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## RESEARCH ARTICLE

Study on Some Pathological aspects of *Salmonella enteridis* Experimental Infection in MiceKhalil H. Al-Jeboori<sup>1</sup>, Laheeb J. Al-Hashimi<sup>1</sup> and Haitham I. Baqir<sup>2</sup>

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## Abstract

Infections with *Salmonella enteridis* consider the main reason of food poisoning in man and infect other animals species and avian. For the study of pathological lesions caused by *S. enteridis* in mice. A local strain of this microbial agent were obtained from the central public health laboratory. Reidentification for this bacterial strain were done using, biochemical test, API – 20, Biomerieux Vitek instrument and slide agglutination test. The LD<sub>50</sub> were  $1.4 \times 10^6$  CFU, then this LD<sub>50</sub> dose were I/P inoculated in thirty three mice and eleven mice inoculated with phosphate buffer saline (pbs) as a control group. Three mice were sacrificed every three days intervals for 40 days. Among the pathological lesions were micro abscesses and granulomas seen in liver along the experimental periods. Amyloid, lymphoid hyperplasia and different pathological inflammatory lesions were recorded in liver, gall bladder, spleen, pancreas, lungs, intestine, kidneys, heart, brain and meninges, lymph nodes and in periton.

**Conclusion:** *S. enteridis* experimental infection gave different pathological lesions in the different organs.

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## Introduction

Infections with *Salmonella enteridis* consider the main reason of food poisoning in man causing Gastroenteritis and infect other animals and avian species including different pathological disorder (1). In certain cases these microbial agents colonize the avian intestine as in apparent carriers and during fecal excretions, it transmitted to other birds and meat contamination occurred (1, 2). *S. enteridis* considered the intra and extra – cellular invasive pathogen pass through ileum epithelia into peyer's patches and through blood or lymphatic vessels reach into other organs (3). Murine salmonellosis is a common disease caused by *S. enteridis* and *S. typhimurium*, associated with severe enteritis and septicemia in other internal organs (4). For the importance of *S. enteridis* in human and animals causing different pathological disorders; this study aimed to identify the different pathological lesions associated with experimental infection of mice with this microbial agent.

## Materials and Methods

A local strain of *Salmonella enteridis* type D obtained from the central public health laboratory. The strain was re identified again as a *S. enteridis* using biochemical tests, API 20 E together with Biomerieux Vitek instrument and slide agglutination test. The lethal dose – 50 (LD – 50) for this microbial agent was  $1.4 \times 10^6$  CFU according to (5) methods.

## Animals Inoculation:

Forty four mice were taken and thirty three were inoculated I/P with 0.25 ml of *S. enteridis* suspension ( $1.4 \times 10^6$  CFU of bacterial suspension) and eleven mice were inoculated I/P with 0.25 ml of phosphate buffer saline (pbs) as a control group. Three mice were sacrificed at three days intervals and one mouse from the control group along the experiment period (40 days). Pieces of pathological lesions were fixed in 10% neutral buffered formalin, processed

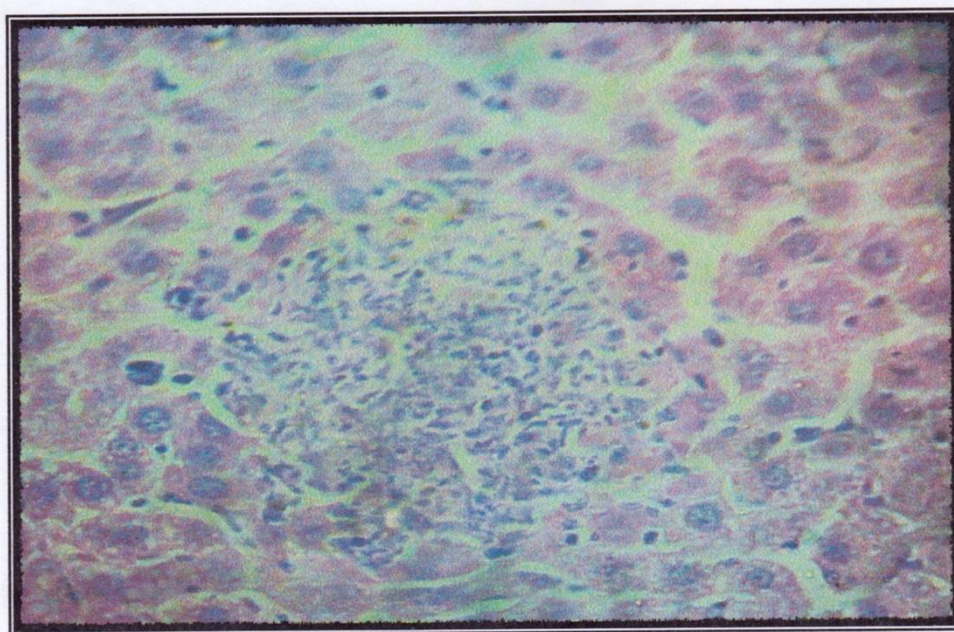
routinely, cut at 5  $\mu$ m thickness and stained with hematoxylin and eosin (H&E) and examined under light microscope (6).

## The Results and Discussion

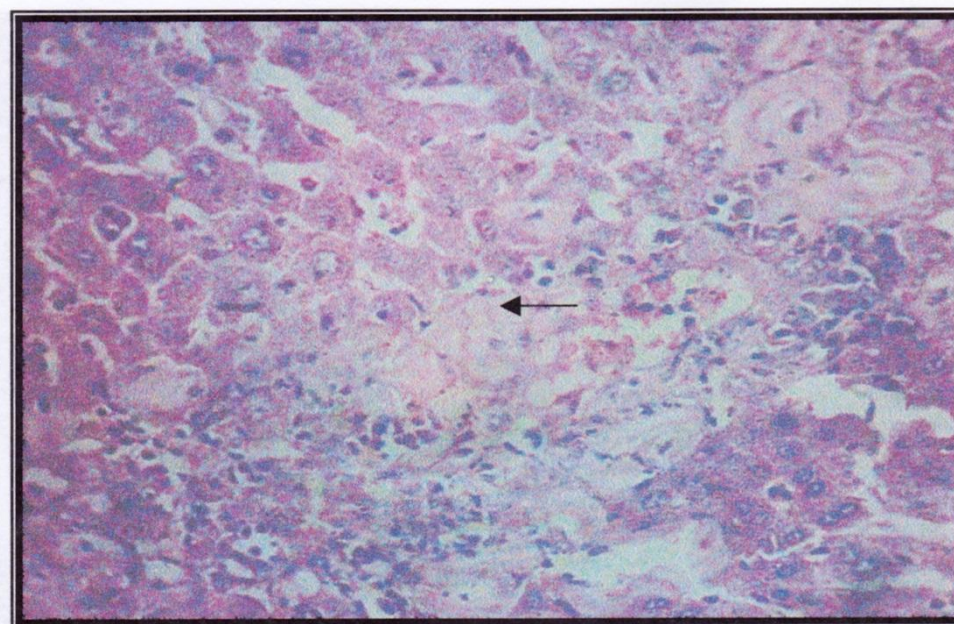
### Histopathological Changes:

#### The Liver:

The lesions began as a micro abscesses composed of the multifocal infiltration of the neutrophils in the liver parenchyma (**Fig – 1**) and adjacent the blood vessels (central vein and in the portal areas), the neutrophils were replaced by mononuclear cells (lymphocytes and macrophages) during the 2<sup>nd</sup> week to form early granulomatous during 3<sup>rd</sup>, 4<sup>th</sup> week of experimental infection. Amyloid infiltration were seen in the sinusoidal walls later on (**Fig – 2**).



**Fig .1 : Liver tissue: showed microabscess composed of neutrophils infiltration ( Hx E) X 400**

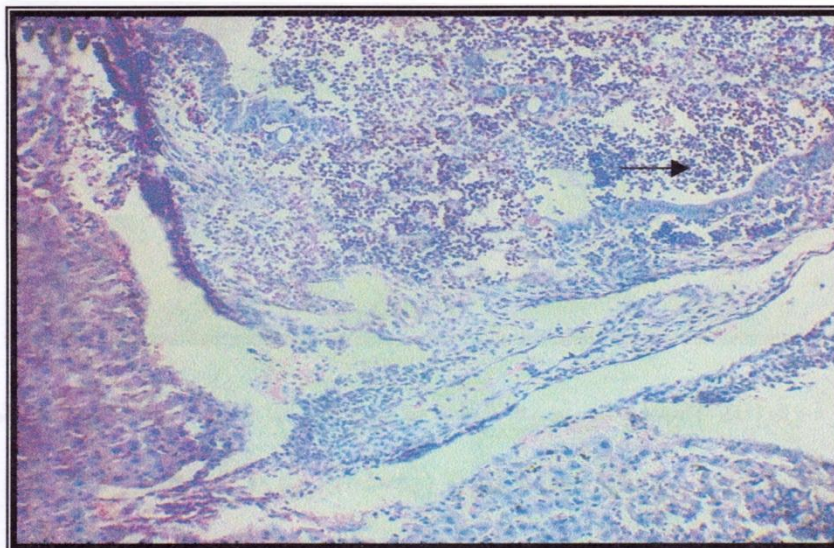


**Fig.2 : Liver : tissue infiltrated with amyloid .( Hx E) X 400**



### The Gall bladder and Bile ducts:

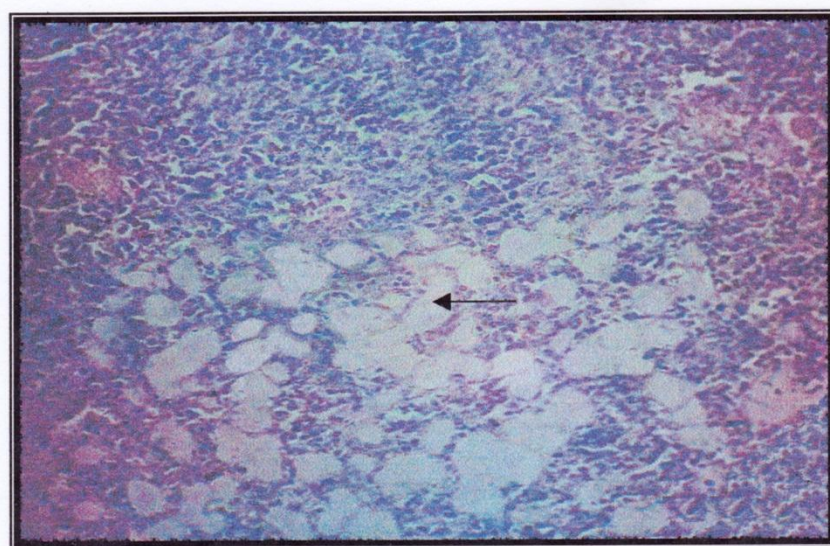
The similar neutrophils infiltration were seen in the lumen of gall bladder and bile duct (**Fig – 3**), these cellular infiltrates were replaced by mononuclear cells at 2<sup>nd</sup> week and at 3<sup>rd</sup> week the mononuclear cells together with fibroblasts were seen in the gall bladder wall with sloughing of their epithelial lining causing chronic cholecystitis.



**Fig-3 : Gall bladder tissue: showed filling their lumen with plus exualade (HxE) X200**

### The Spleen:

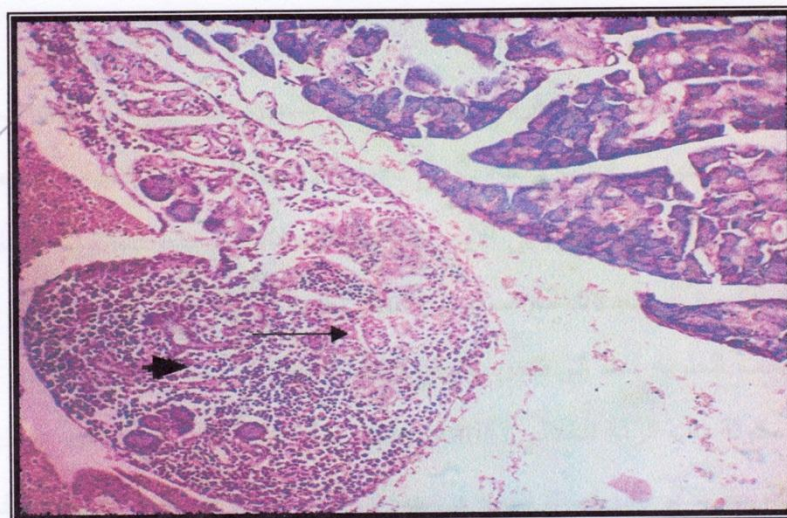
There is extensive lymphoid hyperplasia of white pulp and sever congestion of the red pulp and accentric arterioles of the white pulp. Amyloid infiltrations were seen in the white pulp during the 3<sup>rd</sup> and 4<sup>th</sup> weeks postinoculation(**Fig – 4**).



**Fig – 4 :S.plenic tissue : showed lymphoid hyperplasia of white pulp and amyloid infiltration ( Hx E ) x 200**

### The Pancreas:

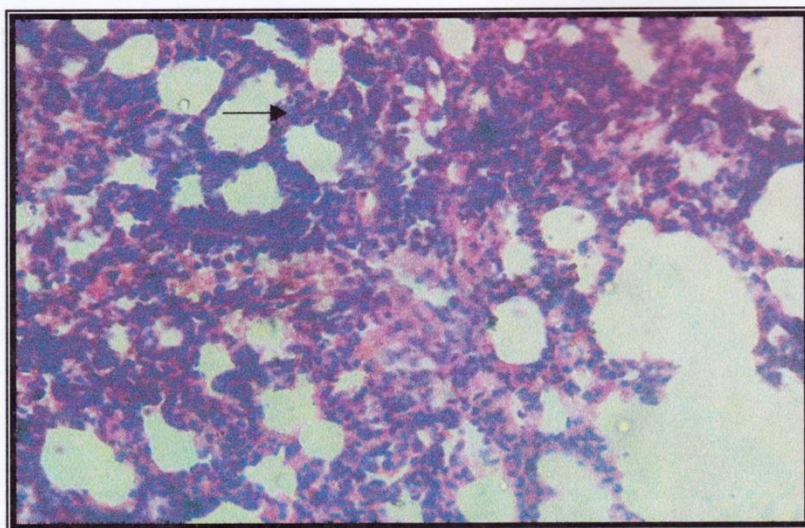
There is infiltration of neutrophils in the peripancreatic tissue and in the pancreas causing distraction of some pancreatic acini(**Fig – 5**). The neutrophils were replaced by mononuclear cells (lymphocytes and macrophages) during the 2<sup>nd</sup> week postinoculation and during the 3<sup>rd</sup> week postinoculation the mononuclear cells and fibroblasts were replaced the infected pancreatic tissue.



**Fig-5 : pancreatic tissue: showed infiltration of neutrophils replacing their acini ( Hx E) x200**

**The Lungs:**

There is peribronchiallymphoid tissue hyperplasia and interstitial pneumonic lesion began as infiltration of neutrophils and congestion of alveolar capillaries the neutrophils were replaced by lymphocytes and macrophages at 2<sup>nd</sup> week together with fibroblasts at the 3<sup>rd</sup> week (Fig – 6).

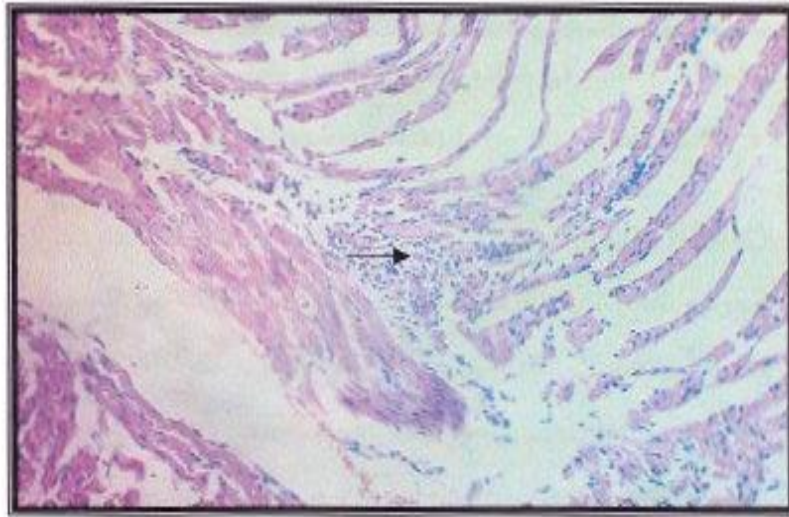


**Fig -6 : lung tissue: showed interstitial pneumonia ( Hx E) x 200**

**The Heart:**

There is infiltration of neutrophils in adipose tissue surrounded the heart and in the myocardium (Fig – 7), the neutrophils were replaced by mononuclear cells and fibroblasts causing focal epicarditis and myocarditis.

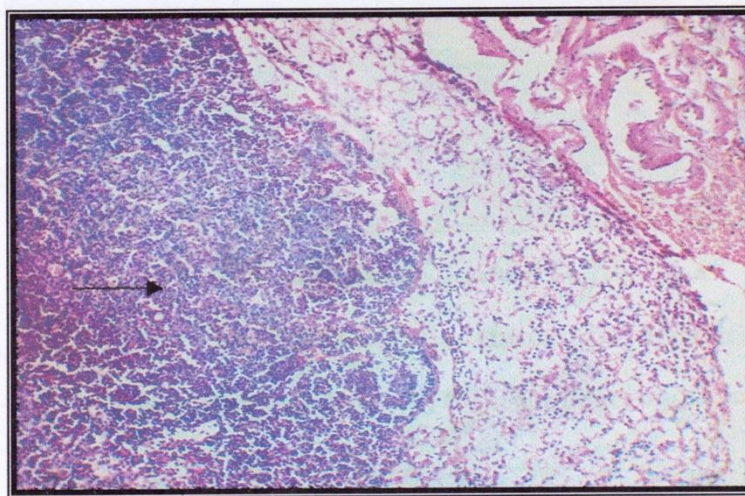




**Fig-7 : Heart : showed neutrophils infiltration in myocardium ( Hx&E) x 200**

**The Lymph nodes:**

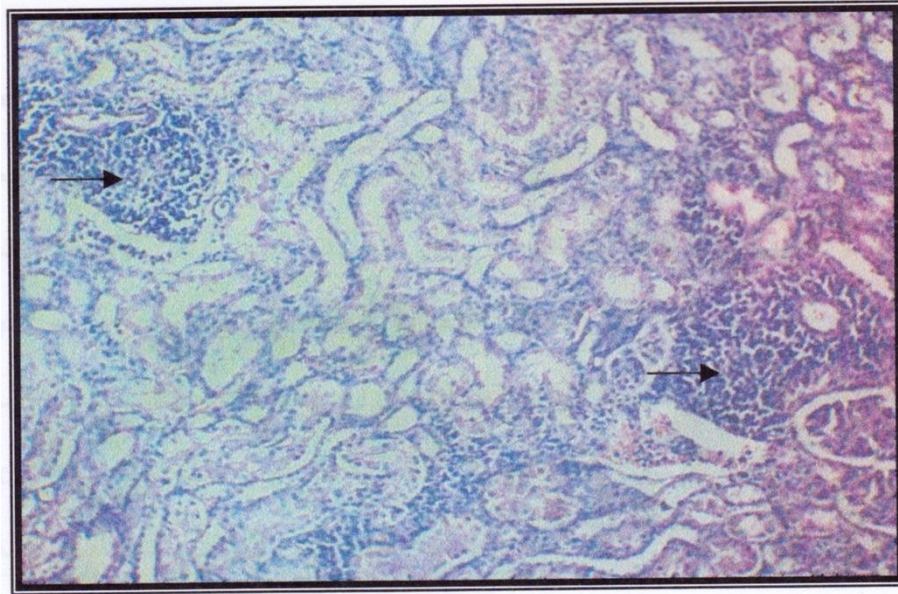
There is extensive lymphoid hyperplasia of cortical region with the secondary lymphoid follicles with germinal centers formation (**Fig – 8**). Also reticuloendothelial cell hyperplasia of the medullary sinuses and neutrophils infiltration in the capsule.



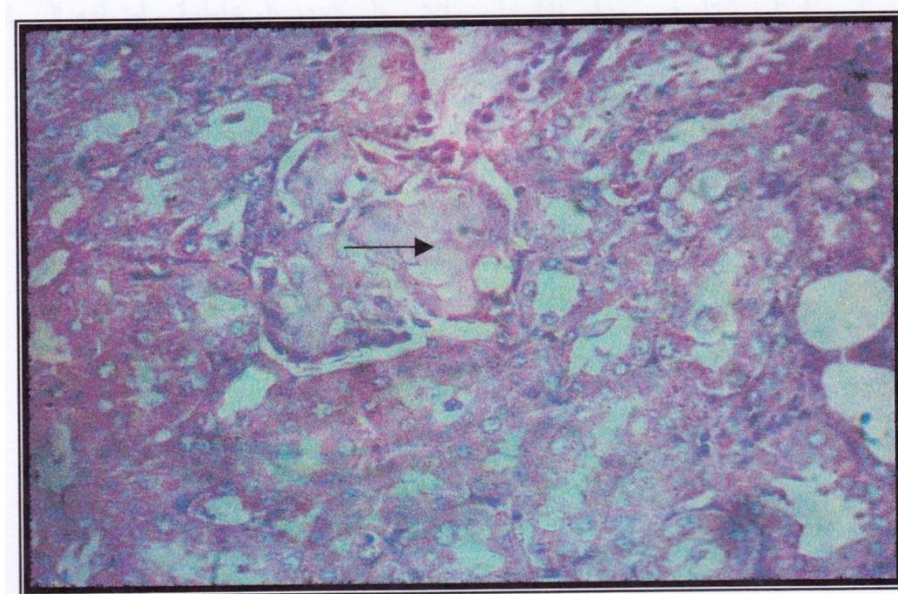
**Fig-8: lymphoid tissue: showed extensive , lymphoid hyperplasia and infiltration of neutrophils in the capsule ( Hx&E) x 200**

**The Kidney:**

There is neutrophils, lymphocytes and macrophages infiltration at the renal interstitial tissue (**Fig – 9**). The similar cellular infiltrations were seen in the renal pelvis and in the renal capsule and periureteric area. Amyloid infiltrations were seen in the glomeruli (**Fig – 10**).



**Fig-9 : Renal tissue: showed infiltration of neutrophils and mononuclear cells in the renal interstitial tissue ( Hx E) x 200**

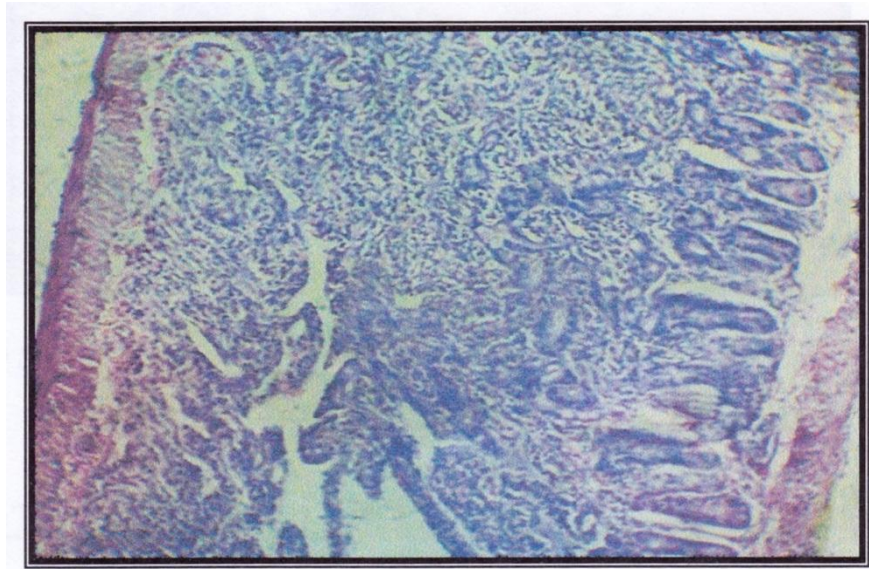


**Fig-10: Renal tissue: showed infiltration of amyloid in the glomeruli ( Hx E ) x400**

#### **The Intestine:**

There is infiltration of neutrophils lymphocytes and macrophages at the intestinal mucosa. Also, these cellular infiltrations together with mucin were seen in intestinal lumen. In chronic cases sloughing of intestinal epithelia and intestinal wall fibrous replacing the intestinal mucous glands (**Fig – 11**).

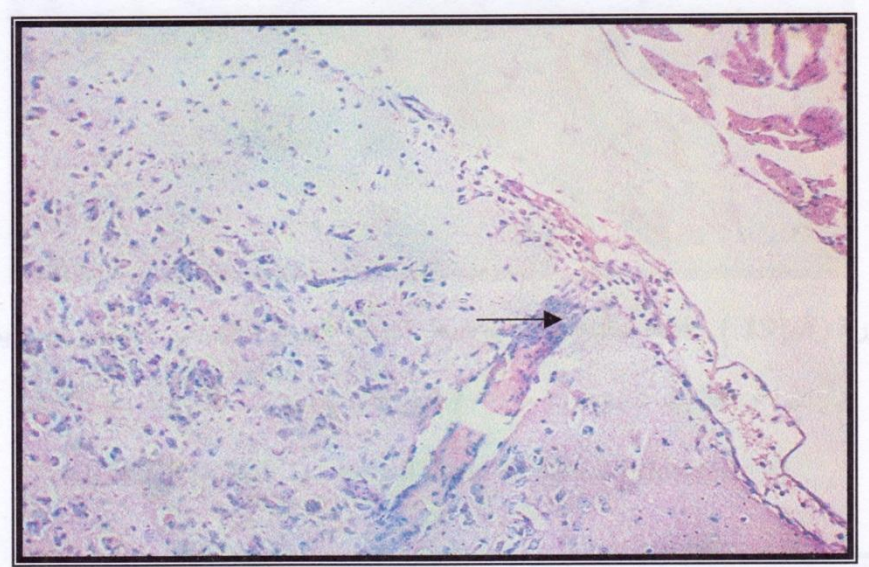




**Fig-11: Intestinal tissue: showed infiltration of mononuclear cells and fibroblasts in the intestinal wall and sloughing of their mucosal epithelia ( Hx E) x 200**

#### **The Brain and Meninges:**

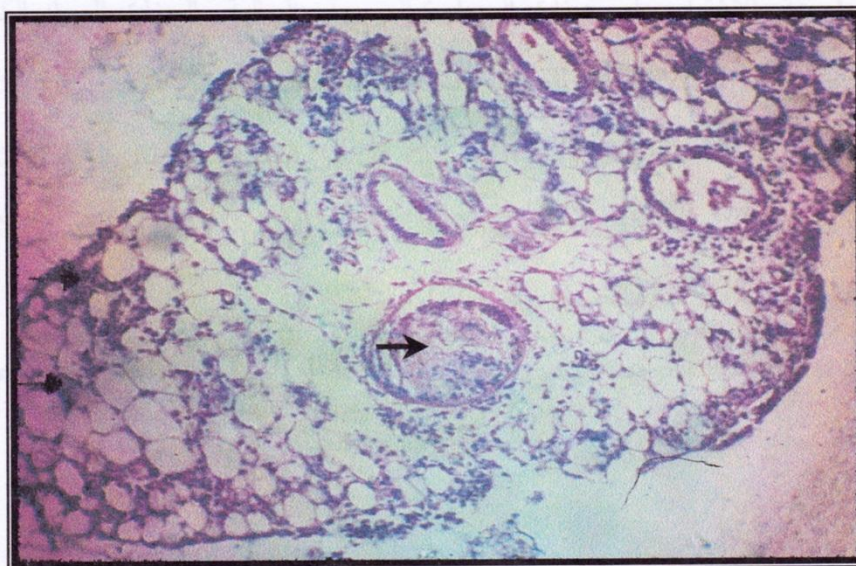
There is perivascular leukocytic cuffing (**Fig – 12**) in the brain and meninges, focal gliosis and neural degeneration.



**Fig -12: Brain : showed congestion of their blood . vessels and perivascular cuffing ( Hx E) x 200.**

#### **The Peritoneum:**

There is infiltration of neutrophils which replaced by lymphocytes and macrophages at the 2<sup>nd</sup> week and these inflammatory cells together with fibroblasts were seen in peritoneal tissue. Also thrombosis (**Fig – 13**) of peritoneal blood vessels.



**Fig-13 :periton: showed infiltration of neutrophils and thrombosis of their blood vessels ( Hx E) x 200 .**

The infection with *S. enteridis* associated with neutrophils infiltration due to the *S. enteridis* endotoxins which attract the neutrophils in all the infected organs, similarly reported by (7). During the 2<sup>nd</sup> and 3<sup>rd</sup> weeks, these neutrophils infiltration transform into early and chronic granuloma which seen in liver similarly reported by (8, 9, 10, 11, 12). During the experimental infection by *S. typhimurium* and *S. typhi* and by *S. paratyphi* A, B. lymphoid hyperplasia in the lymph nodes and in the spleen were reported by (9) following experimental infection with these microbial agents due recurrent immunological stimulation by microbial endotoxins. Amyloid infiltration in different organs such as liver, spleen, kidney were also reported in other animal species following experimental salmonella infection (13) similar lesions were reported in animals used for antisera preparation (14). Other lesion such as encephalitis and meningitis were reported in children due to *S. enteritis* (15). Other important lesions such cholecystitis, cholangitis, enteritis, pancreatitis, pneumonia, myocarditis and interstitial nephritis were seen in the mice in this study were also reported by other markers (10, 11, 12) indicated that these bacterial agents were invasive bacteria infect the all organs and cause the various lesions similarly reported for other salmonella species during the natural and experimental infection in the different animals species (8, 13).

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