Pathological and clinicopathological studies on reperfusion of ischemic intestine

By
A. H. Osman*; M.M. Bashandy**; Noha M. Said**; W.S. Elghoul***
*Department of Pathology; ** Department of Clinical Pathology; *** Department of Surgery, Faculty of Veterinary Medicine Cairo University

SUMMARY

The present work was carried on dogs as experimental model to assess the histological and clinicopathological changes that associated with ischemia-reperfusion injury of intestine.

Serum biochemical changes with correlation to histopathological alterations as a result of induction of ischemia-reperfusion were clarified.

A total 15 healthy male mongrel dogs of weighing 9 to 12 kg and ageing one to five years old were allocated into 3 groups according to the pathway of induction of intestinal ischemia.

Blood samples were taken from dogs before and after induction of ischemia-reperfusion injury for determination serum biochemical and blood coagulation changes. A significant hypercoagulability in all groups at the end of ischemic and reperfusion times were recorded. Serum biochemical changes associated with IRI in intestine revealed a significant increase in (ALT, AST, ALP, LDH and AMYL), enzymes activities with ischemic and reperfusion times, when compared with their activity at zero-time. Blood urea, creatinine and glucose determination showed significant increase in their concentration during ischemia and reperfusion times. Also C-reactive protein revealed a significant increase in their titer with all groups at the end of ischemic time and with reperfusion time.

Ischemia/reperfusion lesions of intestinal region revealed congestion, hemorrhages and cyanosis associated with damage of intestinal epithelium and serosal layer with different grades. The histological findings demonstrated severe destruction of intestinal segment after reperfusion in comparison with ischemia. On the other hand, ischemia/reperfusion were reflected on hepatic and renal parenchyma with different grades according to ischemic pathway.

Keywords: Ischemia/reperfusion – Colon - Coagulation changes- Serum biochemical changes- Gross Pathology – Histopathology.

Referred by
Prof. Dr. Mahmoud Samy Ahmed
Professor of Pathology, Fac. Vet. Med., Cairo Univ.

Prof. Dr. Rawhia M. Emran
Professor of Pathology, Animal Health Research Institute, Dokki
INTRODUCTION

Ischemia means failure of blood supply or inadequate blood flow to an area of tissues (Guido and Isabelle, 1996). It is a dual defect of oxygen deficit and carbon dioxide excess (Benjaminute et al., 1993) and it is a state existing when an organ or tissue has it's arterial perfusion lowered relatively to it's metabolic need (Neville, 2000). It occurs when blood flow to an organ or tissue has ceased "complete ischemia" or is abnormally low "partial ischemia" (Roderick and Keith, 1992).

The chief causes of arterial local ischemia are usually, (1)- occlusion of the arterial supply, (2)- narrowing of arterial lumen, or (3)- mechanical external pressure upon an artery (Ritchie, 1990).

Reperfusion injury is tissue injury that occurs following re-establishment of circulation after an ischemic event (Rustin et al., 1994). Reperfusion is the treatment of choice to save viable tissue following acute ischemia of a vascular territory, but reperfusion of ischemic tissue is associated with local and systemic leukocytic activation and trafficking "specially neutrophil" (Danielle et al., 2005).

Reperfusion injury occurs after restoration of blood flow subsequent to an episode of ischemia (Rustin et al., 1994). Reperfusion of ischemic tissues can lead to several complications that may worsen the ischemic lesion and produce systemic alterations and a life threatening situation (Marcelo and Winston, 2005). Reperfusion injury of ischemic tissue represents an acute inflammatory response that can cause significant morbidity and mortality (Ming Zhang et al., 2004).

Bowel ischemia is a common complex disorder with various primary causes, clinical presentation, high mortality rate and it account for at least 3% of human deaths in USA. In horses ischemic gastrointestinal disease is the most common cause of death; especially acute mesenteric thrombosis carries high morbidity and mortality rate so any delay in diagnosis or treatment aggravates the patients outcome (Hung et al., 1999). Acute mesenteric ischemia is life threatening vascular emergency, due to inadequate tissue perfusion so it requires early diagnosis, intervention to adequately restore mesenteric blood flow and prevent bowel necrosis and patient death (Upendra et al., 2005).

One of the most common causes of intestinal ischemia are mechanical obstruction which occur mainly due to, hernial strangulation, volvulus "twisting of intestinal
segment” and intussusception. In the latter form excessive peristaltic contraction drives the affected segment of bowel immediately distal lies in a sleeve like manner over it and some of the mesentery is included in the portion of bowel that is pushed forward and the resulting local edema lead to local ischemia (Neville, 2000).

Intestinal ischemia reperfusion injury may lead to intestinal barrier dysfunction, resulting in bacterial translocation, which can lead to adult respiratory distress syndrome and sepsis. The most important side effect of ischemia reperfusion injury are multiple organ dysfunction, which may lead to death (Upendra et al., 2005).

The purpose of this study was to determine the degree intestinal damage during ischemia and reperfusion as well as liver and kidney tissue reaction. Coagulation changes, serum biochemical and C-reactive protein alterations were to be clarified.

**MATERIALS AND METHODS**

**Animals**

This study was done on fifteen healthy male mongrel dogs, weighing 9 to 12 kg and ageing one to five years old. The dogs were housed in a separate kennels, and kept under observation for 14 days prior to the day of operation.

**Experimental design:**

A total of 15 male dogs were subjected to induction of intestinal ischemia and reperfusion. The animals were classified into three groups each one contained five animals for induction of (I/RI) through three different pathways.

**Surgical procedures:**

**Anesthesia:** dogs were premedicated with 0.04 mg/Kg atropine sulphate (S/C) and 0.1-0.5 mg/Kg diazepam (I/M), induction of anesthesia was performed by injection of sodium thiopental 2.5 %solution.

**Group (1): Cranial mesenteric artery (CMA) occlusion**

Five dogs were subjected to 2 hours of CMA occlusion and 2 hours of reperfusion. After ischemic period the plastic clips were removed and reperfusion allowed for 2 hours.

**Group (2): Segmental ligation**

Five dogs were subjected to 2 hours of segmental ligation and 2 hours reperfusion “ligation released” . A 30-cm of jejunal segment; a 30-cm of colon segment and their arteries and veins were identified and isolated. Each segment was ligated by using a plastic clips placed at each end of and their arteries and veins were identified and isolated.

**Group (3): Intraluminal distention and decompression**
Five dogs were subjected to 2 hours of intraluminal distention and 2 hours of decompression. A 30 cm of jejunal segment was occluded by using a plastic clips placed at each end. Sterile Ringer's lactated solution was infused into the lumen to induce an intraluminal distention. After ischemic period the plastic clips were removed and reperfusion allowed.

**Sampling**

**A- Blood samples:**
Five blood samples were collected from abdominal aorta from each animal before operation and one & two hours from induction of ischemia as well as perfusion. The blood samples were divided into two parts. The first part was anticoagulated by sod. citrate solution 3.8% (1.8 ml blood +0.2 ml sod. citrate solution), then centrifuged at 2000 rpm for ten minutes for plasma separation and was used for performing the coagulation parameters. The second one collected in plain centrifuge tube and was allowed to clot, then centrifuged at 2000 rpm for ten minutes for serum separation.

**I-Coagulation studies:**

a) Prothrombin time (PT):- measurement the time of fibrin clot formation is according to Moll and Ortel (1997).

b) Activated partial thromboplastin time (APTT):- measurement the time of fibrin clot formation is according to Poller and Thompson, (1992).

**II-Serum studies:**

1. Alanine and aspartate amino transferases (ALT and AST) :
Colorimetric determination of ALT and AST activities was performed according to Reitman and Frankel (1957).

2. Alkaline phosphatase (ALP):
Colorimetric determination of alkaline phosphatase activity was done according to Tietz (1986).

3. Lactate dehydrogenase (LDH):
The principle of the test was depending on the idea that the LDH specifically catalyzes the oxidation of lactate to pyruvate with subsequent reduction of NAD to NADH. The rate at which NADH forms is proportional to LDH activity. The method described determines NADH absorbance increase per minute at 340 nm (Kachmar and Moss, 1976).

4. Amylase (AMYL):
Colorimetric determination of amylase activity was done according to Marshall (1980).

5. Blood urea:
Blood urea was measured colorimetrically at wave length 578 nm was done according to (Searcy, et al., 1967).

6. Serum creatinine:
Serum creatinine was determined according to Henery (1968).
7. Blood glucose:
Colorimetric determination of glucose was determined according to Howanitz (1984)

8. C-reactive protein (CRP):
CRP was determined semi-quantitatively by rapid latex agglutination test according to Fischel (1967).

III. Statistical analysis of data:
All numerical data were statistically evaluated for the mean and standard error for each group. The significance of the results was determined by conducting the least significance difference between different times outlined by Sendecor and Cochran (1989).

IV. Histopathological examination:
Tissue specimens were taken from mucosa and serosa of colon segment at the end of ischemic time and at the end of reperfusion. Liver and kidney specimens were taken at the end of reperfusion time. All specimens were fixed in 10% neutral buffer formalin. The fixed specimens were then trimmed, washed and dehydrated in ascending grades of alcohol, cleared in xylene, embedded in paraffin, sectioned at 4–6 µ thickness and stained with haematoxyline and eosin Bancroft (1994). Histological, grading for epithelial damage of intestinal mucosa was assessed as described by Park et al. (1991), table (1). Hepatic injury was estimated using an ordinal scale modified from Camargo et al. (1997) table (2).

Table (1) Park's histological grading of intestinal epithelial damage:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal mucosa</td>
</tr>
<tr>
<td>1</td>
<td>Subepithelial space</td>
</tr>
<tr>
<td>2</td>
<td>Extended Subepithelial space</td>
</tr>
<tr>
<td>3</td>
<td>Epithelial lifting along villous side</td>
</tr>
<tr>
<td>4</td>
<td>Denuded villi</td>
</tr>
<tr>
<td>5</td>
<td>Loss of villous tissue</td>
</tr>
<tr>
<td>6</td>
<td>Crypt layer infarction</td>
</tr>
<tr>
<td>7</td>
<td>Transmucosal infarction</td>
</tr>
<tr>
<td>8</td>
<td>Transmural infarction</td>
</tr>
</tbody>
</table>
Table (2): Histological grading of liver injury

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No apparent injury on light microscopic examination</td>
</tr>
<tr>
<td>I</td>
<td>Hepatocytes swelling</td>
</tr>
<tr>
<td>II</td>
<td>Cytoplasmic vacuolization, nuclear pyknosis, apoptosis</td>
</tr>
<tr>
<td>III</td>
<td>Focal necrosis</td>
</tr>
<tr>
<td>IV</td>
<td>Massive necrosis of whole hepatic cords &amp; hemorrhages</td>
</tr>
</tbody>
</table>

RESULTS

I- Coagulation changes:

Results of the coagulation study are illustrated in table (3). Intestinal groups showed significant prolongation in PT and PTT time at the end of ischemic and reperfusion times, associated with a significant increase in INR ratio at the end of ischemic time.

Table (3): *Values of coagulation changes during intestinal I/R in different groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Times</th>
<th>PT (seconds)</th>
<th>PTT (seconds)</th>
<th>INR Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cranial Mesenteric Artery occlusion (CMA)</td>
<td>Zero-time</td>
<td>12.67 ± 0.85&lt;sup&gt;a&lt;/sup&gt;</td>
<td>19.00 ± 1.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.11 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>30.50 ± 0.20&lt;sup&gt;b&lt;/sup&gt;</td>
<td>30.00 ± 0.41&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.93 ± 0.02&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>60.00 ± 0.00&lt;sup&gt;c&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;c&lt;/sup&gt;</td>
<td>6.65 ± 0.06&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>60.00 ± 0.00&lt;sup&gt;f&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;f&lt;/sup&gt;</td>
<td>7.10 ± 0.04&lt;sup&gt;f&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>60.00 ± 0.00&lt;sup&gt;c&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;c&lt;/sup&gt;</td>
<td>7.77 ± 0.13&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Segmental ligation (SL)</td>
<td>Zero-time</td>
<td>11.75 ± 0.63&lt;sup&gt;a&lt;/sup&gt;</td>
<td>21.33 ± 0.62&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.03 ± 0.04&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>15.33 ± 0.47&lt;sup&gt;b&lt;/sup&gt;</td>
<td>26.33 ± 0.85&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.39 ± 0.0 b&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>20.00 ± 0.82&lt;sup&gt;c&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1.89 ± 0.10&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>5.58 ± 0.05&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>7.63 ± 0.15&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Dilatation Decompression (DD)</td>
<td>Zero-time</td>
<td>12.67 ± 0.47&lt;sup&gt;a&lt;/sup&gt;</td>
<td>12.33 ± 0.24&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.11 ± 0.04&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>15.67 ± 0.62&lt;sup&gt;b&lt;/sup&gt;</td>
<td>14.67 ± 0.47&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.42 ± 0.07&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>18.00 ± 0.41&lt;sup&gt;c&lt;/sup&gt;</td>
<td>18.67 ± 0.47&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1.64 ± 0.03&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>5.65 ± 0.18&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>60.00 ± 0.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>7.39 ± 0.20&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

*Values represent mean ± S.E.
Mean with different alphabetical letters are significantly difference
II-Biochemical Parameters:-

Statistical analysis of biochemical parameters of canine model subjected to intestinal ischemia reperfusion are illustrated in table (4).

Assay of serum enzymes (ALT, AST, ALP, LDH and AMYL) in intestinal ischemic groups gave a significant increase in their activities with ischemic and reperfusion times, when compared with those of corresponding at zero-time.

The results of blood urea, creatinine and glucose showed significant increase in their concentration during ischemia and reperfusion times table (5).
Table (4): *Changes in enzymatic activities during intestinal I/R in different groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Times</th>
<th>ALT (iu/L)</th>
<th>AST (iu/L)</th>
<th>ALP (iu/L)</th>
<th>LDH (iu/L)</th>
<th>Amylase (iu/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cranial Mesenteric Artery occlusion (CMA)</td>
<td>Zero-time</td>
<td>13.25 ± 2.29a</td>
<td>18.75 ± 1.38a</td>
<td>28.80 ± 1.76a</td>
<td>119.67 ± 3.27a</td>
<td>512.00 ± 9.34a</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>18.25 ± 1.78a</td>
<td>22.50 ± 1.55a</td>
<td>35.95 ± 1.25b</td>
<td>176.93 ± 4.59b</td>
<td>597.25 ± 7.34b</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>24.50 ± 1.44b</td>
<td>30.75 ± 1.65b</td>
<td>37.65 ± 0.92b</td>
<td>206.45 ± 5.90c</td>
<td>676.00 ± 5.40c</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>30.75 ± 0.63c</td>
<td>42.00 ± 2.83c</td>
<td>37.68 ± 0.93b</td>
<td>230.00 ± 2.45d</td>
<td>682.00 ± 11.97c</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>35.25 ± 1.75c</td>
<td>63.67 ± 3.27d</td>
<td>40.68 ± 0.78c</td>
<td>290.00 ± 3.67c</td>
<td>696.67 ± 7.32c</td>
</tr>
<tr>
<td>Segmental Ligation (SL)</td>
<td>Zero-time</td>
<td>13.25 ± 1.75a</td>
<td>17.00 ± 2.12a</td>
<td>26.98 ± 0.96a</td>
<td>114.00 ± 4.92a</td>
<td>542.00 ± 13.18a</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>19.25 ± 1.44b</td>
<td>27.50 ± 1.94b</td>
<td>30.73 ± 0.79b</td>
<td>157.45 ± 5.84b</td>
<td>609.50 ± 12.37b</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>27.00 ± 2.39c</td>
<td>31.00 ± 2.68b</td>
<td>32.85 ± 1.90b</td>
<td>210.25 ± 5.82c</td>
<td>631.67 ± 6.98b</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>29.25 ± 0.48c</td>
<td>39.75 ± 2.95c</td>
<td>32.13 ± 0.72b</td>
<td>237.00 ± 5.16d</td>
<td>628.00 ± 11.52b</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>36.50 ± 1.55d</td>
<td>62.50 ± 3.47d</td>
<td>32.77 ± 1.50b</td>
<td>289.67 ± 2.87c</td>
<td>642.25 ± 11.54b</td>
</tr>
<tr>
<td>Dilatation Decompression (DD)</td>
<td>Zero-time</td>
<td>9.50 ± 0.87a</td>
<td>16.00 ± 1.96a</td>
<td>26.40 ± 1.34a</td>
<td>109.67 ± 2.72a</td>
<td>530.75 ± 5.48a</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>16.00 ± 1.22b</td>
<td>21.00 ± 2.20a</td>
<td>29.13 ± 0.88a</td>
<td>166.00 ± 5.12b</td>
<td>599.00 ± 11.84b</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>21.50 ± 1.32c</td>
<td>29.00 ± 2.04b</td>
<td>31.23 ± 0.79b</td>
<td>215.00 ± 2.68c</td>
<td>670.00 ± 9.39c</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>35.00 ± 1.63d</td>
<td>31.67 ± 2.05b</td>
<td>32.10 ± 1.14b</td>
<td>255.00 ± 2.04d</td>
<td>677.50 ± 6.12c</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>36.50 ± 2.25d</td>
<td>46.33 ± 1.84c</td>
<td>34.83 ± 1.19c</td>
<td>286.50 ± 4.57e</td>
<td>660.50 ± 14.08c</td>
</tr>
</tbody>
</table>

*Values represent mean ± S.E.
Mean with different alphabetical letters are significantly difference.
### Table (5): *Values of urea, creatinine & glucose change intestinal I/R*

<table>
<thead>
<tr>
<th>Group</th>
<th>Times</th>
<th>Urea (mg/dl)</th>
<th>Creatinine (mg/dl)</th>
<th>Glucose (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cranial Mesenteric Artery occlusion (CMA)</td>
<td>Zero-time</td>
<td>14.03 ± 1.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.67 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>59.63 ± 5.08&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>25.75 ± 0.10&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.70 ± 0.09&lt;sup&gt;a&lt;/sup&gt;</td>
<td>79.27 ± 2.46&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>28.70 ± 2.48&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.75 ± 0.07&lt;sup&gt;b&lt;/sup&gt;</td>
<td>173.95 ± 5.74&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>45.45 ± 0.59&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.90 ± 0.04&lt;sup&gt;c&lt;/sup&gt;</td>
<td>184.00 ± 9.80&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>44.90 ± 0.20&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1.08 ± 0.02&lt;sup&gt;d&lt;/sup&gt;</td>
<td>197.50 ± 8.78&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Segmental ligation (SL)</td>
<td>Zero-time</td>
<td>17.75 ± 1.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.57 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>83.40 ± 2.90&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>29.16 ± 1.68&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.60 ± 0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>116.80 ± 2.94&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>29.16 ± 1.68&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.72 ± 0.03&lt;sup&gt;b&lt;/sup&gt;</td>
<td>119.50 ± 1.43&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>29.33 ± 1.22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.91 ± 0.03&lt;sup&gt;c&lt;/sup&gt;</td>
<td>165.00 ± 2.86&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>36.67 ± 1.81&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.96 ± 0.02&lt;sup&gt;d&lt;/sup&gt;</td>
<td>182.93 ± 8.56&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Dilatation decompression (DD)</td>
<td>Zero-time</td>
<td>15.73 ± 1.21&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.70 ± 0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>67.67 ± 3.70&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>20.50 ± 0.41&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.72 ± 0.03&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>77.80 ± 1.73&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>24.78 ± 0.95&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.74 ± 0.03&lt;sup&gt;b&lt;/sup&gt;</td>
<td>135.00 ± 5.72&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>32.00 ± 1.63&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.83 ± 0.04&lt;sup&gt;c&lt;/sup&gt;</td>
<td>183.60 ± 0.99&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>33.67 ± 1.25&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.86 ± 0.04&lt;sup&gt;c&lt;/sup&gt;</td>
<td>187.83 ± 8.70&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

*Values represent mean ± S.E.

Mean with different alphabetical letters are significantly difference.

**III-C-reactive protein changes:**

A significant increase in C-reactive protein titer noted in all intestinal groups at the end of ischemic time and with reperfusion time table (6).
Table (6): *Value of CRP changes during intestinal I/R

<table>
<thead>
<tr>
<th>Group</th>
<th>Times</th>
<th>CRP (mg/µl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cranial Mesenteric Artery Occlusion (CMA)</td>
<td>Zero-time</td>
<td>6.00 ± 0.00\textsuperscript{a}</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>7.50 ± 1.50\textsuperscript{b}</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>9.00 ± 1.70\textsuperscript{c}</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>15.00 ± 1.70\textsuperscript{d}</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>21.00 ± 1.70\textsuperscript{e}</td>
</tr>
<tr>
<td>Segmental Ligation (SL)</td>
<td>Zero-time</td>
<td>6.00 ± 0.00\textsuperscript{a}</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>6.00 ± 0.00\textsuperscript{a}</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>7.50 ± 1.50\textsuperscript{b}</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>10.50 ± 1.50\textsuperscript{c}</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>15.00 ± 1.73\textsuperscript{d}</td>
</tr>
<tr>
<td>Dilatation Decompression (DD)</td>
<td>Zero-time</td>
<td>6.00 ± 0.00\textsuperscript{a}</td>
</tr>
<tr>
<td></td>
<td>1 hr ischemia</td>
<td>10.50 ± 1.50\textsuperscript{b}</td>
</tr>
<tr>
<td></td>
<td>2 hr ischemia</td>
<td>13.50 ± 1.50\textsuperscript{c}</td>
</tr>
<tr>
<td></td>
<td>1 hr reperfusion</td>
<td>15.00 ± 1.22\textsuperscript{d}</td>
</tr>
<tr>
<td></td>
<td>2 hr reperfusion</td>
<td>19.50 ± 1.50\textsuperscript{e}</td>
</tr>
</tbody>
</table>

*Values represent mean ± S.E.
Mean with different alphabetical letters are significantly difference.

**IV-Pathological results:**

**A-Cranial mesenteric artery occlusion:**

The gross lesions were recorded after one hour from cranial mesenteric artery occlusion revealed congestion of mesenteric blood vessels and oedema of intestinal wall. On the other hand, severe intestinal congestion and hemorrhages from mesenteric borders and vessels were demonstrated after 2 hours from occlusion with cyanosis of intestinal wall (Plate, A-1). Histologically, cranial mesenteric artery occlusion revealed subepithelial oedema and shrinkage of intestinal villi grade (2). The lamina propria of colon segment showed hemorrhages and leukocytic infiltration mainly neutrophils, macrophages and lymphocytes (Plate, A-2). Sloughing of serosal mesothelial lining of colon with intact basement membrane was noticed. The subepithelial connective tissue showed oedema with inflammatory cells infiltration mainly neutrophils. Focal hemorrhagic areas were also seen (Plate, A-3).

Hemorrhages, swelling and cyanosis of colon were noticed
after 1 hour from reperfusion. Mesenteric hemorrhages and cyanosis were remarked after 2 hours from reperfusion (Plate, A-4). Histological section of colon revealed subepithelial oedema with atrophy of intestinal villi grade (2). The lamina propria showed severe hemorrhages with leukocytic infiltration mainly neutrophils and macrophages (Plate, A-5). Serosal mesothelial lining was desquamated with intact basement membrane. The subepithelial connective tissue revealed congestion, oedema, hemorrhages and inflammatory cells infiltration mainly neutrophils and macrophages (Plate, A-6).

The pathological alterations in case of reperfusion of colon appeared more severe in comparison with ischemic stage.

**Liver** after two hours of reperfusion showed perihepatitis characterized by capsular and subcapsular infiltration with neutrophils and macrophages. The hepatocytes showed swelling, granular cytoplasm and pyknotic nuclei grade (II). Some hepatocytes revealed intracytoplasmic fat globules of variable size with peripheral eccentric nuclei (Plate, A-7).

**Kidney** after two hours of reperfusion showed shrinkage of glomerular tuft and widening of Bowman's space. The proximal convoluted tubules showed swelling of its epithelial lining with intraluminal albumin cast (Plate.A-8).

**B-Segmental ligation:**

Hemorrhages and congestion appeared from the mesenteric borders and vessels of the ligated colonic segments after one hour of ligation. Colonic segments showed moderate congestion and hemorrhage with increase of its thickness after two hours from ligation (Plate, B-1). Histologically, the colonic mucosa showed subepithelial oedema extended along villous side and atrophy of some intestinal villi grade (3). The lamina propria revealed inactive intestinal glands and leukocytic infiltration mainly neutrophils and macrophages (Plate, B-2). Colonic serosa after 2 hours of segmental ligation revealed desquamation of mesothelial lining with intact basement membrane. The subepithelial connective tissue showed oedema, congestion of blood capillaries and inflammatory cells infiltration mainly neutrophils (Plate, B-3).

One hour after release of ligation colon segment revealed hemorrhages, congestion and cyanosis of colonic segment in comparison with non ligated segments. Severe congestion and hemorrhages were observed after 2 hours from ligation released (Plate, B-4). Reperfusion of colon segment after 2 hours was associated with damage of colonic mucosa characterized by...
transmucosal infarction accompanied with severe hemorrhages and leukocytic infiltration grade (7). The lamina propria showed necrosis of intestinal gland, hemorrhages and leukocytic infiltration (Plate, B-5). Colonic serosa showed sloughing of mesothelial lining. Oedema of subepithelial connective tissue with marked dilation of lymph vessels as well as inflammatory cells infiltration mainly neutrophils, macrophages and lymphocytes were observed (Plate, B-6).

Liver showed subcapsular leukocytic infiltration mainly neutrophils and macrophages which extended to the hepatic parenchyma. The hepatic lobules showed congestion of its sinusoids with few mononuclear cells infiltration. Swelling of hepatic cells with central pyknotic nuclei were seen. Some hepatocytes showed focal area of coagulative necrosis characterized by loss of cell details with presence of architecture outline grade (III) (Plate, B-7).

Kidney showed hypercellularity of glomerular tufts that included proliferation of endothelial and mesangial cells with leukocytic infiltration. The proximal and distal convoluted tubules showed swelling of their epithelial lining with intraluminal albuminus droplets (Plate, B-8).

C- Segmental dilatation / decompression:
Mild congestion was observed after one hour from dilatation. Congestion of colon segment was increased after two hours from dilatation, with an engorgement of colonic arteries with blood (Plate, C-1). Histologically, colonic mucosa showed sloughing of its epithelial lining and loss of villous tissue grade (4). The lamina propria was infiltrated with neutrophils and macrophages (Plate, C-2). Colon serosa after two hours of dilatation displayed sloughing of some mesothelial cells. Oedema and leukocytic infiltration of subepithelial connective tissue were observed. Moreover the inflammatory reaction was extended to the muscular layer which characterized by focal aggregation of inflammatory cells inbetween the muscle bundles mainly neutrophils and macrophages, massive leukocytic infiltration of both serosa and muscular layers by numerous numbers of neutrophils were noticed (Plate, C-3).

One hour after decompression, colon segment became more congested and hemorrhagic with severe engorgement of blood vessels with blood in comparison with colon segment during dilatation. Hemorrhage and congestion was increased after two hours from decompression (Plate, C-4). Colonic mucosa demonstrated loss of intestinal villi and oedema of subep-
The lamina propria showed atrophy of intestinal gland with massive hemorrhages (Plate, C-5). Decompression of colon segment after two hours was accompanied with desquamation of mesothelial lining with intact basement membrane. The subepithelial connective tissue showed oedema which extended to muscular layer. Inflammatory cells were infiltrated both serosa and muscular layers which consisted of neutrophils and macrophages (Plate, C-6).

Liver showed congestion of both central veins and sinusoids after two hours of colon segment decompression. Inflammatory cells were infiltrated the sinusoids and also around the central veins which consisted mainly of neutrophils and macrophages. Hepatic lobules showed disorganization of hepatic cords, nuclear pyknosis and apoptosis of hepatocytes grade (II) (Plate, C-7).

Kidney revealed obvious histological reaction especially the cortical region. The glomerular tufts were dilated and occupied most of Bowman's capsule which also contained free red blood cells. The renal tubules showed necrobiotic changes especially the proximal convoluted tubules (Plate, C-8).
Plate. (A)

Fig. (1) Colon showing congestion and hemorrhages from mesenteric vessels were demonstrated after 2 hours from occlusion.

Fig. (2) Intestinal villi of colon revealed grade (2) and the lamina propria showed hemorrhages and leukocytic infiltration after 2 hours from occlusion (H&E X200).

Fig. (3) Colon showing sloughing of serosal mesothelial lining oedema and inflammatory cells infiltration subepithelial connective tissue after 2 hours from occlusion (H&E X200).

Fig. (4) Colon showing mesenteric hemorrhages and cyanosis were remarked after 2 hours from reperfusion.

Fig. (5) Intestinal villi revealed grade (2) and the lamina propria showed severe hemorrhages with leukocytic infiltration after 2 hours from reperfusion (H&E X200).

Fig. (6) Colon showing desquamation of serosal mesothelial lining with congestion, oedema, hemorrhages and inflammatory cells infiltration subepithelial connective tissue (H&E X200).

Fig. (7) Hepatic lobule showing perihepatitis and hepatocytes revealed grade (II) (H&E X200).

Fig. (8) Kidney showing shrinkage of glomerular tuft and swelling of tubular epithelial lining with intraluminal albuminous cast (H&E X200).
Plate. (B)
Fig.(1) Colonic segments showing moderate congestion and hemorrhage after two hours from ligation.
Fig.(2) Colonic mucosa showing subepithelial edema extended along villous side grade (3) (H&E X200).
Fig.(3) Colonic serosa after 2 hours of segmental ligation revealed desquamation of mesothelial lining with edema, congestion and inflammatory cells infiltration of subepithelial connective tissue (H&E X200).
Fig.(4) Reperfusion of colon segment after 2 hours showing hemorrhages, congestion and cyanosis.
Fig.(5) Reperfusion of colon segment after 2 hours showing transmucosal infarction grade (7) (H&E X200).
Fig.(6) Colon showing desquamation serosal mesothelial lining and the subepithelial connective tissue revealed congestion, edema, hemorrhages and inflammatory cells infiltration (H&E X200).
Fig.(7) Hepatic lobules showing perirepititis and hemorrhage; hepatocytes revealed grade (III) (H&E X200).
Fig.(8) Kidney showing hypercellularity of glomerular tufts and swelling of tubular epithelial lining with intraluminal albumin cast (H&E X200).
Plate. (C)

Fig. (1) Colon segment after two hours from dilatation showing engorgement of colonic arteries with blood.

Fig. (2) Colonic mucosa showing sloughing of its epithelial lining and loss of villous tissue grade (4) (H&E X200).

Fig. (3) Colon serosa after two hours of dilatation displayed sloughing of mesothelial cells, oedema and leukocytic infiltration of subepithelial connective tissue (H&E X200).

Fig. (4) Colon showing hemorrhage and congestion after two hours from decompression (H&E X200).

Fig. (5) Colonic mucosa demonstrating grade (5), the lamina propria showing atrophy of intestinal gland with massive hemorrhages after two hours from decompression (H&E X200).

Fig. (6) Decompression after two hours of colon segment after showing desquamation of mesothelial lining, oedema and inflammatory cells infiltration of subepithelial connective tissue extended to muscular layer (H&E X200).

Fig. (7) Hepatic lobule showing disorganization of hepatic cords, nuclear pyknosis and apoptosis of hepatocytes grade (II) (H&E X200).

Fig. (8) Kidney showing dilatation of glomerular tufts with red blood cells with necrobiotic changes renal tubules (H&E X200).
DISCUSSION

Timely diagnosis and surgical intervention for ischemia are challenging clinical problems (Williams, 1998). The restoration of blood flow is the treatment of choice to save viable tissue following acute ischemia of a vascular territory. Nerveless, reperfusion of ischemic tissues can be accompanied by significant local, remote and systemic inflammatory events that may limit the beneficial effects of blood flow restoration (Danielle et al., 2005).

The current study was planned to demonstrate the gross and histopathological changes following ischemia-reperfusion injury as well as some serum biochemical constituents.

Ischemia-reperfusion injuries was promoted the regional production of inflammatory mediators, expression of cell adhesion molecules on endothelial and immune cell surfaces and increase the precoagulatory properties of vascular endothelial cells (Schwarz et al., 1999). The increase in precoagulatory properties was in agreement with the result of the present work, as it obvious from results that prothrombin time (PT), activated partially thromboplastine time (PTT) and INR ratio was significant prolonged after induction of ischemia in all groups in comparison with value before induction of ischemia. In addition, more prolongation occurs after reperfusion. The same results were noticed by (Monreal et al., 2000). Imaz et al., (2002) reported that hypercoagulation may be attributed to the release of endogenous mediators such as platelet activation factor in inflammatory disorders. Other studies claimed hypercoagulability to absorption of endotoxin from intestine (Weiss and Rashid, 1998).

Measurement of serum biochemical markers may be useful in management and diagnosis of ischemia-reperfusion injury. Assay of serum ALT and AST in our experiment showed a significant increase in its activity and may be due to tissue injury and inflammatory mediators which are associated with ischemia-reperfusion injury. It is well known that ALT enzyme is nearly specific for hepatocellular injury in dogs and increased serum levels parallel the magnitude of hepatocellular injury in acute cases. AST enzymes occur in most cells; however, it is useful in evaluating hepatocellular injury (Kaneko, 1997).

Reports of Upendra et al., (2005), were in a agreement with our results as they found that elevation in ALT and AST values in ischemia reperfusion mainly due to chemical mediators that cause cell injury and leakage of enzymes. This result was confirmed by the histopathological changes observed in liver after ischemia reperfusion.
injury, liver cells showed degenerative changes associated with congestion, mononuclear cells infiltration and some hepatocytes showed coagulative necrosis.

In the present work serum ALP showed a significant increase with intestinal ischemia-reperfusion, this could be attributed to tissue injury associated with ischemia and reperfusion (Wollin et al., 1981).

Serum ALP is found primarily in intestine, kidney, liver and bone; moreover kidney and intestine have the greatest activity per gram of tissue (Kaneko et al., 1997). This was in close agreement with the findings of higher ALP activities were associated with greater intestinal damage and renal infarction (Hoover et al., 1988). So increase in ALP increase probability of surgery and worse prognosis (Saulez et al., 2004).

LDH results of the present experiment recorded a significant increase in its activity with intestinal and renal ischemia-reperfusion. LDH activity is present in all the cells of the body predominantly in cytoplasm of the cell. Even small mass of damaged tissues causes leakage of enzymes and increasing its level in serum significantly (Kaneko et al., 1997). Such results were in agreement with that reported by Thompson (1990). As he found that LDH were more likely to be elevated during intestinal ischemia. Furthermore, Hoover et al., (1988), observed that LDH was significantly increased during renal ischemia-reperfusion. This increase may be contributing to tissue breakdown that occur during ischemia-reperfusion injury (Uday Kumar et al., 2003).

It is well known that α-amylase enzyme found in several tissues of dogs including intestine and kidney (Mocharla et al., 1990). Moreover the specificity of serum amylase increases as a clinical marker of exocrine pancreas (Corazza et al., 1994). The present study recorded a significant increase in amylase activity during intestinal ischemic time and continued with reperfusion, this was in agree with that recorded by Yang et al., (2004). This finding might be attributed to that formation of free radicals due to ischemia and reperfusion causing pancreatic injury resulting in escape of these enzymes to the circulation.

The obtained data of serum urea and creatinine in canine model subjected to intestinal ischemia-reperfusion injury showed a significant increase. These results were in agreement with Yang et al., (2004), who found that urea concentration increase significantly in rat after intestinal ischemia-reperfusion. The increase in urea concentration may be contributing to perfu-
sion defect that occur as a result of dehydration. Hypovolemia occur in this study mainly due to surgical intervention and hemorrhage from mesentery during ischemia-reperfusion. Dogs don't tolerate hypovolemia which may lead to development of acute renal failure. Development of azotemia associated with increase in creatinine concentration noted in the present study may be due to dehydration which lead to decrease in glomerular filtration rate or primary acute renal failure which developed from perfusion defect (Balint et al., 1975). Histopathological changes observed in renal tissue after ischemia-reperfusion as congestion and inflammatory cells infiltration mainly neutrophils support the changes in renal function tests.

Glucose estimation revealed significant hyperglycemia which appeared during ischemia and after reperfusion in all groups. This may be due to the effect of anesthetic drugs used during experiment; Ilkiw (2002) reported that dogs are capable of increasing cortisol levels in response to surgical stimulation. So that hyperglycemia occurs in response to increase in the cortisol level.

CRP is considered the most diagnostically important of the acute phase protein in humans (Maurer, 1985). Moreover, Caspi et al., (1987) indicates that it may be equally valuable in dogs. This interest in generated the potential for use of acute phase proteins to provide an early and reliable signal to the clinician of the presence of any inflammatory disease. The results of CRP in the present study indicated that CRP was significantly increasing in all groups. This result was in agreement with experimentally induction of inflammation in horse showed significant increase in CRP (Imaz et al., 2002).

The increase in CRP in all groups may be contributing to a defensive mechanism against further injury that occur during ischemia and continued after reperfusion (Kaneko et al., 1997). CRP level increase with the severity of bacterial translocation in acute intestinal obstruction (Cevikel et al., 2004).

Intestinal ischemic segment appeared thick, dark red to purple in color and edematous. The mucosa and neighboring mesentery become congested and hemorrhagic. During reperfusion, the bowel wall thickened progressively and become more congested hemorrhagic and blackened in color there were sever congestion, edema and hemorrhage of the mesentery. These findings were coincided with that reported by (Carol et al., 1991).

Acute intestinal ischemia was accompanied by massive infarction of the mucosa and inner muscular
layer. The bowel wall becomes congested, edematous and the mucosa undergoes necrosis and ulceration. Bleeding into the lumen is a consequence of the previous changes. The lesions are characteristically patchy and may affect both the small intestine and colon (Vinay et al., 1997).

The ascending colon is more susceptible to seromuscular layer damage (Robin et al., 2001). Experimental pathological study on horses subjected to ischemia for 70 minute and followed by 60 minute reperfusion revealed that after 70 minute of jejunal ischemia there was serosal capillary congestion but minimal edema or leukocyte infiltration. The mesothelial cell layer was either partially or completely absent. After reperfusion the mesothelial cell loss, serosal edema, erythrocyte and leukocyte infiltration were significantly increased. These findings agreed with that reported by Robin et al. (2001).

Other experiment on rats found that after ligation of mesenteric artery for one hour total villous loss with some crypts, normal stroma and patent vessels were noted (Upendra et al., 2005). While (Byron, 2001) found that after ligation of mesenteric artery for one hour and followed by one hour reperfusion in rats, the jejunal serosal and muscle layers loss it's mesothelial cell layer, serosal edema and increase neutrophils numbers.

After distention and decompression of the colon in rat, the mesothelial cell layer was completely absence with serosal edema and increased serosal neutrophils. The histological changes associated with clinical cases of colonic infarction include epithelial sloughing, congestion, hemorrhage, edema and necrosis of mucosa and edema and hemorrhage were observed in submucosa (Meschter et al., 1986). These findings were similar to that observed in our study in dogs.

Horse subjected to intestinal intraluminal distention /decompression for 120 minute recorded a mesothelial cell loss, moderate serosal edema, lymphatic dilution, and erythrocyte infiltration. After decompression, serosal edema, hemorrhage, and leukocyte infiltration increased (Robin et al., 2001). In other study performed in foals distention of the jejunum for 2 hour followed by decompression resulted in serosal edema and cellular infiltration (Lundin et al., 1989). In similar study intraluminal distention for 2 hour followed by 60 minute of decompression revealed mucosal neutrophilic infiltration (Dabareiner et al., 1993).

The changes that observed in small bowel are more dramatic than that observed in large bowel
over the same period of ischemia (Jubb and Kennedy, 2000). The abnormalities observed in the ischemic or reperfusion of colonic serosal layer was partially mesothelial cell loss (Robin et al., 2001). Colon suffer from thromboembolism disease showed epithelial sloughing, ulceration, necrosis and hemorrhage of the underlying lamina propria, and inflammatory cell infiltration. These findings were agreed with that observed by Barclay et al. (1980). On the other side, the epithelial sloughing was similar to that observed in spontaneous or experimentally induced intestinal ischemia in several species of animals and man (Hagluns et al., 1980).

Intestinal ischemia reperfusion injury may lead to intestinal barrier dysfunction, resulting in bacterial translocation, which can lead to adult respiratory distress syndrome and sepsis. The most important side effect of ischemia reperfusion injury is multiple organ dysfunctions, which may lead to death (Yang et al., 2004 and Upendra et al., 2005).

Conclusion:

Coagulation values revealed hypercoagulability is characteristic for ischemia - reperfusion injury. Also values of serum enzymes showed significant increase in their activities was associated with ischemia and continued even after reperfusion. A marked increase in C-reactive protein levels was highly associated with bacterial translocation and multiple organ dysfunctions that associated with ischemia reperfusion injury. Histopathological findings, clarified the ischemic picture of colon by different pathways as well as demonstrated that reperfusion lesions which were more severe in comparison with ischemia.

REFERENCES


of Virginia Polytechnic Institute and State University.


Henery, R. J. (1968): "Clinical Chemistry Principles and


"Identification of a specific self-reactive IgM antibody that initiates intestinal ischemia / reperfusion injury." Immunology, 101 (11): 3886-3891.


**Rustin, M; Moore; Alicia, L; Bertone; Michael, Q.; Bailey; William, W.; Muir,W. L. and Beard, M. (1994):** "Neutrophil accumulation in the large colon of horses during low flow ischemia and reperfusion."


دراسة باناثولوجية وباناثولوجية اكلينيكية على انقاص إنسيباد الدم ثم إعادة الإرواء في الأعصاب

أحمد عثمان*، مصطفى بشندى**، نهى سيد***، وحيد الغول****
قسم الباناثولوجيا - قسم الباناثولوجيا الإكلينيكية - قسم الجراحة والتخدير والأشعة - كلية الطب الباطني - جامعة القاهرة.

المصطلح العربي

أجريت هذه الدراسة على الكلاب كمثال لحيوانات التجارب لمعرفة التغييرات البايثولوجية والبايثولوجيا الإكلينيكية المصاحبة لانقاص الدم ثم إعادة الإرواء في الأعصاب.
تمت هذه الدراسة على 15 كلب من الكلاب الضالة من الجنسين والتي تزن حوالي 12-19 كجم وترموج أعمارها ما بين 5-10 سنوات. وقد تم تقسيم الكلاب إلى ثلاثة مجموعات تبعًا لطريقة انقاص إنسيباد الدم في الأعصاب.

تم تجميع عينات الدم قبل و بعد تعرض الكلاب لانقاص إنسيباد الدم وبعد إعادة الإرواء لمعرفة التغيرات الكيميائية وكذلك التغير في نسبة النحل. أُبرمت نتائج الفحص الكيميائي عن وجود زيادة مع انقاص إنسيباد (ALT, AST, ALP, LDH, Amylase) معنوية في نشاط كل من إنزيمات الدم وكذلك مع إعادة الإرواء المقارنة بنسبتهم قبل التجربة. وكذلك زيادة مستوى كل من البولينا والكرياتينين والجلوكوز. كما أوضحت نتائج التجربة عدوى فرط في نسبة التحلل مع جميع المجموعات أثناء نقص إنسيباد الدم وإنقاد الإرواء.

ظهرت نتائج التحليل الأخصائي لـ CRP عن وجود زيادة في هذا البروتين في نهاية الساعة الثانية من نقص إنسيباد الدم عن الأعصاب وكذلك بعد الإرواء.

وقد أوضحت الدراسة البايثولوجية وجود احتقان وانزفة ووزغة ولفة للإعاقة بدرجة إضافة أن انقاص إنسيباد الدم ثم إعادة الإرواء في الأعصاب. وأُبرمت النتائج على أن التغييرات البايثولوجية المصاحبة للإرواء كانت أكثر حدة عن تلك المصاحبة لانقاص إنسيباد الدم. وقد انعكست التغييرات المصاحبة لانقاص إنسيباد الدم ثم إعادة الإرواء في الأعصاب على كل من الكبد والكلي بدرجات متقاربة تبعًا للطريقة المستخدمة لانقاص إنسيباد الدم في الأعصاب.

المحمومون:

- د. محمود سامي
- د. رحية عمران