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A case of bronchogenic adenocarcinoma in a guinea pig *(Cavia porcellus)*

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Summary

A spontaneous case of bronchogenic adenocarcinoma in an adult female albino guinea pig characterized by presence of proliferative neoplastic bronchial epithelial cells forming nests, and vacuolation in the liver parenchyma, is reported.

Introduction

The guinea pig *(Cavia porcellus)* is an important laboratory animal for studies of infectious diseases, nutritional diseases and neoplasms; they are also popular pet animals *(Flecknell, 2002)*. Spontaneous tumours are relatively uncommon in the guinea pig and they are mostly reported in older animals (*Kitchen et al., 1975*), especially in animals older than 3 years with an incidence rate as high as 30% *(Greenacre, 2004)*. Tumours of the respiratory system and liver were previously reported by Rogers & Blumenthal (1960) and Vannevel & Wilcock *(2005)*. Among the respiratory tumours, reports of naturally occurring bronchogenic adenocarcinoma in guinea pigs are very rare. In this communication, a case of bronchogenic adenocarcinoma in a female guinea pig which died naturally is reported.

Materials and Methods

An adult female albino guinea pig which was kept at the Central Animal Facility, AIIMS, New Delhi, was found dead. A thorough necropsy examination was conducted, gross findings were recorded and organs showing lesions were collected in 10 % neutral buffered formalin and processed for routine histopathology and stained with hematoxylin and eosin (H&E) and Periodic acid Schiff (PAS) stain as per Bancroft & Gamble *(2008)*.

Results

On post-mortem examination, numerous whitish nodules of variable size were diffusely distributed on the surface of the lungs exhibiting a disseminated pattern of spread of tumour. The liver was pale and enlarged and greasy on its cut surface. Histopathologically, the section of lung revealed, multifocal nodular growth with the involvement of bronchi and bronchioles. The tumour comprised large anaplastic cuboidal epithelial cells which had proliferated to form clusters or irregular acini or nests (Fig. 1). The tumour cells were hyperchromatic and arranged in single or multiple layers. Sloughed cells were found in the lumen of the tumour indicating the occurrence of metastasis. Adjacent alveolar spaces exhibited atelectasis and peripherally emphysema (Fig. 2). PAS staining showed no intracytoplasmic mucin secretion. The tumour showed the characteristic features of bronchogenic adenocarcinoma. In addition to the changes in lung, the liver showed moderate to severe microvesicular steatosis. The presence of vacuoles having peripheral nuclei, a characteristic of

lipid accumulation was seen in the perilobular areas of the liver along with dilated sinusoids (Fig. 3). Various apoptotic hepatocytes were also seen in the liver parenchyma.

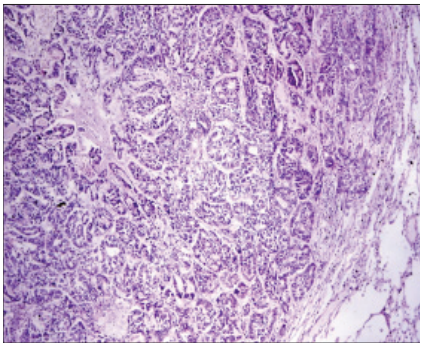

Figure 1. Bronchial epithelial cells proliferation forming clusters or irregular acini or nests. H&E X 40

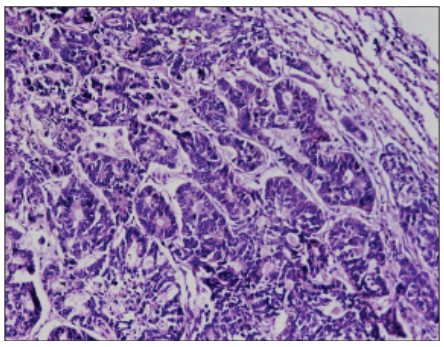

Figure 2. Bronchial epithelial cells proliferation forming clusters or irregular acini or nests which compressed the adjacent alveolar wall. H&E X 40

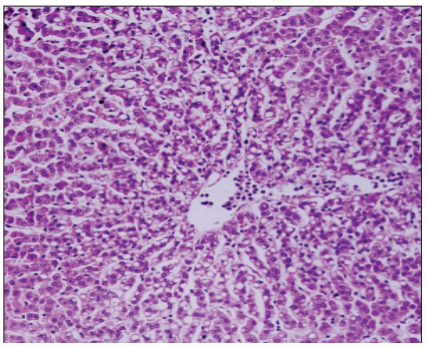

Figure 3. Liver showing moderate to severe microvesicular steatosis in the pericentral areas along with dilated sinusoids H&E X10.

Discussion

To date only small numbers of spontaneous tumours have been observed in guinea pigs which indicates that this species is probably one of the mammals least prone to tumours.

Different types of spontaneous tumours including bronchogenic papillary adenomas, carcinomas of the skin, trichofolliculomas, ovary teratomas and tumours of the mammary glandhave been reported in guinea pigs;the most common tumour of guinea pigs is bronchogenic papillary adenoma *(Rogers & Blumenthal, 1960; Kitchen et al., 1975; Greenacre, 2004)*. Goldberg *(1920)* has reported a case of bronchogenic adenocarcinoma in the guinea pig. Pulmonary adenomas and pulmonary adenocarcinomas were found in guinea pigs with concomitant interstitial pneumonia *(Fischer, 1956)*. Our findings of bronchogenic adenocarcinoma are in consonance with these reports.

The natural cancer resistance of the guinea pig has been shown by studying spontaneous tumours as well as experimental cancer induction with a number of carcinogenic agents *(Frank & Chasterman, 1962)*. Although it belongs to the rodent class, the guinea pig reacts differently to that of other rodents. Guinea pigs are considered to be a good model for better understanding of the growth and development of respiratory system tumours caused by carcinogenic substances or cigarette smoke *(Fiala et al., 2005)*. Fiala and his co-workers *(2005)* induced preneoplastic lung lesions, including bronchial hyperplasia, dysplasia and squamous metaplasia in guinea pigs.

Steatosis depends on an imbalance of fat intake and endogenous lipogenesis, and also on the capability of the liver to oxidize fat and deposit esterified fats and triglycerides intended for export in the form of lipoprotein *(Burt et al., 1988)*. In the guinea pig, high concentrations of free cholesterol are present in the liver (*Angelin et al., 1992*). They have moderate rates of hepatic cholesterol synthesis (*Reihner et al., 1990*; *Fernandez et al., 1990*) and catabolism (*Reihner et al., 1991*).

In our study, development of nodular growth in the lung parenchyma causes anorexia and respiratory insufficiency. Anorexia may cause mobilization of tissue fat reserve. Fatty changes in many animals have been associated with periods of anorexia. Reduction in carbohydrate intake and fat metabolism for energy leads to the development of fatty changes in the liver of guinea pigs (*Schaeffer & Donnelly, 1997*). Restricted feeding triggers non-alcoholic fatty liver disease (NAFLD) leading to perilobular steatosis of the liver *(Makovický et al., 2012)*. During starvation, liver cells mobilize tissue fat reserves *(Hernandez-Espinosa et al., 2006)* leading to fatty liver. Moreover, fatty liver is also observed in guinea pigs fed on high cholesterol and fat diet (*Ye et al., 2013*). It is presumed that fat accumulation is due to inability of the parenchyma cells to process excessive fat.

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