AMYLOIDOSIS IN DROMEDARY CAMEL (ONE-HUMPED) IN ASWAN SLAUGHTER HOUSES, EGYPT

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ABSTRACT

The amyloidosis are a group of disorders in which soluble proteins aggregate and deposit extracellularly in tissue as insoluble fibrils, causing progressive organ dysfunction. A definitive diagnosis of amyloidosis is based on the histological presence of amyloid substance in tissue specimens. Under light microscope, amyloid substance appears as homogenous pink extracellular deposits in hematoxylin and eosin. On stained sections with Congo red, the extracellular deposits appear orange red in color. In this study, amyloidosis was diagnosed by histopathological examination under light microscope in the liver and kidney tissues from 30 adult dromedary camels (one-humped) collected from a total 50 adult dromedary camels (one-humped) that were presented for slaughter at Draw abattoir in Aswan Governorate between 2018 and 2019. Amyloid deposits in the kidney were located around Bowman’s capsule, mesangial areas and peritubular interstitial tissues. Amyloid was also seen in liver were located around blood vessels and along the sinusoids. Concurrent lesions of amyloid in liver include fatty changes, hydropic degeneration and necrosis. Also Concurrent lesions with amyloid in kidneys include proliferative glomerulonephritis and necrosis in tubules.

Keywords: Camel, Amyloid, Dromedary camel

INTRODUCTION

Amyloidosis constitutes a group of diseases in which proteins deposit extracellularly in tissues as insoluble fibrils (Dember, 2006). Amyloid deposits may be localized, organ-limited or generalized and can affect any tissue or organ type (Urban et al., 1993; Monzawa et al., 2002; Rocken and Shakespeare, 2002). Progressive organ involvement leads to organ malfunction and death usually resulting from renal and/or cardiac involvement. Liver and spleen are major sites of involvement (Monzawa et al., 2002; Park et al., 2003). Even when suspected clinically and radiologically, the diagnosis of amyloidosis mainly depends upon detection of amyloid deposits in tissue biopsy sections (Kim et al., 2003).

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MATERIAL AND METHODS

-Material:
The present study has been carried out on 30 adult Dromedary camels (one-humped) collected from a total 50 adult dromedary camels (one-humped) that presented for slaughter at Draw abattoir in Aswan Governorate between 2018 and 2019. Livers and kidneys were collected for processing.

-Methods:
The obtained tissue specimens were fixed in 10% neutral buffered formalin. The specimens routinely processed, paraffin embedded, sectioned at 4 - 5 micron thickness. The prepared slides were stained by H&E and Congo red stains (Bancroft and Stevans, 1993).

RESULTS

Amyloid deposits in renal and hepatic tissues in 30 adult Dromedary camels (one-humped). In H&E stained, amyloid appeared pale eosinophilic, homogeneous extracellular material. In Congo red – stained- sections, amyloid appeared orange red.

In kidneys: amyloidosis was diagnosed in kidneys by histopathological examination under light microscope. Amyloid deposited around Bowmans capsule (Figs. 1A, 1B), mesangial area (Figs. 2A, 2B) and at the peritubular interstitium tissues (Figs. 3A, 3B). Tubules adjacent to amyloid deposits were variably atrophied and were occasionally hydropic degeneration and necrosis (Fig. 3B). Amyloid deposits within glomeruli concurrent with expanding the mesangial area and proliferative glomerulonephritis (Fig.2A).

In Livers: amyloidosis was diagnosed in liver by histopathological examination under light microscope. Amyloids were located around blood vessels (Figs. 5A, 5B) and along the sinusoids (Figs. 4A, 4B). Fatty change (Figs. 4A, 4B, 5A, 5B), hydropic degeneration (Fig. 6A) and necrosis in liver cells (Fig. 6B) are concurrence with amyloid deposits.
Fig. 1A: Kidney showing amyloidosis. Notice the presence of homogenous pink extracellular material deposits around Bowmans’ capsule. H&E stain. 10x40
Fig. 1B: Kidney showing amyloidosis. Notice the presence of homogenous orange red extracellular material deposits around Bowmans’ capsule. Congo red stain. 10x40.
Fig. 2A: Kidney showing amyloidosis. Notice the presence of homogenous pink material deposits in the mesangium of glomerulus with proliferative glomerulonephritis. H&E stain. 10x40.
Fig. 2B: Kidney showing amyloidosis. Notice the presence of homogenous orange red material deposits in the mesangium of glomerulus. Congo red stain. 10x10.
Fig. 3A: Kidney showing amyloidosis. Notice the presence of homogenous pink material deposits within the renal interstitium. H&E stain. 10x40.
Fig. 3B: Kidney showing amyloidosis. Notice the presence of homogenous orange red material deposits within the renal interstitium. Tubules adjacent to amyloid deposits were variably atrophied. Congo red stain. 10x10.
Fig. 4A: Liver showing amyloidosis. Notice the presence of homogenous pink extracellular material deposits along the sinusoids. Fatty change with amyloid deposits. H&E stain. 10x10.
Fig. 4B: Liver showing amyloidosis. Notice the presence of orange red extracellular material deposits along the sinusoids with fatty change. Congo red stain. 10x10.
Fig. 5A: Liver showing amyloidosis. Notice the presence of homogenous pink extracellular material deposits around central vein with fatty change. H&E stain. 10x10.
Fig. 5B: Liver showing amyloidosis. Notice the presence of orange red extracellular deposits around central vein with fatty change. H&E stain. 10x10.
Fig. 6A: Liver showing amyloidosis. Notice the presence of homogenous pink extracellular material deposits along the sinusoids. Hydropic degeneration with amyloid deposits. H&E stain. 10x40.
Fig. 6B: Liver showing amyloidosis. Notice the presence of homogenous pink extracellular material deposits along the sinusoids. Necrosis with amyloid deposits. H&E stain. 10x40.
DISCUSSION

In this study amyloidosis was diagnosed in the liver and kidney tissues in 30 adult Dromedary camels (one-humped). Nakamura et al. (1995) also found cerebral senile plaques in an aged female camel (two-humped) of more than 20 years old.

Amyloid is a complex glycoprotein, comprising fibrillar and globular proteins, linked to polysaccharides, moreover, the amino acid content in amyloid in different from that in serum and tissue proteins, hyaline, and collagen (Shishkin, 2008). Amyloid protein and carbohydrate fractions are tightly bound to each other (Segev et al., 2012). The cells of the mononuclear phagocyte system usually inactivate amyloid; however, either decrease in their activity or amyloid hyperproduction result in amyloid deposition in the tissue (Naumenko, 2005). primary amyloid (Immunocytedyscrasia) is the most common form of amyloidos in humans but not in animals, on other hand secondary amyloid (reactive systemic amyloidosis) is the most common form of amyloidosis in animals, and the amyloid is deposited in kidney, liver, spleen and lymphnodes (Zachary and McGavin, 2012). Amyloidosis development depends on prolonged, abnormally high concentration of the serum amyloid A (SAA) protein in the blood serum, which is normally low (Real et al., 2014). In blood, SAA increased only during periods of active, systemic inflammation (Lozanski et al., 1996). Inflammatory conditions cause release of amyloidogenic cytokines (Colegrove et al., 2009). The mechanisms of amyloidogenesis are variable and include abnormal protein production, overproduction or decreased excretion of wild-type proteins and hereditary mutation (Szczepankiewicz et al., 2018).

Moreover, stress has been proposed as a factor in development of amyloidosis in some species (Cowan and Johnson, 1970; Germann et al., 1990; Papendick et al., 1997). The tissue tropism of amyloid deposition in affected camels was similar to that reported in other species (DiBartola and Benson, 1989). Kulikov et al., 2017 reported that the initial stages of amyloidosis in Shar-Pei dogs is characterized by glomerular amyloid deposits; then, the process spreads to the tunica propria of the tubules, whereas pronounced disease involves the interstitial connective tissue between tubules. This process lead to atrophy of the tubules and glomeruli, extending to their disintegration. Also, in case of hepatic amyloidosis, amyloid is deposited in parenchyma, along the sinusoids within the space of Disse, or in blood vessel walls. Hepatocytes are severely compressed by extensive accumulation of amyloid and they may atrophy or nearly disappear (Yong Moon Shin, 2011).

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dاء النشواني هو مجموعة من الاضطرابات التي تجمع فيها البروتينات القابلة للذوبان وتترسب خارج الخلية في الأنسجة كبروتينات غير القابلة للذوبان، مما يتسبب في اختلال وظيفي في الأعضاء. يعتمد التشخيص النهائي للداء النشواني على وجود النشواني في عينات الأنسجة في الفحص النسيجي المرضي. تحت المجهر الضوئي، تشكل الأميلويد المصبغة بالهيماتوكسيلين والأيوزين روابط خارج الخلية بلون متجانس وردي وبدع صباغتها بالأحمر الكونغولى. تكون الرواسب خارج الخلية برتقالية حمراء اللون. في هذه الدراسة تم تشخيص داء النشواني في أنسجة الكبد والكلى من 30 من الإبل البالغة (واحدة تحديدا) تم جمعها من مسلخ دراو في محافظة أسوان بين عامي 2018 و2019 عن طريق الفحص النسيجي تحت المجهر الضوئي. توجد روابط أميلويد في الكلية حول كبسولة بومان والغشاء المتوسط لكيبيبات الكلى والمساحات الينية حول الفيتيات البولية. كما شوهد أميلويد في الكبد حول الأوعية الدموية وعلى طول شدة الجبلي. ومن أهم التغييرات الهيستوبيولوجية التي ظهرت في أنسجة الكبد متزامنة مع الأميلويد كانت تجففات وفساد دهني في خلايا الكبد وكذلك بعض التكثير في الكبد و كانت الأع lashes المتزايدة مع أميلويد في الكلية التهاب كبيبات الكلى التكتئي وكذلك بعض التكثير للخلايا الطلائية المبطنة القنوات البولية.
