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SOME STUDIES ON RENAL AFFECTIONS IN CAMELS

(With One Table & 17 Figures)

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بعض الدراسات على اصابات الكلى فى الجمال

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لمعرفة نسبة ونوع اصابات الكلى فى الجمال اجريت هذه الدراسة على ٤٨٠ جمل سودانى وقد تم اخذ العينات من مجازر القاهرة. تم عمل الفحوصات الظاهرية والهستوباثولوجية والبكتريولوجية لعينات الكلى. كانت نسبة الاصابات الكلوية الكلية ٤,٣٧٥% امكن تقسيمها الى اربعة مجموعات وهى:- التهاب الكلوة الكبيبى المتزايد للنسيج الضام بنسبة ١,٤٥٨% والالتهاب الكلوة الكبيبى الداخلى المتزايد للشعيرات الدموية بنسبة ٠,٤١٦% والتهاب الكلوة الكوبيبى السدادى بنسبة ٠,٤١٦% والتهاب الكلوة الخلالى بنسبة ٢,٠٨٣%. كما تم عزل بعض الانواع من البكتريا من بعض العينات الكلوية المصابة.

SUMMARY

A survey of the prevalence and types of renal lesions was carried out on 480 Sudanese camels obtained from Cairo abattoirs. The macroscopic, histopathological and bacteriological examinations of camel's kidneys were performed. The incidence of total renal lesions among examined camels was 4.375 % and classified into four groups and included:- Mesangioproliferative glomerulonephritis (GN) (1.458%), Endocapillary proliferative GN (0.416%), Embolic GN (0.416%), Interstitial nephritis (2.083%). Different types of bacterial isolates were obtained from some cases.

Key words: *Kidney, camel, nephritis*

INTRODUCTION

Camel's meat is an important source of animal protein in Egypt. To face the increased requirements of meat in our country, large numbers of camels are imported from the Sudan for slaughtering in some abattoirs in Egypt.

The available literatures dealing with the renal pathology in camels are rather few except sporadic studies had been reported by Meszaros and Graf (1981), Vitotic (1987), Abdel-Baset (1991), Hatem (1991).

The present study is done to know the correlation between the pathologic changes of kidneys in imported camels with causative bacterial organisms.

MATERIAL & METHODS

Gross pathology:

A total of 480 camels' kidneys were collected at different seasons over 2 years from Cairo abattoirs and examined for the various pathological affections. Kidneys of slaughtered camels were examined for the existence of gross pathological changes.

Histopathology:

kidney samples obtained from slaughtered camels were fixed in 10 % neutral buffered formalin. Fixed tissues were dehydrated in a series of alcohols and processed for paraffin embedding technique. Sections were stained with haematoxyline and eosin (HE.) (Bancroft and Stevens, 1982).

Bacteriological examination:

Portions of the kidney were transported (on ice) to the laboratory for bacteriological examination. Culturing was carried on liquid media (nutrient broth & brain heart broth) and solid media (5% sheep blood and MacConkey agar media) according to Cowan and Steel (1965).

RESULTS

According to histopathological changes involving glomeruli, renal tubules or interstitium, the kidney lesions could be classified into four groups. The incidence of each group was presented in table(1).

Table 1: Incidence of renal affections in camels

Renal lesions	Affected camels	
	Number	Incidence
Mesangioproliferative GN	7	1.458
Endocapillary proliferative GN	2	0.416
Embolic GN	2	0.416
Interstitial N	10	2.083
Total	21	4.375

Mesangioproliferative GN:

In these cases, kidneys showed moderate swelling and congestion. Microscopically, the glomeruli were congested and in over 50 per cent of them showed moderate to marked mesangial cell proliferation and a corresponding increase in mesangial matrix leading to obliteration of many capillary lumina (Fig. 1, 2). In some glomeruli, mesangial proliferation was more prominent at the hilus (Fig. 3). Lymphocytes were seen in large numbers in most glomerular tufts (Fig.1, 2, 3). Mesangial cell proliferation together with leucocytic infiltration gave the impression of hypercellularity. In two cases mesangial cells was proliferated at the hilus of the glomeruli with marked increase in mesangial matrix. The parietal layer of Bowman's capsule showed thickening and hyalinization (Fig. 4). The renal tubules were dilated and contained casts. Mild peritubular lymphocytic infiltration was occasionally observed (Fig.1). No conspicuous changes were observed in the tubular epithelium. Such cases were negative for bacterial isolation.

Endocapillary GN:

In these cases, kidneys were swollen and soft in consistency. The cortex was congested and showed petechial haemorrhages. Microscopically the glomeruli were congested and appeared hyperceullar due to endothelial and mesangial cells proliferation together with leucocytic infiltration of glomerular capillaries(Fig. 5, 6,7). The renal tubular epithelium showed hyaline droplet and hydropic degeneration (Fig. 6, 8). The interstitial tissue showed congestion and haemorrhages (Fig. 8). No bacterial isolates were obtained from these cases.

Embolic GN:

In these cases, the kidneys were pale and firm in consistency. The cut surface was pale in color. Microscopically, in one case most glomeruli showed marked lobulation of glomerular tufts, congested glomerular capillaries, presence of bacterial colonies in the lumina of glomerular arterioles. The glomerular tufts were infiltrated with lymphocytes. The mesangium was greatly increased (Fig. 9). The renal tubular epithelium showed granular degeneration and the interstitial arterioles were congested and some of them contained bacterial colonies (Fig. 10). *Escherichia coli* was isolated from this case. In the another case (septic embolic GN), the glomerular tufts in most glomeruli were partially obliterated with numerous neutrophils and few numbers of lymphocytes. The tubular epithelial cells showed nuclear pyknosis and

karyorrhexis with intertubular and intracpithelial lymphocytic infiltration (Fig. 11). *Corynbacterium pyogens* was isolated from this case.

Interstitial nephritis:

In these cases, kidneys were slightly swollen. The cut surface was congested and revealed petechial haemorrhages. The capsule was easily removed. In some cases, kidneys revealed subcapsular pin-headed grayish foci. Microscopically the interstitial capillaries in both cortex and medulla were congested. Haemorrhages were frequently observed (Fig. 12). Perivascular, periglomerular and intertubular aggregations of lymphocytes and fibroblast cells were frequently observed (Fig. 13, 14,15). Mononuclear cell infiltrations were observed in peritubular interstitium or aggregated in focal areas replacing destroyed renal tubules (Fig.16). The renal tubular epithelium showed granular degeneration, desquamation or even coagulative necrosis (Fig. 12, 17). The glomeruli showed mild changes. The bacteriological examination revealed *protues mirabilis* from two cases and *E. coli* from one case.

DISCUSSION

In the present study it was of interest that mesangioproliferative GN was the highest incidence of renal lesions with negative bacterial isolation. It could be attributed to the fact that the mesangium is the most sensitive structure of the glomerulus. It is the first to react to irritation, with an increase in mesangial matrix and in the number of mesangial cells, and it is the last to return to normalcy after other glomerular pathologic changes have disappeared (Rotter, 1983). Mesangial cell proliferation is a basic and first response of the glomerulus to injury to phagocytize material filtered out of the vascular system at the renal corpusle (Rosen, 1983). Interlukin1(IL-1) released from stimulated mesangial cells by the deposition of IgA immune complexes, some cytokines produced by monocytes or neutrophils infiltrating to the glomeruli and/or some complement-associated mediators may account for the proliferation of epithelial and endothelial cells in the glomeruli (Matsumoto *et al.*, 1988). Almost any exogenous or endogenous antigen capable of remaining in the circulation long enough could trigger immune-complex formation (Brack, 1988). Immune complex glomerulonephritis is the common form among nephritic syndromes in human and animals (Yasushi *et al.*, 1992). In animals, Immune complex glomerulonephritis resulting from deposition in the kidneys of

circulating antigen-antibody complexes induced by many agents including parasites (Winter and Majid, 1986). In dogs it was revealed that in the course of glomerular IgA deposition, mesangial cells firstly proliferated and subsequently other types of cells, those are endothelial and epithelial cells, proliferated according to the amount of IgA deposits (Yasushi *et al.*, 1992).

In the present study, camels' kidneys with different forms of glomerulonephritis, with the exception of those with embolic GN that revealed *E. coli* and *C. pyogens*, revealed no evidence for bacterial infection. *E. coli* and *C. pyogens* undoubtedly were related to septicaemia or acute septic disease (Maxie, 1993). Embolic GN is the result of bacteraemia caused by several bacterial species in which bacteria lodge in glomeruli and to a lesser extent in interstitial capillaries and cause the formation of multiple foci of inflammation throughout the renal cortex (Anthony and Roger, 1995) In general, glomerulonephritis may cause by several etiological agents. It had been found in cattle infected with bovine virus diarrhea (Cutlip *et al*, 1980). Abdel-Baset (1991) reported that in camels, cutaneous lesions could lead to glomerulonephritis. While Hatem (1991) assumed that it may due to chronic illness as pneumonia, meteritis and liver abscesses. In humans, the etiologic agents may be endogenous or exogenous, antigens or factors (George and Kamal, 1998).

In the present study, interstitial nephritis was the most common lesion encountered. *protues mirabilis* and *E. coli* were isolated from some cases. Chauhan *et al.* (1976) isolated *E. coli* and *Klebsilla spp.* from kidneys of calves suffered from white spotted kidneys. Brack (1988) found that cortical tubulointerstitial nephritis was an integral part of IgM mesangial nephropathy of callithrids Moreover Abdel-Baset (1991) and Hatem (1991) isolated *protues mirabilis* and *E. coli* from some camels' kidneys with interstitial nephritis but they found that it might also attribute to cutaneous lesions as parasitic and bacterial infections or due to toxic material. Anthony and Roger (1995) stated that the interstitial nephritis is the result of bacterial and viral septicaemias, whereby these infectious agents infect the kidney tubules and incite an inflammatory response in the interstitium. The later authors added that, *E. coli* septicaemia in calves can result in interstitial nephritis in which, localization of bacteria occurs in the interstitial capillaries, migrate throughout vascular endothelium, persist in the interstitial spaces and immigrate via the lateral intercellular junctions to reach tubular lumina.

The tubular epithelial cells undergo degeneration and necrosis due to either toxic effects of the bacterium or accompanying interstitial inflammatory reaction. Immune response is the another well-documented mechanism for the production of interstitial nephritis. Depostion of immune complex in or interactions between antibasement membrane antibodies and tubular basement membrane can initiate immune mediated tubulointerstitial nephritis in human beings. At present, immune- mediated mechanisms are questionable as causes of interstitial nephritis in domestic animals.

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LEGENDS OF FIGURES

Fig. (1): Kidney micrograph showing mesangial cell proliferation, greatly increased mesangial matrix, glomerular tufts are infiltrated with lymphocytes and mild peritubular lymphocytic infiltration. HE. X400.

Fig. (2): Kidney micrograph of a camel showing congestion and hyperceullarity of the glomerular tufts due to mesangial cell proliferation and lymphocytic infiltration. HE. X400.

Fig. (3): Kidney micrograph showing congested glomerulus, has a large numbers of mesangial cells and mesangial proliferation at the glomerular hilus. The renal tubules are dilated and contain casts. HE. X400.

Fig. (4): Kidney micrograph of a camel showing increases of the glomerular mesangial matrix, proliferation of mesangial cells and visceral layer of the Bowman's capsule at the hilus of the glomerulus with thickening and hyalinization of the parietal epithelial layer. HE. X400.

Fig. (5): A micrograph showing glomerular hypercellularity, hydropic degeneration of the renal tubular epithelium, presence of hyaline casts in collecting ducts and mononuclear inflammatory cell infiltration in interstitial tissue. HE. X250.

Fig. (6): Higher magnification from the previous figure showing hypercellularity of the glomerulus due to endothelial and mesangial cells proliferation with leucocytic infiltration. The renal tubular epithelium showing hydropic degeneration. HE. X400.

Fig. (7): Kidney micrograph of a camel showing hypercellularity of the glomerulus due to endothelial and mesangial cells proliferation and lymphocytic infiltration. The proximal tubules contain cell debris. HE. X250.

Fig. (8): Kidney micrograph showing hyaline droplet degeneration of the tubular epithelium, peritubular mononuclear cell infiltration, congestion and haemorrhages in interstitial tissue. HE. X400.

Fig. (9): Kidney micrograph showing marked lobulation of glomerular tufts, congested glomerular capillaries, the glomerular tufts infiltrated with lymphocytes with presence of bacterial colonies in the lumina of two adjacent arterioles and greatly increased mesangium. The interstitial capillaries are congested and the renal tubular epithelium showing granular degeneration. HE. X400.

Fig. (10): Kidney micrograph showing granular degeneration of the tubular epithelium and presence of bacterial colonies in interstitial arterioles. HE. X400.

Fig. (11): Kidney micrograph of a camel showing that the glomerular capillaries in a portion of the glomerulus partially obliterated with numerous neutrophils and lymphocytes. The tubular epithelial cells showing nuclear pyknosis and karyorrhexis with intertubular and intraepithelial lymphocytic infiltration. HE. X400.

Fig. (12): Kidney micrograph showing coagulative necrosis of the renal tubular epithelium with interstitial congestion and haemorrhages. HE. X250.

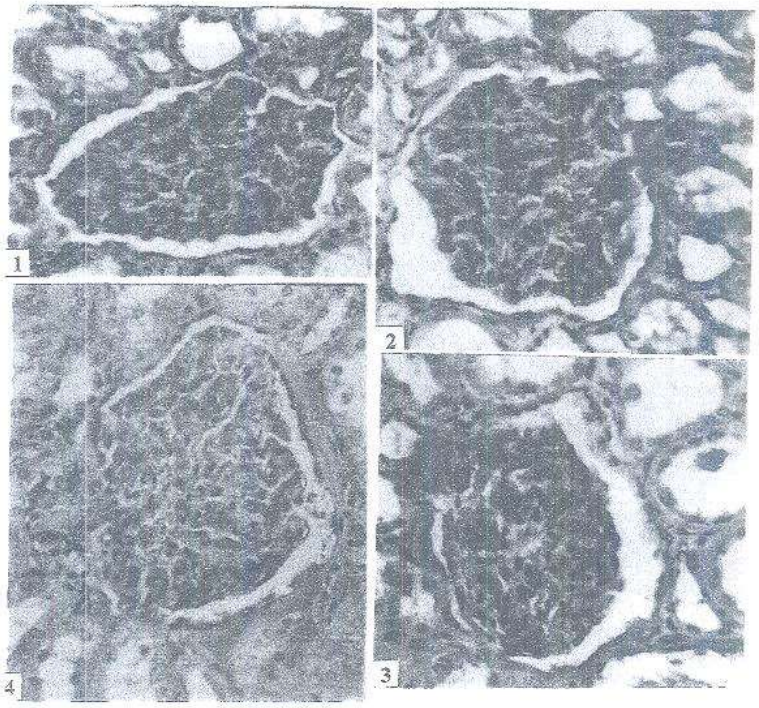
Fig. (13): Kidney micrograph showing perivascular and intertubular lymphocytic infiltration. HE. X400

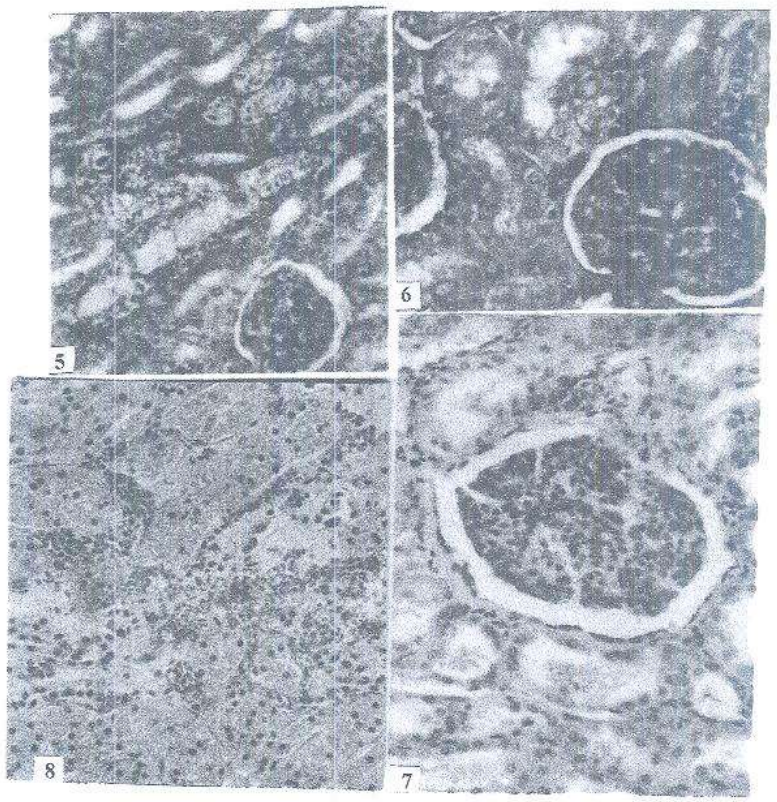
Fig. (14): Kidney micrograph showing periglomerular and focal interstitial mononuclear cell infiltrates. HE. X400

Fig. (15): Kidney micrograph showing intertubular lymphoid cell infiltration and fibroblastic proliferation. HE. X400.

Fig. (16): Kidney micrograph showing mononuclear cell infiltration in peritubular interstitium and in a focal area replacing the destroyed renal tubules. HE. X250.

Fig. (17): Kidney micrograph showing granular degeneration of the renal tubular epithelium with desquamation of tubular epithelium of individual renal tubule. HE. X1000

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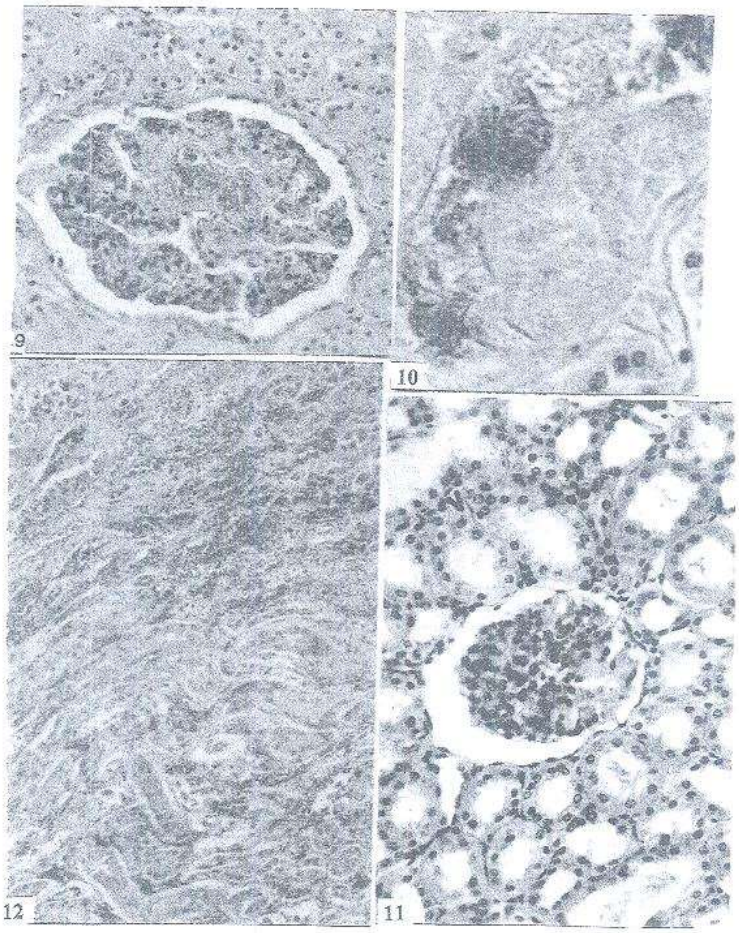

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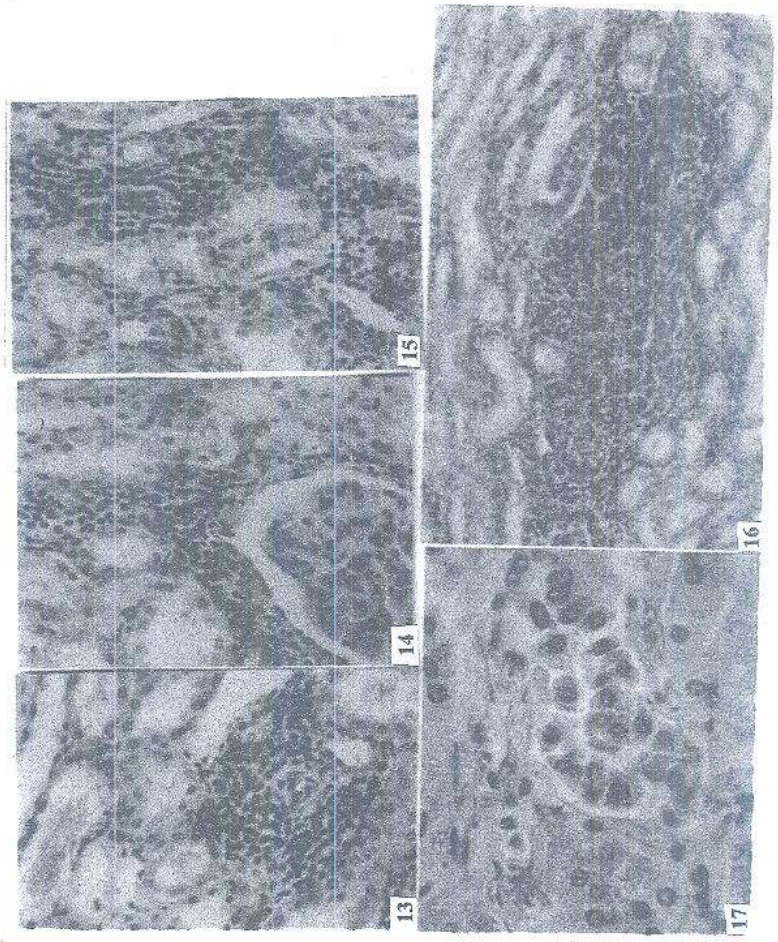
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