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RESEARCH ARTICLE

Bacillary dysenteriae : (1) Study on experimental pathology of Shigella dysenteriae type 1 in white mice following intraperitoneal infection.

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Abstract

Shigellosis is a highly infectious disease affecting man and the different animal species. For the importance of this disease, this study aimed at to describe the pathological lesions associated with *Shigella dysenteriae* type1 following experimental infection. For this reason a local strain of *Shigella dysenteriae* type1 were obtained from public health laboratory. The strain were reidentified using biochemical tests, API-20 test, slide agglutination test and Sereny test in Guinea pigs (induce a Keratoconjuctivitis).The LD50 for this strain was 8×10^3 CFU in 0.25ml of bacterial suspension were injected 1/P in 45 mice 3 mice sacrificed at 2 days intervals, then different pathological lesions were recorded including a microabscesses in liver, spleen which transform into granulomatous lesions later on. In addition the neutrophils infiltration, mononuclear cells infiltration were seen in the lungs, kidney, brain, periton, heart, intestine together with lymphoid hyperplasia in the lymph node and lymphoid tissue of spleen, peyer's patches of intestine and peribronchial lymphoid tissue of the lungs.

INTRODUCTION

Shigellosis is an infectious disease affecting children at different ages, associated with a dysenteric diarrhea admixed with mucous and blood and abdominal pain and fever. It distributed all over the world accompanied by high mortalities in children [(1)] especially in poor and crowded countries [(2)].The infection with *Shigella dysenteriae* type 1 considered to be highly fatal associated with the different inflammatory lesions and microabscesses occurred in the intestinal mucosa induced by this microbe a highly pathogenic Shigella species in addition to hemorrhage and pseudodiphtheric membrane on intestinal mucosal surface [(3)]. Monkeys considered to be the natural host for shigella in addition to human being, also Guinea pigs and other laboratory animals might be affected by this microbial agent [(4)]. from then importance of this fatal disease this study aimed to study the different Pathological lesion associated with experimental infection of white mice with *shigella dysenteriae* type 1.

Materials and Methods

A local strain of *shigella dysenteriae* type 1 obtained from public health laboratory. The strain were re-identified to be sure *shigella dysenteriae* type 1 [(5)]using cultural, biochemical test, API - 20 tests slide agglutination test and Sereny test in Guinea pigs causing Keratoconjunctivitis [(6)]. The LD 50 – for this organism were 8×10^3 CFU [(7)]. Then 45 mice were taken and injected 1/p with 8×10^3 CFU (0.25ml of bacterial suspension) three mice were sacrificed at 2 days intervals and different lesions were recorded and pieces of lesions were fixed in 10% neutral buffered formalin, processed routinely, cut at 5 M thickness and stained with hematoxyline and eosin (H×E) and examined under light microscope [(8)].

The Results and Discussion

The Spleen:

The lesions began as a congestion, edema and infiltration of neutrophils causing a microabscesses in white pulp (fig-1) and at 2nd week there is a hyperplasia of white pulp due to proliferation of mononuclear cells (lymphocytes, macrophages and plasma cells) with germinal centres formation, also there is hyperplasia of reticuloendothelial cells lining red pulps and splenic cords hyperplasia. Finally the mononuclear cell hyperplasia of white pulp accompanied with fibroblastic proliferation.

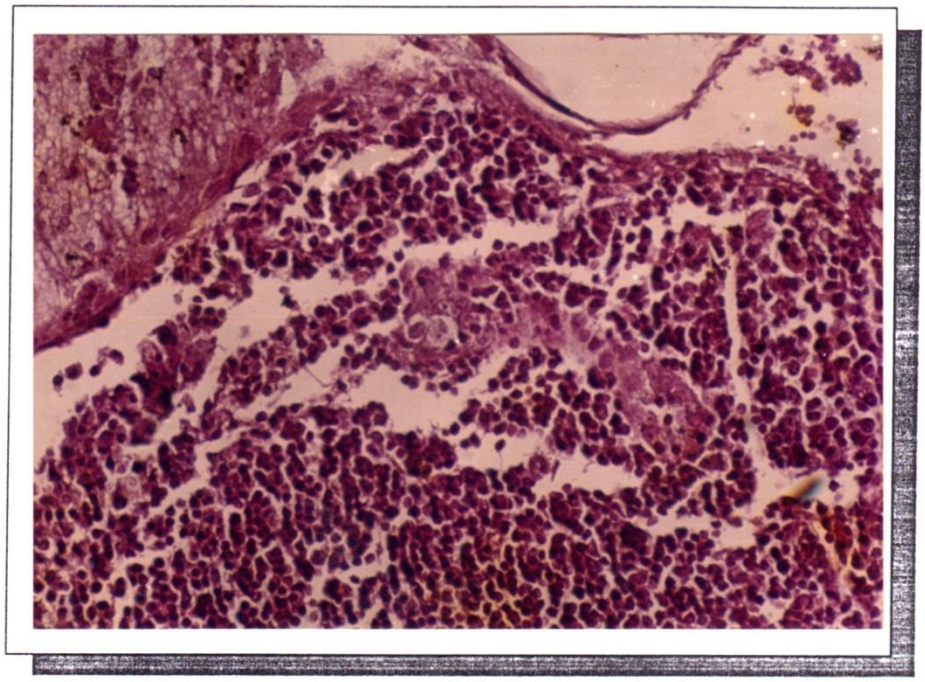

Fig. – 1 : spleen showed infiltration of neutrophils and mononuclear cells in the white and red pulps forming abscess (H *δ*E) X200

The Liver:

The lesions began as a coagulative necrotic areas with a pyknotic and Karyorrhectic nuclei (Fig-2), also infiltration of neutrophils in these areas causing microabscesses. At 2nd and 3rd weeks post infection the microabscess transform into early granulomatous lesions with replacing of neutrophils by mononuclear cells (lymphocytes, macrophages and plasma cells) and fibroblasts at the portal area and adjacent to central vein. Chronic cholangitis and cholecystitis were accompanied the hepatic lesions during the 3rd and 4th weeks post infection.

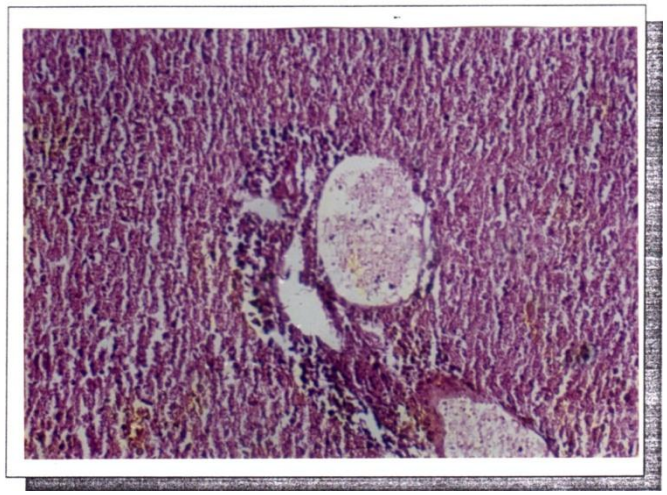

Fig. 2 : liver tissue showed coagulative necrosis and infiltration of neutrophils in the portal area (H*δ*E) X100

The Lungs:

The main lesions were suppurative bronchiolitis, congestion and peribronchial lymphoid hyperplasia at the first and second weeks post infection, also there is infiltration of mononuclear cells and fibroblast in alveolar walls causing the interstitial pneumonia together with edema, emphysema and focal pulmonary fibrosis (Fig-3).

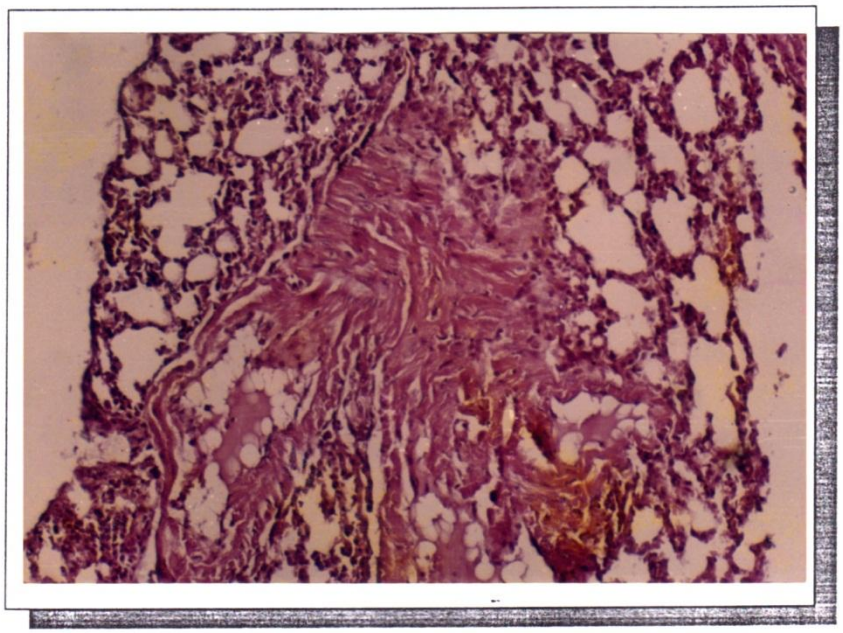

Fig. 3: lung tissue showed chronic interstitial pneumonia (H*δ*E) x200

The Kidneys:

The lesion began as on acute interstitial nephritis characterized by infiltration of neutrophils between glomeruli and renal tubules, these inflammatory cells replaced by mononuclear cells (lymphocytes, macrophages and plasma cells) and finally with fibroblasts proliferation to become chronic interstitial nephritis also dilation of Bowmon's capsule and proliferation of mesangial cells (Fig-4).

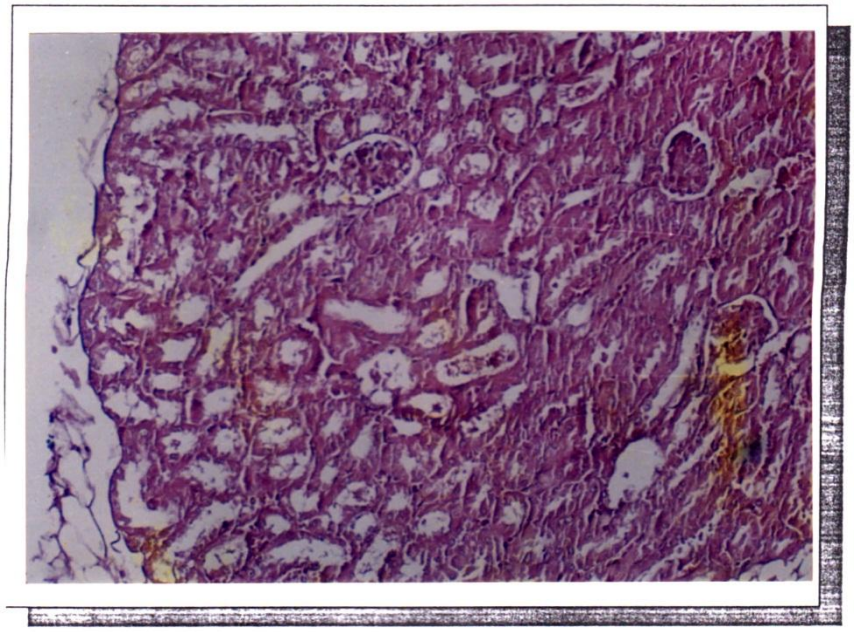

Fig. 4 : kidney showed dilation of Bowman's space and degeneration of renal tubules (H*δ*E) X100

The Brain and meninges:

There is infiltration of neutrophils, perivascular leukocytic cuffing and focal gliosis and degeneration of neuron cells (Fig-5).The similar cellular infiltrations were seen together with lymphocytes and macrophages in meningeal tissues, also congestion of blood vessels in meninges.

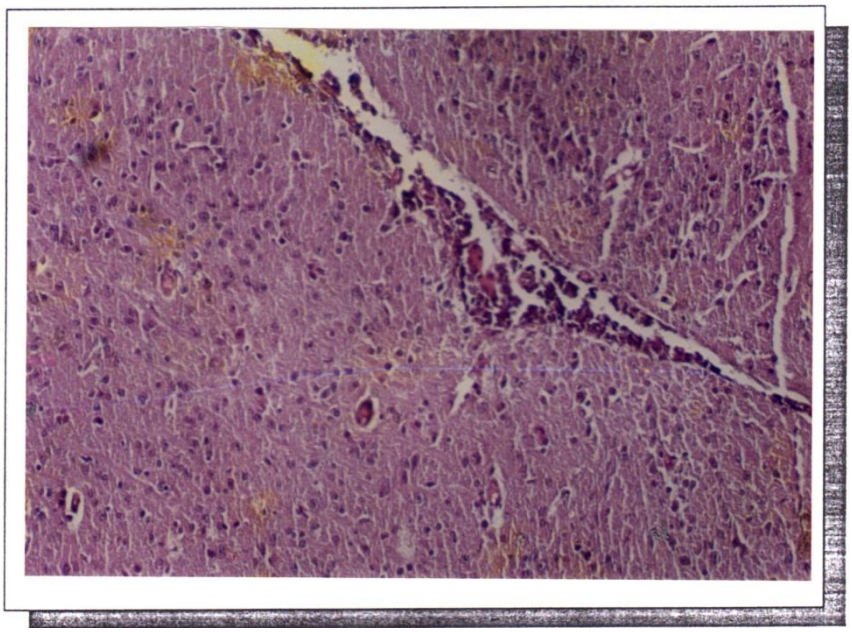

Fig. 5 : Brain showed perivascular leukocytic cuffing (H*δ*E) X100

The Intestine:

It showed hyperplasia of Peyer's patches due their infiltration by lymphocytes, macrophages and plasma cells, sloughing of the intestinal epithelia lining the mucosa (Fig-7) due their infiltration by neutrophils and lymphocytes and macrophages together fibroblasts proliferation later on in the submucosal layer of the intestine.

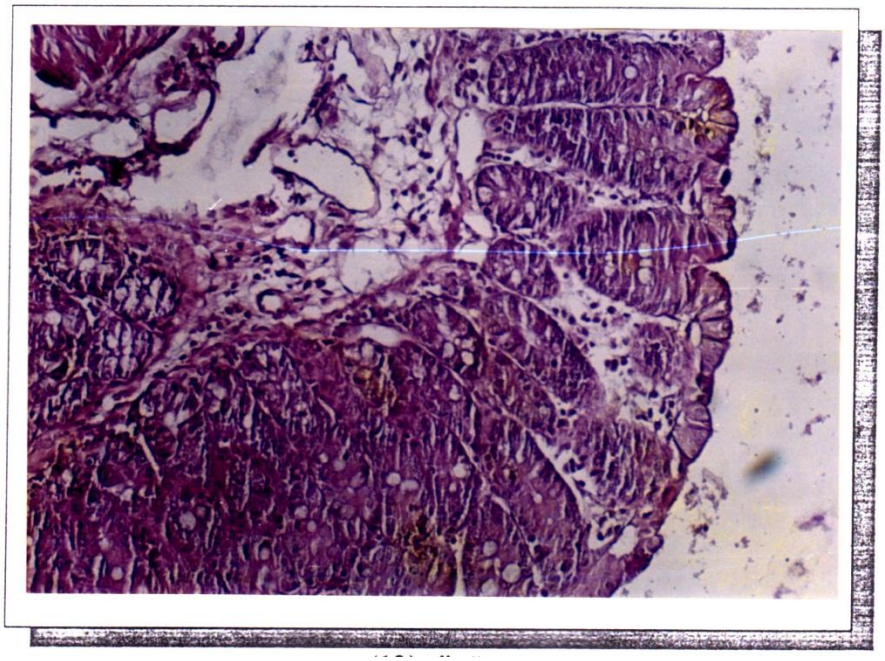

Fig. 6 : Intestine showed infiltration of neutrophils in mucosal layer and mucous secretion in their lumen (H*δ*E) X200

The Periton:

There is infiltration of neutrophils, congestion and these cellular infiltrations replaced by lymphocytes and macrophages in peritoneal tissue and around blood vessels, also focal fibrosis of peritoneal tissue later on.

The Heart:

It showed congestion, infiltration of neutrophils in the epicardium and myocardium, these cellular infiltrations replaced by mononuclear cells (lymphocytes and macrophages) later on (Fig-7).

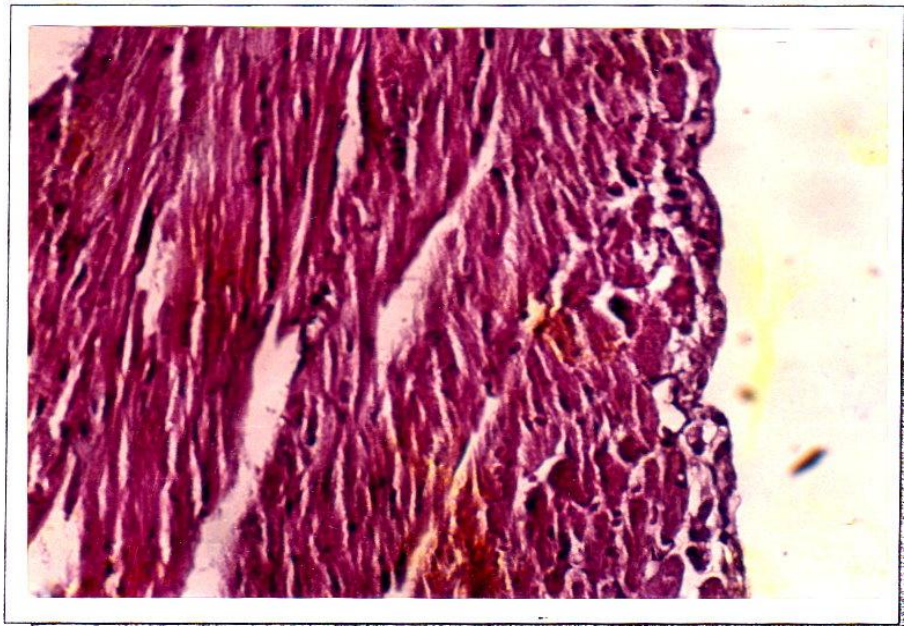

Fig. 7 : Heart tissue showed mild inflammatory cells infiltration and degeneration of myocardial fibres (HδE) X200

The Mediastinal Lymph Node:

The lesions began as acute lymphadenitis characterized by infiltration of neutrophils, lymphocytes and macrophages with a focal microabscesses formation in the lymphoid tissue. During the 3rd and 4th weeks the lymphoid tissue showed hyperplasia of lymphoid follicles with the new germinal centers formation. Also some of neutrophils, macrophages and lymphocytes infiltrated the medullary sinuses and fibrosis of lymphoid tissue later on.

The infection with *Shigella dysenteriae* type 1 induced sever lesions in this study, it began as an infiltration of neutrophils, congestion and edema forming a micro abscesses which were prominent in liver and spleen. These cellular infiltrations were also seen in the heart, lymph node, brain, periton. These inflammatory cells infiltration in these areas occurred under the effect of multiplication of *Shigella dysenteriae* and their endotoxins which considered to a chemotactic factor for neutrophils at the beginning of infection through 24 hrs post infection and then replaced by lymphocytes and macrophages later on [(9)]. These inflammatory cellular response associated with fibroblasts proliferation as a defence mechanism against microbial infection during the chronic stage of infection[(10)], the infection with this microbial agent involve the whole body organs following the l/p experimental infection by *Shigella dysenteriae* type 1, indicated that theses bacterial agents proliferated from the site of injection (periton) and through lymph and blood reach into different organs and induced the different lesions similarly with other species of Shigella due to their multiplication and production of the endotoxins [(11,12)] in these organs even in brain which rarely infected with this microbial agent[(13)] whereas, other organs such as intestine severely infected[(14)] together with other internal organs in this study. Similar bacterial distribution and pathological lesions were demonstrated in other internal organs with other species of Shigella and Salmonella in mice [(12, 15)].

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