Benha Veterinary Medical Journal 39 (2020) 20-23

Official Journal Issued by Faculty of Veterinary Medicine

Benha Veterinary Medical Journal

Journal homepage: https://bvmj.journals.ekb.eg/

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Original Paper

Histopathological approach to interstitial lung diseases in camel in slaughterhouses, Aswan, Egypt

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ARTICLE INFO

Keywords

Camel

Histopathology

Interstitial pneumonia

Pneumonia

Received 12/09/2020
Accepted 22/10/2020
Available On-Line 20/01/2021

ABSTRACT

Interstitial lung disease includes a wide range of pulmonary diseases associated with varying clinical appearance, therapeutic response and prognosis which are a real challenge for diagnoses. The aim of this study is to described the different histopathological patterns of interstitial pneumonia in camels slaughtered in Draw abattoir in Aswan government to provide clinicians with information on the pathological features of interstitial pneumonia, which in most cases may allow for a specific diagnosis and a careful correlation with clinical findings. Also, a pattern-based histopathological approach to interstitial lung disease can help guide clinicians in choosing suitable treatment and provide camel owners with correct prognoses. A total of 42 lungs of camels were examined for histopathological changes. 30 cases of interstitial pneumonia were detected among the 42 examined camels' lungs. Interstitial pneumonia in camels has variable pathologic pictures. Fibrotic and cellular interstitial pneumonia are considered as the two main histologic subtypes. Nodular patterns were recognized as uncommon histologic changes. This study concluded that cellular, fibrotic and nodular forms are the common patterns of interstitial pneumonia in camels.

1. INTRODUCTION

Pulmonary lesions interferes with pulmonary functions, especially blood oxygenation and supply of oxygen (Anosa, 1983) that reduces animals productivity and causes massive economic loss to farmers (Zubair et al., 2004 and Kane et al., 2005).

Interstitial pneumonia with different histological appearances is the most common type of pneumonia in the inspected camels (Nourani and Rohani, 2009). It is a broad category of inflammatory disease that is characterized by alveolar septa being primarily involved in lesion pathogenesis.

Histologically, the alveolar septa interstitium is known as the space between the alveolar epithelial cells and the capillaries (Cheville, 1994; Norris et al., 2002) Various alterations in interstitial pneumonia injury and repair can lead to a common progression to interstitial fibrosis (Corcoran et al., 1999). The fibrosis process in the alveolar parenchyma frequently begins insidiously and has an unclear etiology (Crystal et al., 2002). The diagnosis and treatment of chronic interstitial fibrosis can be very difficult, because permanent loss of pulmonary function has often occurred before clinical symptoms are recognized (Norris et al., 2005).

2. MATERIAL AND METHODS

The present study was carried on the lungs of 42 adult camels slaughtered in the Aswan government's Draw abattoir between 2018 and 2019.

Small tissue specimens were collected from the lungs and immediately fixed in 10% neutral formalin buffered.

The specimens were routinely processed, and 4-5 µm tissue paraffin sections were prepared and stained by H andE stains (Bancroft and Stevans, 1993)

3. RESULTS

In this study, thirty cases of interstitial pneumonia were recorded among 42 examined lungs of adult dromedary camels (one-humped) slaughtered at the Draw abattoir in Aswan governorate between 2018 and 2019. The centralization of the lesions was mainly in the alveolar walls and its interstitium.

Based on the level of fibrosis, interstitial pneumonia was divided into three categories cellular, fibrotic and nodular. Without significant fibrosis, cellular interstitial pneumonia demonstrated prominent inflammation, cellular infiltration of the interstitium and alveolar space. This infiltrate was composed of lymphocytes and fibrin exudate (Fig. 1 and Fig. 2). Mixed cellular and fibrotic interstitial pneumonia showed both inflammation and fibrosis (Fig. 3A and B). Fibrotic interstitial pneumonia showed a severe fibrosis with/without inflammation (Fig. 4). In addition, fibroblastic foci defined where the normal resolution mechanism has failed (Fig. 5A and B, and Fig.6). The presence of discrete nodules in the lung parenchyma consists of lymphocytic cellular aggregation (Fig. 7A and B)

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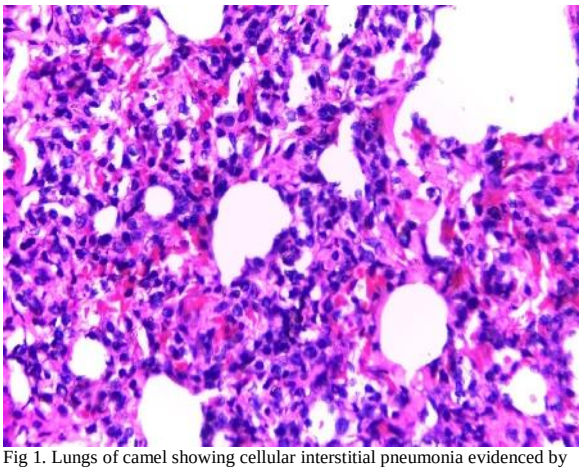

Fig 1. Lungs of camel showing cellular interstitial pneumonia evidenced by thickening of the alveolar septa due to chronic inflammatory cellular infiltration and deposition of fibrin. H &E stain X400.

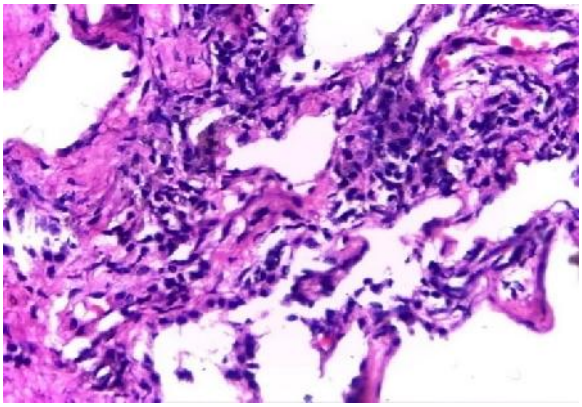

Fig 2. Lungs of camel showing cellular interstitial pneumonia represented by thickening of alveolar septa due to chronic inflammatory cellular infiltration and fibrosis. H & E X400.

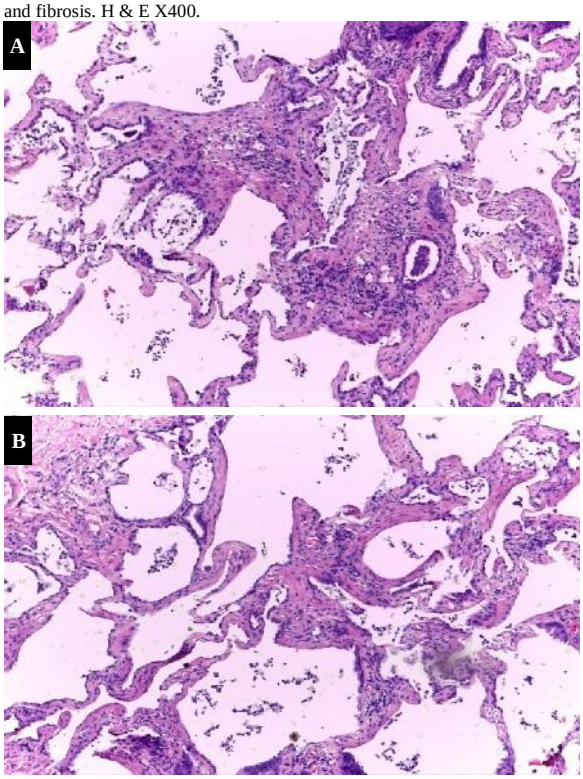

Fig 3.A and B. Lung of camel showing fibrotic pattern of interstitial pneumonia represented by severe interstitial fibrosis with chronic inflammatory cellular infiltration and cystic dilatation of the bronchiole that filled with cellular debris. H &E X100.

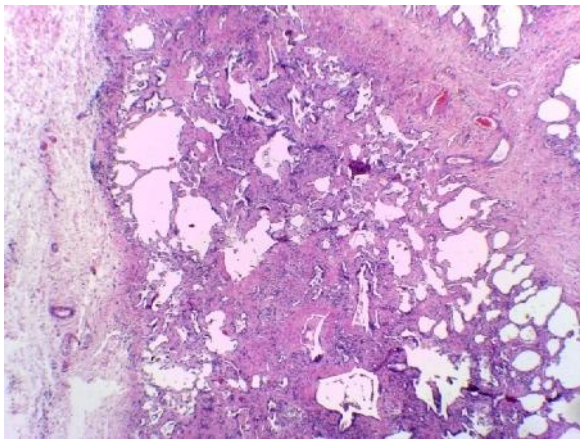

Fig 4. Lung of camel showing fibrotic pattern of interstitial pneumonia represented by severe pulmonary fibrosis of interstitial tissues and cystic dilatation of airways. H &E stain X100.

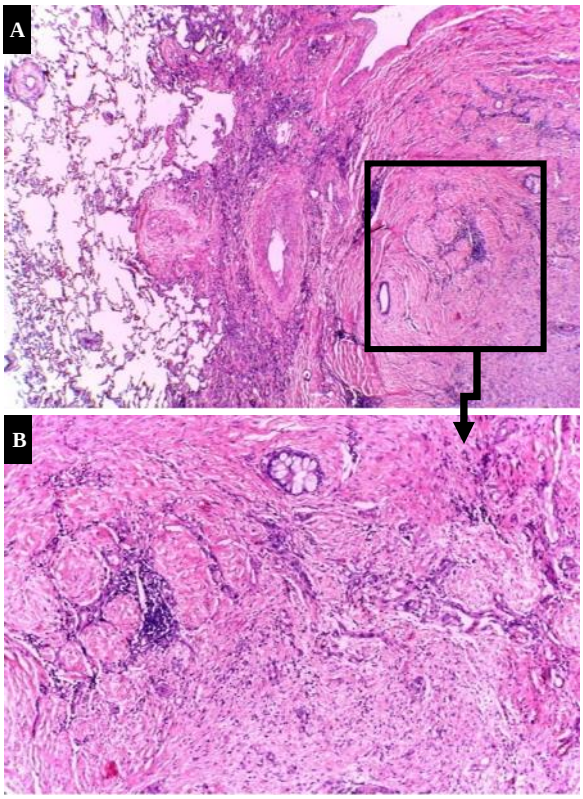

Fig 5A. Lung of camel showing fibrotic pattern of interstitial pneumonia evidenced by extensive destruction of pulmonary tissue which replaced by fibrosis. H &E stain X40. Fig 5B. High power of previous figure showing pulmonary fibrosis infiltrated with mononuclear cells and metaplasia of the lining epithelium of the bronchiole. H &E stain X100.

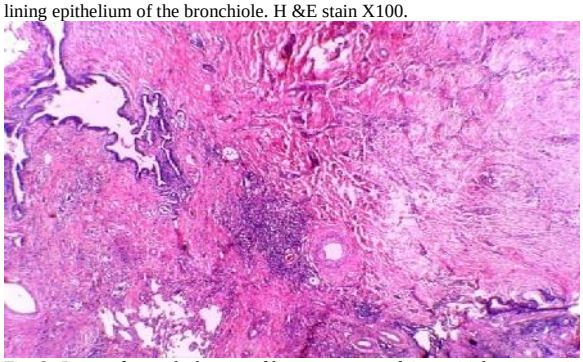

Fig 6. Lung of camel showing fibrotic pattern of interstitial pneumonia evidenced by extensive pulmonary fibrosis with focal mononuclear inflammatory cellular aggregation and cystic dilatation of the bronchiole. H &E stain X100.

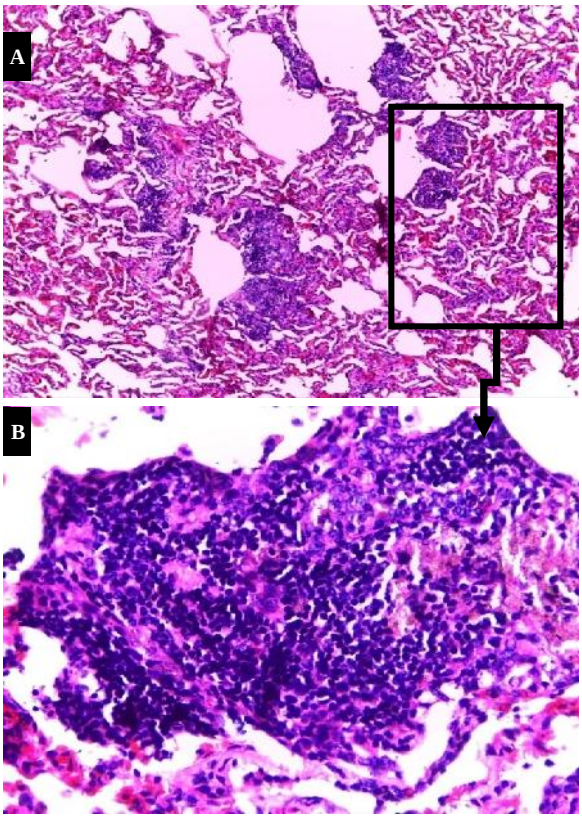

Fig 8A. Lung of camel showing nodular pattern of interstitial pneumonia represented by focal aggregations of mononuclear inflammatory cells in interstitial tissues. H &E stain X100. Fig 8B. Lung of camel showing nodular pattern of interstitial pneumonia represented by focal aggregations of mononuclear inflammatory mostly lymphocytes. H & E stain X400.

4. DISUCSSION

In the present study 30 cases of interstitial pneumonia were recorded among camels slaughtered at Aswan abattoirs. Interstitial pneumonia in camels has variable pathologic pattern (cellular, fibrotic and nodular). Nourani and Rohani, (2009) reported that interstitial pneumonia with different histologic appearances were classified broadly as acute and chronic interstitial pneumonia and considered as the most common form of pneumonia within the examined camels that may point to different etiological agents or developmental stages of pneumonia.

The occurrence of pneumonia in camels is typically low and when detected it indicates the nature of chronicity (Elfaki et al., 2002). The declared predisposing factors to bacterial and viral pneumonia lesions are climatic changes, stress factors, rearing systems, unhygienic conditions, sudden changes in feedstuff and a low-level herd health condition. Outbreaks of pneumonia are usually observed in camels throughout the change from the dry to the season of rains (Al-Tarazi, 2001). Atypical interstitial pneumonia reported in a number of domestic animal species (mainly cattle) in response to a diverse group of etiological agents. The best-known syndrome is that associated with pulmonary toxicity following the grazing of the pasture that has abrupt change from dry to lush, green condition. The possible pathogenesis is the metabolism of L-tryptophan in the pasture to 3- methyl indole in the rumen. This is, in the shift, changed to a pneumotoxic compound in the Clara cells of the lung, and it induces gross and microscopic characteristics lesions of atypical interstitial pneumonia (Dungworth, 1985). Moreover, agents under which certain circumstances may give rise to these changes of atypical interstitial pneumonia include acute viral pneumonia (Schiefer et al, 1974), Hypersensitivity reactions have frequently been speculated to cause atypical interstitial pneumonia in feedlot cattle (Schiefer et al., 1974; Jensen et al., 1976; Wikse, 1985). Also, many reports link dust to atypical interstitial pneumonia in feedlot cattle (Smith, 1998).

Idiopathic interstitial pneumonia is a category of heterogeneous diseases with uncertain etiology; characterized by pulmonary parenchymal distortion caused by varying degrees of inflammation and fibrosis. (Diken et al., 2019). Fibrosis, either in the form of irregular collagen deposition or the proliferation of collagen synthesizable fibroblasts (Visscher and Myers, 2006). In idiopathic interstitial pneumonia, pulmonary fibrosis is the major cause of high mortality and morbidity (Coline et al., 2015). The epithelial injury and fibroblast foci activation are early events that cause a series of changes eventually lead to the reorganization of pulmonary tissue compartments (Selman et al., 2001; Noble and Homer, 2004).

Sites of "acute" injury were characterized by denudation with breakdown of injured epithelial basement membranes, migration of fibroblasts / myofibroblasts to airspaces, and deposition of extracellular matrixes. (Katzenstein, 1985; Myers and Katzenstein, 1988; Morishima et al, 2001; Selman et al., 2001). Furthermore, intra-alveolar fibrosis, generated from organization of inflammatory exudates (Cordier et al., 1994; Cordier, 1999).

5. CONCULOSION

This research concluded that cellular, fibrotic and nodular forms are the main patterns of interstitial pneumonia in camels.

ACKNOWLEDGEMENT

Thanks are due to Dr. Wafaa Ahmad Aly (Ph.D. in Forensic Medicine and Veterinary Toxicology, and Manager of Veterinary Quarantine Department of Assiut) for arrangement and submission

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