

Histopathological Study of Trypanosomiasis in Camels of Al-Diwaniyah Province

A. H.A. Al-shabbani
Coll. of Vete. Med./Unive.
of Al-Qadisiya

Kh. A. Dawood
Coll. of Med./Unive. of
Al-Qadisiya

Gh. A.Jassem
Coll. of Vete. Med./Unive.
of Al-Qadisiya

Abstract

The present study includes knowledge the histopathological changed of trypanosomiasis that is caused by *Trypanosoma evansi* in camels. Sample including: kidney, liver and spleen. The histological manifestation of the liver in the acute stage of trypanosomiasis shows coagulative necrosis, hemosiderin infiltration and billrubin. In the chronic stage shows infiltration of fibrous tissue and connective tissue between the lobules of the liver and decrease number of hepatic cells with hemorrhage, red blood cells among the hepatic cells. Lesions of spleen in the acute stage reveals hemorrhage and active lymphoid follicles with clumping of trypanosoma in spleen surrounded by inflammatory cells, in chronic stage fibrosis between the cells with increase thickness of trabeculae. In chronic stage of trypanosomiasis in kidney show glumerulonephritic due to precipitation of immune complexes as homogenous and calcified glumerulus due to precipitation calcium with desquamation of epithelial cells which are lined the urinary tubules and full the lumen of tubules, glomerulus suffer from shrink of the tuft, increase thickness of Bowman's capsule due to infiltration of fibrous tissue.

Key Word: Histopathological, Trypanosomiasis, Camels

Introduction

Trypanosoma evansi infection is one of the major problems that affect the productivity of camels, which leads to either lower in the working efficiency or even may result in death of the animal (1). Camel trypanosomiasis, also known as surra, is caused by *Trypanosoma evansi*, is an important cause of economic losses in camel rearing areas, causing morbidity of up to 30% and mortality of around 3% (2). There is a considerable degree of variation in the severity of the pathological effects of *T. evansi*, this variation is ascribed to the difference in virulence of individual strains, susceptibility of the host, local epizootiological conditions and stress factors (3). *T. evansi* is a blood protozoa but visceral forms have also been reported in heart, optic lobes, cerebrum, liver, kidney and lungs, course of infection and degree of pathogenicity depend upon the species of host and virulence of *T. evansi* strain, the pathology associated with trypanosomiasis

includes lymphadenopathy, splenomegaly, hepatomegaly, anemia, testicular enlargement, hemosiderosis and consolidation of the anterior lung lobes (4). These changes are not considered pathogenomic for the disease, except if trypanosome are present (5). Other lesion can include glumerulonephritis, calcification of glomerulus, renal tubular necrosis, non-suppurative meningo-encephalomyelitis, focal poliomalacia, keratitis, ophthalmitis, orchitis, interstitial pneumonia and bone marrow atrophy, splenic and lymph node hypertrophy occur during the acute phase but the lymphoid tissues are usually exhausted and fibrotic in the chronic stage (6). The increases in size of the spleen, lymph nodes and liver were associated with congestion, increases in cell density related to increased immunological reactions in the spleen and lymph nodes as well as increase in numbers, size and activity of the phagocytic cells in these organs (7).

Materials and Methods

Methods

Animals

A total number of 200 organs samples were collected from the camels (*Camelus dromedarius*) which were slaughtered in Al-Diwania Abattoir .

Samples Collection

Organs Collection

Specimens from liver ,kidney , spleen and kept in plastic container contain 10% formaline .

Histopathological Procedure

Samples were taken from the kidney ,liver and spleen in size 1-2 cm³ by using sharp blade and scalpel then used . The samples were kept in 10% formalin approximately one week at room temperature; after that the tissues were processed by routine processing methods (8):

1. Dehydration. 2.Clearing. 3.Infiltration.
- 4.Embedding. 5.Sectioning. 6.Rehydration.
- 7.Staining. 8. Drying, clearing and labeling. 9. Mounting.

Dehydration by use, series spiraling of alcohol beginning with 50%, 60%, 70%, 80%, 90% and 100% for 2 hours into all concentration. Clearing is by using Xylen in two stages for

1/2 hour to each stage. Infiltration is by putting the tissue specimens in paraffin wax on (56-58°C) the temperature of the paraffin wax baths for two stages about two hours to each stage. Embedding the specimens within paraffin wax and let for 24 hours. Sectioning the specimens at 5-7 µm by using Rotary microtome. A thin smear from the Mayer's egg albumin to fix the sections strip on glass slides. Rehydration by alcohol abdicable series beginning with 100%, 90%, 80%, 70%, 60% and 50%. The histological slides were dried in a thermal oven at (40 °C) for 24 hours then the section strip was stained. Finally, staining the samples by Hematoxylin and Eosin stains for demonstrating the general histological components of the tissue. Mounting is the process of putting the samples on a clean glass slide then adding a amount of Canada balsam and covering it. The stains: Alum Hematoxylin and Eosin prepared as follows :

1. Hematoxylin crystals 5 gm.
2. Alcohol 100 (50 ml).
3. Ammonium or potassium alum 100 gm.
4. Distilled water 1000 ml.
5. Mercuric oxide (red) 2.5 gm.

Results

responses and noticed these clumping were under magnification was surrounded by inflammatory cells fig.(5).

Kidney

The histopathological section from kidney In the chronic stage of trypanosomiasis show glomerulus suffer from glomerulonephritis, precipitation homogenous acidic material (red color) in lumen of Bowman's capsule, hemorrhage inside and outside the glomerulus, epithelial cells are full in the center of urinary tubule fig.(6). In the chronic stage the glomerulonephritic showed, precipitation of immune complexes as homogenous material full all the space of Bowman's capsule and calcified glomerulus also was noticed due to precipitation calcium fig.(7). glomerulus with very thick Bowman's capsule and shrinkage of the tuft of capillaries fig.(8).

Microscopic Pathological Changes

Liver

The histological section of the liver in the acute stage of trypanosomiasis shows coagulative necrosis, hemosiderin infiltration and bilirubin, fatty degeneration in hepatic cells around the central vein fig (1). In the chronic stage shows infiltration of fibrous tissue and connective tissue between the lobules of the liver (liver cirrhosis) and decrease number of hepatic cells with hemorrhage, red blood cells among the hepatic cells fig (2).

Spleen

The spleen in the acute stage of trypanosomiasis shows hemorrhagic and active lymphoid follicles fig. (3). Clumping of trypanosoma in spleen surrounded by inflammatory cells fig.(4), clumping of trypanosome in spleen occurred due to immune

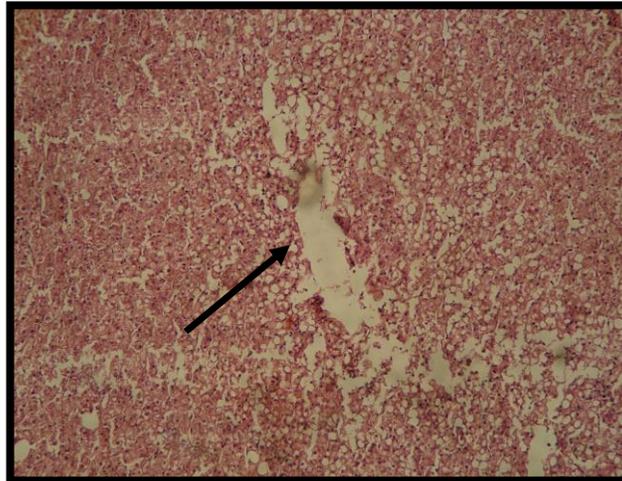


Fig. (1) Liver infected with acute stage of trypanosomiasis shows fatty degeneration in hepatic cells around the central vein. H&E. stain 10X.

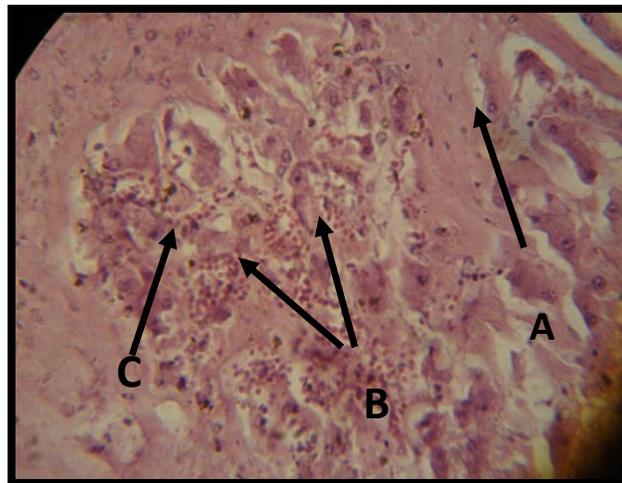


Fig. (2) Liver in chronic stage of trypanosomiasis shows infiltration of fibrous tissue between the lobules of the liver (arrow A) with hemorrhage, red blood cells among the hepatic cells (arrow B) H&E. stain 40X.

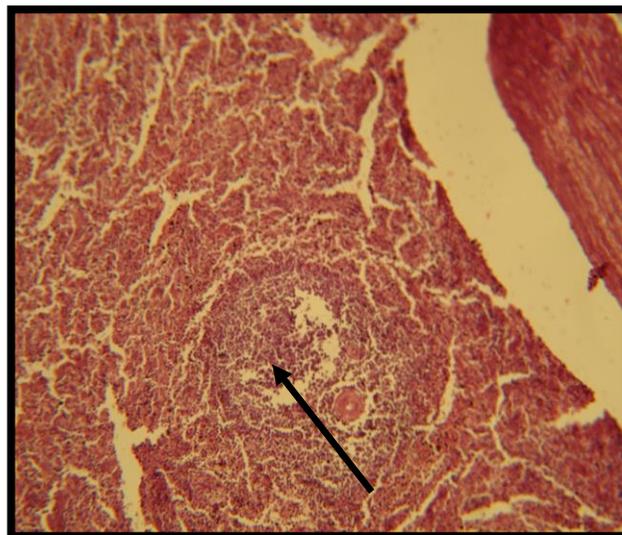


Fig. (3) Spleen in acute stage of trypanosomiasis shows active lymphoid follicles. H& E. stain 10X

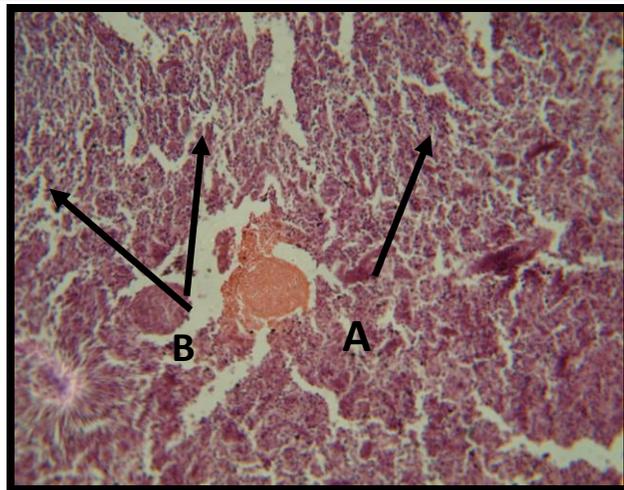


Fig. (4) Spleen infected with acute case shows clumping of *Trypanosome evansi* (arrow A) surrounded by inflammatory cells (arrow B). H& E. stain 10 X

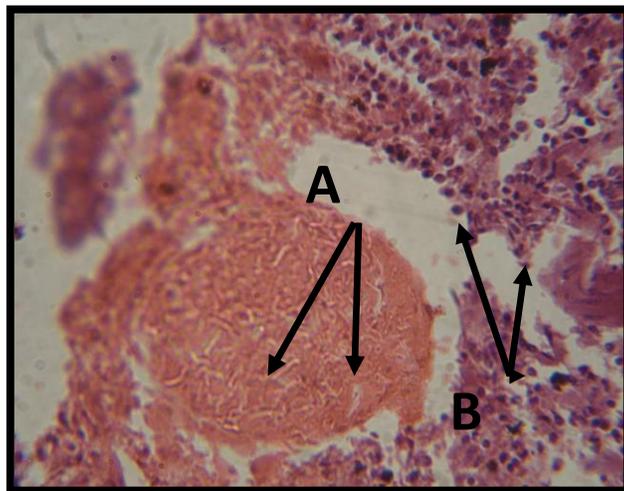


Fig. (5) Spleen infected with acute case shows clumping of trypanosome (arrows A) surrounded by inflammatory cells (arrow B). H& E. stain 40X.

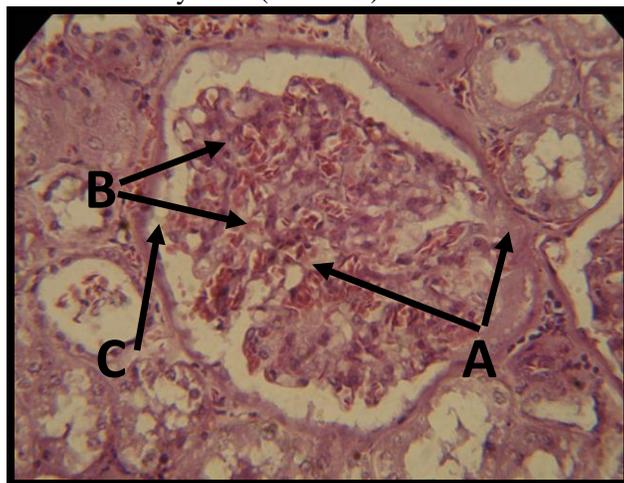


Fig. (6) Kidney infected with chronic stage of trypanosomiasis shows glomerulonephritis ,precipitation of homogenous acidic material (arrow A), hemorrhage inside & outside the glomerulus (arrow B), epithelial cells in center of urinary tubules (C). H&E. stain 40X.

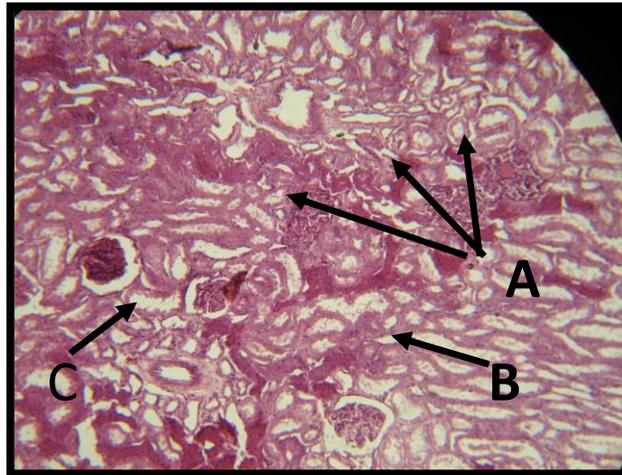


Fig. (7) Kidneyinfected with chronic stage of trypanosomiasis shows three of glomeruloi suffer from glomerulonephritis (A), intact glomerulus (B) & calcified glomerulus (C) . H& E. stain 10X.

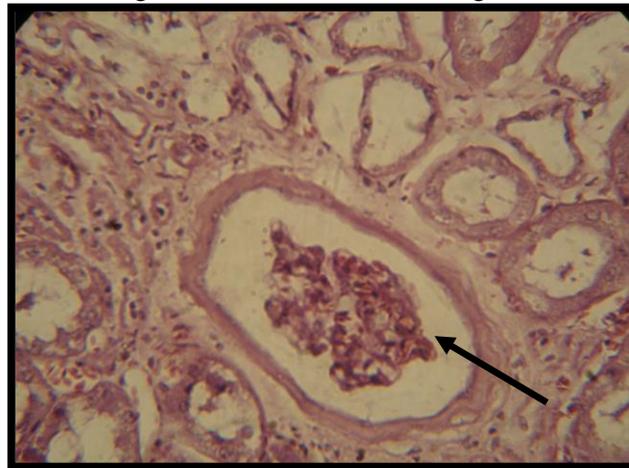


Fig. (8) Kidney infected with chronic stage of trypanosomiasis shows glomerulus with very thick Boman's capsule & shrinkage of the tuft of capillaries . H&E. stain 40 X.

Discussion

Histopathological study reveals many microscopical changes of liver in acute stage of trypanosomiasis which included necrosis, hemosiderin, fatty degeneration in hepatic cells around the central vein. In the chronic stage show infiltration of fibrous tissue and connective tissue between the lobules of the liver, with hemorrhage, present of red blood cells among the hepatic cells. In other studies In rabbits infected with *T.evansi* Dawood (9) recorded hepatomegaly and hemosidrosis in acute stage, fibrosis, thiking of liver capsule and necrosis in chronic stage. Ogunbanwo *et al* .(10) in rat infected with *T. brucei brucei*

showed necrosis of hepatocytes around the central vein, swollen hepatocytes (cloudy swelling as a result of mild accumulation of water) surrounding the necrotic region, inflammatory cells around the central vein. In rabbits infected with *T.evansi* Rodrigues *et al.* (11) showed the liver there was moderate lymphoplasmacytic periportal hepatitis, Kupffer cell hypertrophy, and hemosiderosis. The changes were destructive and irreversible, fatty change, binucleated cells and calcified cells seen by Mbaya *et al* .(12) in rat infected by *T. brucei*. Abd El-Baky and Salem (13) recorded fatty degeneration in which the

hepatocytes contain round, sharp and delineated vacuoles in camels. Hepatic changes may occur due to trypanosomes infection or their products and presence haemosiderin due to lysis of blood cells. Lesions of spleen in acute stage of trypanosomiasis reveals hemorrhagic and active white follicle and clumping of trypanosoma in spleen surrounded by inflammatory cells. In ovine Omotainse and Anosa (14) observed hyperplasia of the red pulp, congestion of the sinuses, enlargement of the lymphoid nodules, increased erythrophagocytosis, haemosiderosis and proliferation of plasma cells were the lesions seen in the spleen in acute *T. congolense*, *T. vivax* and *T. brucei* infections. The lesions were generally more pronounced in *T. brucei* infections than in *T. congolense* and *T. vivax* infections. Abd El-Baky and Salem (13) showed the presence of histocyte, reactive lymphocyte and severe aggregation of histocytes with the appearance of *T. evansi* parasites in spleen of rat. Garba (15) recorded massive splenic hyperplasia with expanded lymphoid follicles and moderate erythrophagocytosis occurred in the spleen of mice infected with the *T. congolense*. The most important lesion observed in the kidney in chronic stage of trypanosomiasis shows glomerulus suffer from glomerulonephritis,

precipitation acidic in lumen of Bowman's capsule, epithelial cells are full in the center of urinary tubule. In advance chronicity stage of trypanosomiasis was showed glomerulonephritic due to precipitation of immune complexes as homogenous and calcified glomerulus due to precipitation calcium with desquamation of epithelial cells which are lined urinary tubules and full in the lumen of tubules, glomerulus suffer from shrink of the tuft, increase thickness of Bowman's capsule due to infiltration of fibrous tissue. Omotainse and Anosa (14) showed in acute *T. congolense* infection diffuse congestion of the intertubular blood vessels and in chronic *T. vivax* infection accumulation of proteinous material in the Bowman's capsule of the glomerulus. Abd El-Baky and Salem (13) showed the presence of renal casts due to the renal tubular damage at the 7th day of infected rat with *T. evansi*. Garba (15) observed mild to moderate focal and diffuse infiltrations of lymphocytes, plasma cells and macrophages occurred in the interstitium of the renal medulla, cortex, and in the glomerular areas of mice infected with *T. congolense*, also showed immunoperoxidase staining revealed trypanosomes in the renal capillaries.

References

1. Luckins, A.G. (1998). Epidemiology of Surra; Unanswered questions. J. Protozool. Res. 8, 177-181 Lumsden W.H.R., Kimber C.D., Strange M. 1977. *Trypanosoma brucei*; detection of low parasitaemias in mice by a Miniature Anion-Exchanger-Centrifugation Technique. Trans.R. Soc. Trop. Med. Hyg. 71: 421-424.
2. Enwezor, F. N. C. & Sackey, A. K. B. (2005). Camel trypanosomiasis - a review. Vet. arhiv 75, 439-452.
3. Hoare, C.A. (1972). The trypanosomes of mammals. A zoological monograph. Blackwell Scientific Publications, pp 555-593.
4. Biswas, D.; Choudhury, A. & Misra, K.K. (2001). Histopathology of *Trypanosoma evansi* infection in bandicoot rat. I. Visceral organs. *Exp Parasitol.* 99(3):148-59.1.
5. Dargantes, A.P.; Campbell, R. S. ; Reid, S.A. & Copeman, D.B. (2005b). Experimental *Trypanosoma evansi* infection in the goat. II. Pathology. *Journal of Comparative Pathology* 133(4): 267-276.
6. Schmidt, T. G. and Roberts, L. (2005). *Foundation of parasitology.* 7th

- edition. The McGraw-Hill Companies.
7. Taylor, M.A.; Coop, R.L. and Wall, R.L. (2007). Veterinary parasitology. 3th edition. Blackwell publishing, Australia.
8. Luna, L. G. (1968). Manual of histological staining methods of the armed forces institute of pathology. 3rd ed., McGRAW – HILL BOOK Co, P3.
9. Dawood, K. A. (1996). The immunopathological effect of animal infected with *Trypanosoma evansi*. Ph. D. Thesis College of Veterinary Medicine , University of Baghdad .
10. Ogunbanwo , J. A.; Agbonlahor , D.E.; A. Adamu , A.; Dalyop, P. Stephen O. Elesha , S. O.& Fagbenro-Beyioku , A.F. (2001). Effects of anti-protozoal drugs and histopathological studies on trypanosome species. FEMS Immunology and Medical Microbiology 30 : 73-83.
11. Rodrigues, A.; Figuera, R.A.; Souza, T.M.; Schild, A.L.& Barros, C.S.L. (2009). Neuropathology of naturally occurring *Trypanosoma evansi* infection of horses. Veterinary Pathology 46: 251-258.
12. Mbaya, A. W.; Kumshe , H. A.; Geidam , A.Y. and Wiam, I. (2011). Effect of Diminazene Aceturate (Berenil®) on the Pathology of Experimental *Trypanosoma Brucei* Infection In African Giant Rats (*cricketomys Gambianus*). Nigerian veterinary Journal 32(3): 192 – 198.
13. Abd EL-Baky, A. A. and Salem, S. I. (2011). Clinicopathological and cytological studies on naturally infected camels and experimentally infected rats with *Trypanosoma evansi*. World Applied Sciences Journal 14 (1): 42-50.
14. Omotainse, S. O. and Anosa, V. O. (2009). Comparative histopathology of the lymph nodes, spleen, liver and kidney in experimental ovine trypanosomosis. Onderstepoort Journal of Veterinary Research 76:377–383 .
15. Garba, M. (2011). Pathogenicity, Histopathology and Immunoperoxidase Staining of Trypanosomes in *Trypanosoma congolense* Infected Balb/C Mice. Nigerian Veterinary Journal 32(2): 102 – 111.

دراسة التغيرات النسيجية للمثقبات في جمال محافظة الديوانية

غيداء عباس جاسم
كلية الطب البيطري / جامعة القادسية

خيري عبد الله داود
كلية الطب / جامعة القادسية

أمال حسن عبد الشباني
كلية الطب البيطري / جامعة القادسية

الخلاصة

تضمنت هذه الدراسة معرفة التغيرات النسيجية التي تسببها مثقبية أفانسي في الجمال أظهرت الإصابة بتغيرات مرضية في الأعضاء في الحالات الحادة والمزمنة إذ أن نسيج الكبد في حالة الإصابة الحادة تنكس دهني في الخلايا الكبدية حول الوريد المركزي، أما في الإصابة المزمنة يلاحظ تشمع وتليف الكبد إذ تمت ملاحظة ارتشاح الأنسجة الليفية والضمادة بين الفصوص الكبدية بكثافة عالية وتناقص أعداد الخلايا الكبدية وانحسارها إلى تجمعات صغيرة فقط كما يلاحظ النزف وارتشاح كريات الدم الحمراء حول الخلايا الكبدية، أما التغيرات النسيجية في الطحال في الإصابة الحادة أظهرت تجمع لطيفلي التريبانوسوما محاطاً بالخلايا الالتهابية وفي الإصابة المزمنة يلاحظ التليف بين الخلايا وزيادة عدد الخلايا اللمفاوية والبلعمية كما يلاحظ وجود مجال فارغ في مركز الجريبات اللمفية lymphoid follicles بسبب هجرة تلك الخلايا إلى اللمف، الدم والأعضاء المصابة الأخرى. أما التغيرات الكلوية في الإصابة الحادة يلاحظ التهاب الكبيبات الكلوية، كما يلاحظ النزف داخل وخارج الكبيبات الكلوية، كما يلاحظ امتلاء النبيبات الكلوية بمادة متجانسة عديمة التراكيب الخلوية مما يدل على ترسب المعقدات المناعية في الحالات المزمنة كما أظهرت الإصابة المزمنة تكلس و التهاب الكبيبات الكلوية مع توسف الخلايا الطلائية المبطنة للنبيبات الكلوية وسقوطها في تجويف النبيبات مع زيادة سمك محفظة بومان لأرتشاح الألياف والنسيج الضام حولها.

الكلمات المفتاحية: النسيجية، التريبانوسومياسز، الجمال