard it solely as a pulmonary carcinosarcoma which has a fortuitous resemblance to foetal lung. It has the capacity for progressive and unlimited growth of tissues which are derived from that single germ layer.

The bronchial adenoma which was detected in the present investigation showed the same histopathological features described as papillary adenoma by **Meuten (2002)**.

Histopathologically, different patterns of pulmonary adenocarcinoma had been detected in a variety of phenotypic manifestations. The detected tumors were composed entirely or largely of coarse distorting and irregular papillary or acinar glandular structure with central fibrotic scar, leading to the diagnosis of adenocarcinoma with mixed subtypes.

Bauermeister *et al.* (1966) and **Souza *et al.* (1965)** classified the pulmonary blastomas as a distinct type of carcinosarcoma. The rarity of this tumour and the variable natural history of the previously published cases warrant a case report in order to clarify further the characteristics of this unusual group of tumours **(Barson *et al.*,1968)**. Therefore, we would be in agreement with this view of **Bauermeister *et al.* (1966)** and **Souza *et al.* (1965)**.

Conclusion: It is more likely that it is a true carcinosarcoma because its epithelial element is adenomatous, incidentally has some resemblance to foetal lung, and would be better named a pulmonary adenocarcinosarcoma.

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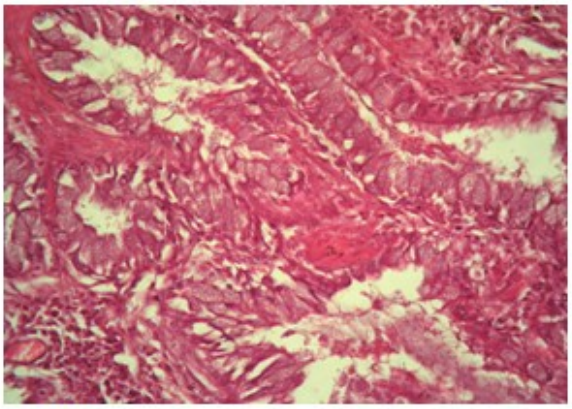
Figure legends

- **Fig. (1):** Photomicrograph of bronchial mucous gland adenoma showing; mucous containing acini lined by single layer of well differentiated goblet cells. **H&E X 400**
- **Fig. (2):**Photomicrograph of bronchial mucous gland adenoma showing; tubulopapillary structure with frond- like formation composed of connective tissue core and lined by single epithelial layer. **H&E X 200**
- **Fig. (3):** Photomicrograph of bronchial mucous gland adenoma showing; stratified ill-differentiated tumor cells. **H&E X 400**
- **Fig. (4): a**- Photomicrograph of atypical adenomatous hyperplasia, the alveoli lined by large cuboidal cells. **H&E X 400**

b- Adjacent areas showing collapsed alveoli. **H&E X 200**

Fig.(5): Photomicrograph of papillary adenocarcinoma showing; vascularized thick connective tissue with significant collagen deposition which covered by large ciliated cuboidal epithelia. **H&E X 400**

- **Fig. (6):** Photomicrograph of papillary adenocarcinoma showing; adenomatous lesion with mucous production**. H&E X 200**
- **Fig.(7):** Photomicrograph of papillary adenocarcinoma without mucous production.**H&EX 200**
- **Fig.(8):** Photomicrograph of papillary adenocarcinoma showing; papillae protrusion.**H&EX400**
- **Fig. (9):** Photomicrograph of Acinar adenocarcinoma [WDFA] showing; nodular aggregation of large cuboidal cells given a feature of compact adenomatous nodular lesion. **H&E X 400**
- **Fig. (10):** Photomicrograph of Acinar adenocarcinoma [WDFA] showing; canalization of the nodular neoplastic lesion forming acini with different sizes and shapes. **H&E X400**
- **Fig. (11):** Photomicrograph of Acinar adenocarcinoma [CBPB] showing; thickening of the pleura with solid mass of loosely arranged mesenchyma had foci of inflammatory cells infiltration and multi cystic lumen formation. **H&E X 100**
- **Fig. (12):** Photomicrograph of adenocarcinoma with cavitary lesions. Cavities lined by single epithelial layer and surrounded by fibrous parenchyma infiltrated by inflammatory cells. **H&E X 100**



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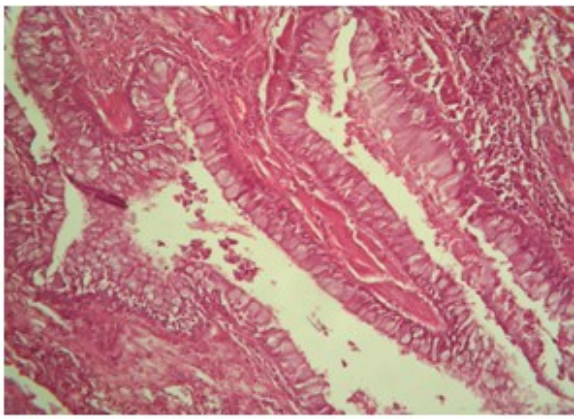

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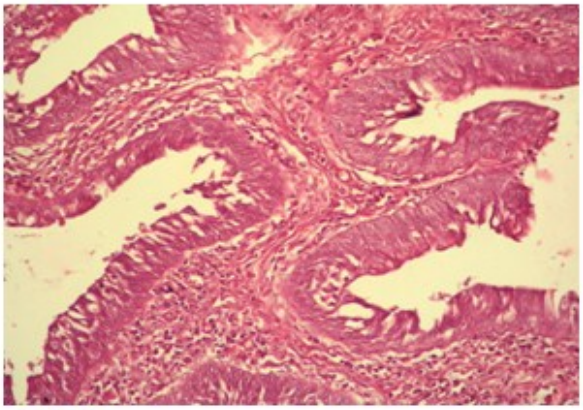

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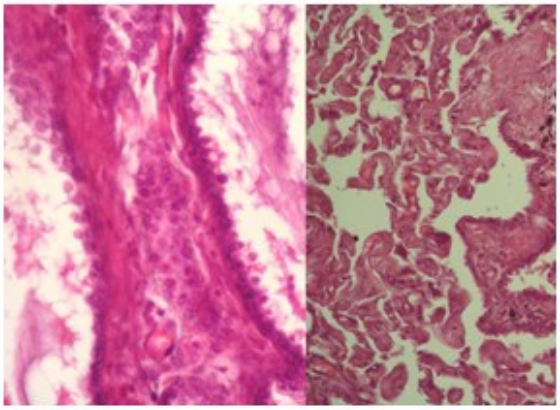
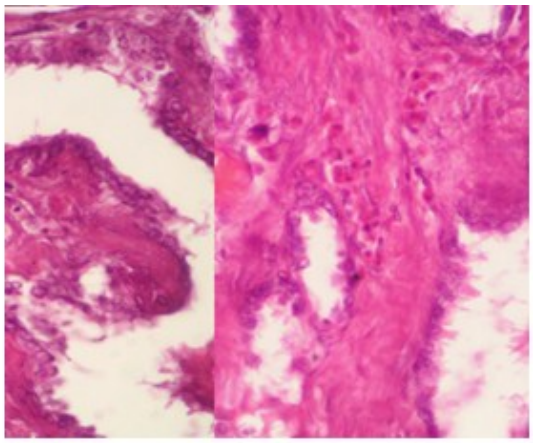


Fig.4: a } b }



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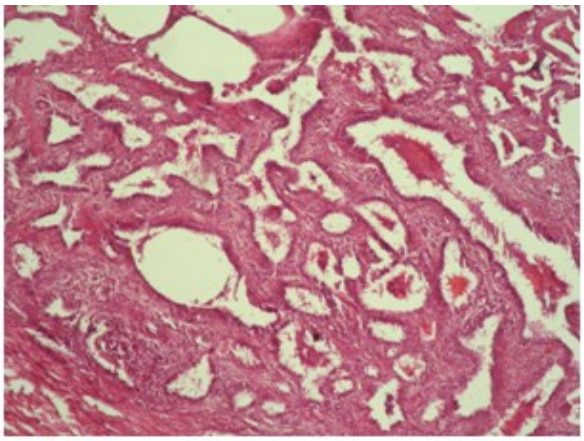

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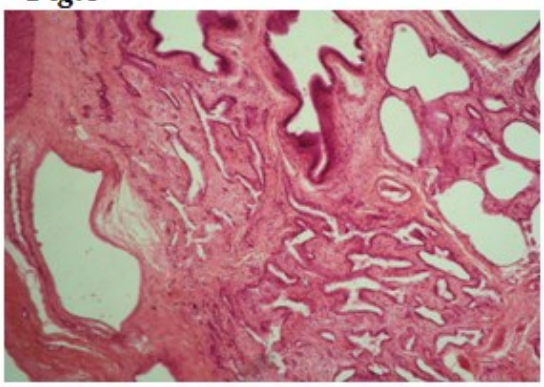

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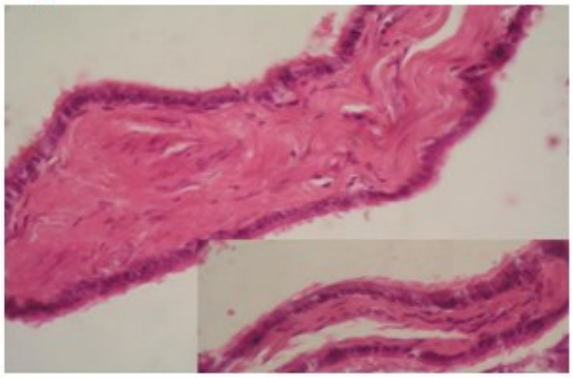

Fig.8

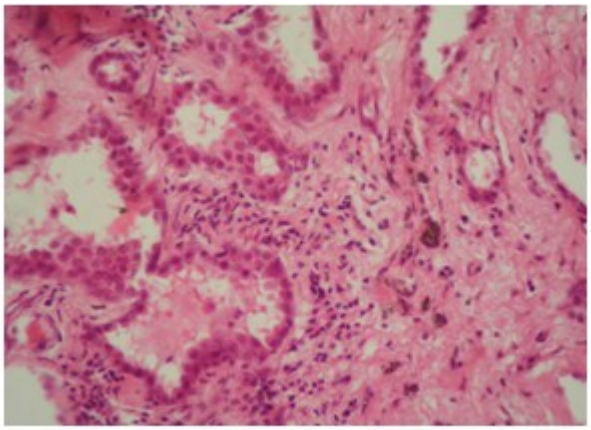

Fig.9

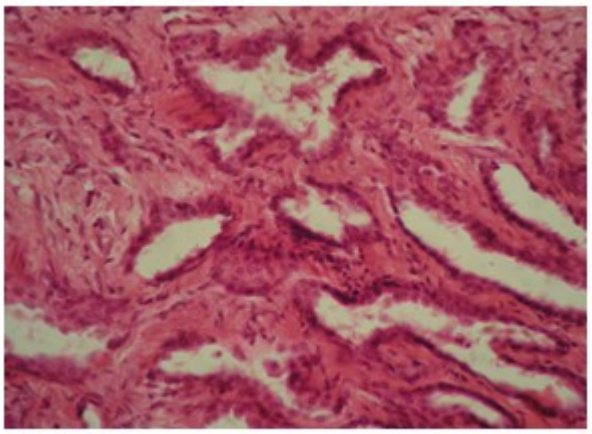

Fig.10

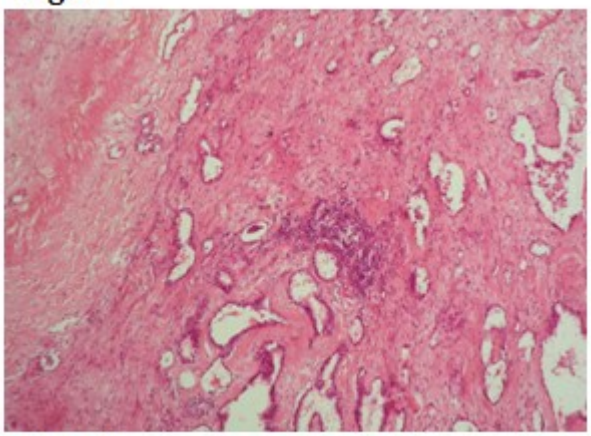

Fig.11

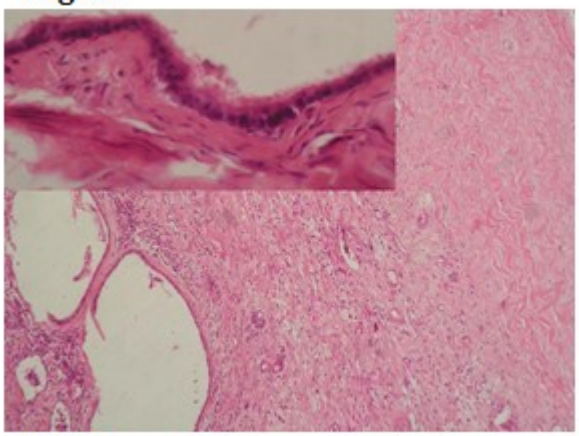

Fig.12

فحص الأورام الرئوية فى العائلة الخيلية

نجلاء محمد عبد العزيز القلماوى

باحث – قسم الباثولوجيا- معهد بحوث الصحة الحيوانية

الملخص العربى

تمثل صحة الجهاز التنفسى واحد من أهم العوامل لحيوانات العائلة الخيلية و التى تتميزبطبيعة العمل الشاقة. هذه الدراسة للتعرف على من أنواع الأورام الرئوية المختلفة فى الفصيلة الخيلية. تم تجميع الرئة من حيوانات العائلة الخيلية وقد شملت 52/70 من الحمير و18/ 70 من البغال الواردة كحيوانات ذبح بحديقة الحيوان بالجيزة. عينات الأنسجة تم تجميعها عشوائيا من الرئة مع أهتمام خاص للحالات الغير طبيعية. و قد أظهرت النتائج التقارب فى نسب حدوث أنواع السرطان الرئوية فى هذه الدراسة و التى شملت الأورام الغدية ذات الأصناف المختلطة و الأورام الرئوية الجذعية و الأورام الغدية الشعبية.