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EXPERIMENTAL STUDY

Bacterial translocation in experimental stroke: what happens to the gut barrier?

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Abstract: The reasons of post-stroke infections are still incompletely understood. Bacterial translocation (BT), the passage of viable microbes across an even anatomically intact intestinal barrier, has been described in many critical illnesses. To date, it has not been studied as a source of infection in an animal stroke model. To address this, a permanent left middle cerebral artery occlusion (MCAO) model in rats was used. After 24, 48, and 72 hours (h), sham and experimental groups were sacrificed and samples were taken for BT. Similarity between bacteria detected in tissues (blood, mesenteric lymph node, liver, spleen, and lung) and intestinal microflora was shown with phenotypic methods and antibiotyping. Possible ileum tissue injuries were shown by histopathologic examination (including morphometric analysis). Although there was no bacterial proliferation in the sham groups, 55.5 %, 45.4 %, and 30 % bacterial proliferation was detected in MCAO groups at post-operative hour 24, 48, and 72, respectively. In MCAO groups the bacterial proliferation in tissues and ileum tissue injury scores were higher over time compared to sham groups ($p<0.05$). Our findings support the view that stroke, itself leads to mucosal damage and bacterial translocation *(Tab. 5, Fig. 2, Ref. 27)*. Full Text (Free, PDF) *www.bmj.sk.*
Key words: stroke, bacterial translocation, ICAM-1, infection, inflammation, gut barrier.

Stroke is one of the most frequently encountered problems in neurology clinics. Infections and sepsis are the major complications that determine the morbidity and mortality in these patients (1–5) The incidence of infections, which are a leading cause of death in patients with stroke varies from 21 % to 65 % in the literature (1, 2). Although old age, diabetes mellitus, severe neurologic impairment, immunity disorders, malnutrition, unsafe swallowing with dysphagia, subsequent aspiration, and presence of oral pathogens are considered to be major contributors to the high incidence of infections and sepsis after stroke (3, 4), the reasons for the high incidence of infections and sepsis in patients sustaining a stroke are still incompletely understood and the mechanisms of infections complicating the clinical course of acute stroke have received limited investigation (5–8).

Damage to intestinal mucosa in inflammatory and metabolic responses to critical illnesses has been increasingly recognized (6–9). Bacterial translocation (BT) was defined as "the invasion of indigenous intestinal bacteria through the gut mucosa into normal sterile tissue, causing disease" (9–11). To date, it has not been studied as a source of infection in an animal stroke model. However, despite decades of research and clinical trials, there is inadequate information about the effects of stroke on gut barrier function and bacterial translocation (5–19).

To the best of our knowledge, the role of stroke, leading to bacterial translocation in an animal model, has not been investigated before. Therefore, in the study reported here, we investigated the effects of stroke on gut barrier and bacterial translocation in rats.

Materials and methods

Experimental design and Focal Brain Ischemia

Fiftyseven male Wistar rats weighing 220 to 250 g were anesthetized with ketamine hydrochloride (50 mg/kg intramuscularly). The animals were randomly assigned to either the left middle cerebral artery occlusion (MCAO) or sham group. The surgical procedure of permanent MCAO was performed as described elsewhere (14). Briefly, a 4-0 polypropylene (Ethicon, USA) monofilament suture was introduced into the common carotid artery under ketamine hydrochloride anesthesia, while body temperature was kept constant (37 °C) with heating pad. The filament was prepared by blunting the tip near a flame under 10x magnification. MCAO was accomplished by further advancing the filament in the internal carotid artery until a faint resistance was

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Tab. 1. Experimental Design.

Groups	(n)	Operation	**Postoperative evaluation date (hr)**
Group Sham I	6	Sham	**24**
Group Sham II	6	Sham	**48**
Group Sham III	6	Sham	**72**
Group I	9	MCAO	**24**
Group II	11	MCAO	**48**
Group III	**10**	**MCAO**	**72**

n – number of animals, MCAO – Median cerebral artery occlusion

felt; the filament was left there until sacrifice. After MCAO was performed, animals were returned to their home cages and allowed free access to food and water. For the sham group animals, all procedures except intraluminal filament insertion were performed. The animals were randomized into four experimental groups as follows (Tab. 1): In groups sham I, sham II and sham III, animals were sham operated and sacrificed in postoperative hour 24, 48 and 72, respectively. In groups I, II and III, MCAO were performed and sacrificed in postoperative hour 24, 48 and 72, respectively.

Determination of Infarction

After 24, 48 and 72 hours (h), sham and experimental groups were sacrificed and MCA infarction was evaluated by 2,3,5-triphenyltetrazolium chloride (TTC) staining method as described elsewhere (15). If MCA infarction could not be confirmed macroscopically or if the animal died before sacrifice, animals from the MCAO group were excluded from the study.

Surgical Procedures

After terminal anesthetic administration, all blood was obtained by sterile cardiac puncture. Thoracotomy and laparotomy were carried out under aseptic conditions. Then, under sterile technique, samples from lung, liver, spleen, and mesenteric lymph node (MLN), and ileal content were obtained. Special care was taken to ensure that there was no contamination between samples. Lung and ileal tissue samples were also collected and preserved in 10% formalin for histopathologic examination.

Ethics

All surgical procedures were approved by the Animal Ethics Committee of Faculty of Medicine, Zonguldak Karaelmas University, Turkey.

Testing for translocation of Bacteria

BT from the intestinal lumen was defined on the basis of a positive culture of lung, spleen, liver, MLNs and jejunum. A piece of lung, spleen, liver, MLNs and jejunum were removed, respectively. The tissues were weighed and homogenized in tryptic soy broth. These homogenates (100 uL) were plated on blood agar and eosin methylene blue (EMB) agar. For the jejunum, a 1/100 dilution was also prepared and 100 L of this dilution was plated additionally. After 16–18 h incubation period at 37 °C, plates were examined. After the appropriate incubation periods, individual clones were identified and quantified (15–18). Bacterial typing was performed by conventional typing methods (18, 19). Similarity between bacteria detected in tissues and intestinal microflora was shown with phenotypic methods based on identification at species level and antibiotyping (20). For antibiotyping, antimicrobial susceptibility of the strains was investigated by the standardized disk-diffusion method performed on Mueller-Hinton agar, according to Kirby-Bauer and following the criteria of the Clinical Laboratory Standards Institute (21). Plates of Mueller-Hinton agar were inoculated with a bacterial suspension equivalent to a 0.5 McFarland standard and incubated aerobically at 35 °C for 18 h. The diameters of inhibition zones were measured for each antibiotic and the results recorded as resistant or sensitive according to the criteria of CLSI.

Histopathologic Examination

The terminal ileum was removed after sacrifice. The specimens were fixed in 10 % formalin, embedded in paraffin, stained with hemotoxylin and eosin (H&E).

Morphometric analysis of ileal tissue: Villous height, middle villous segment diameter, and crypt depth in H&E-stained slides from all ileum samples were determined by using an image analyzing program (Leica QWINPlus v. 3.1.0). Villous surface area was calculated according to the formula: surface area = αdh (d, diameter; h, villous height). At least ten well-oriented crypt-villous units per ileum sample were measured (9).

Immunohistochemical Examination (ICAM Expression in Ileum)

For immunohistochemical studies, immunostaining was performed according to avidin-biotin-peroxidase complex technique. Paraffin sections were collected on slides, deparaffinized and dehydrated. Endogenous peroxidase activity was blocked using 3 % hydrogen peroxide for 10 min. The sections were incubated with primary antisera including ICAM-1 antibody (Dilution 1:25; Mouse monoclonal Novocastra, Visionbiosystems, UK) for 1 h at room temperature. After washing in phosphate-buffered saline (PBS), the tissues were incubated with biotin conjugated secondary antibody and then streptavidin-biotin-system for 30 min at room temperature. The reactions were visualized by diaminobenzidine tetrahydrochloride. The sections were counterstained using hematoxylin stain, then cleared and mounted.

Microscopic evaluation was performed by one pathologist blinded to the groups. The ICAM-1 expression was scored by immunohistochemical staining in one light microscope area with a magnification of 100x, using the following scoring system. Immunohistochemical staining intensity was scaled from 0 to 4 as: 0, no detectable positive cells; 1, very low density of positive cells; 2, moderate density of positive cells; 3, higher but not maximal density of positive cells; and 4, highest density of positive cells (9).

Tab. 2. BT ratios of the groups.

Groups	Ratios (n)
Group Sham I	0/6
Group Sham II	0/6
Group Sham III	0/6
Group I	5/9
Group II	5/11
Group III	**3/10**

Statistical Analysis

Alveolar distension and collapse index between MCAO and sham groups and the subgroups according to hours (24, 48, and 72 h) were investigated by using likelihood ratio chi-square test. As AEI, ACI, API, villous height, villous diameter, and crypt depth showed normal distribution, a general linear model was used for statistical analysis. While determining differences in ileal villous surface area between and within the groups, factorial analysis of variance with two factors was used followed by Tukey post-hoc test. While comparing groups according to immunohistochemistry, Kruskal-Wallis test was used. Significant differences were determined using Mann-Whitney test with Bonferroni adjustment. P values 0.05 were considered as statistically significant. SPSS (ver. 11.5) program was used for all statistical computations.

Results

The distribution of animals according to groups was as follows: MCAO groups (24 h (n=9), 48 h (n=11), 72 h (n=10)) and sham groups (24 h (n=6), 48 h (n=6), 72 h (n=6)).

Microbiological evaluation in terms of viable bacterial overgrowth revealed that in MCAO groups, BT was present in 34 of the 120 samples (28 %) from 13 of 30 animals (43 %) (Tab. 2). Of these 34 samples, viable bacterial overgrowth was determined in 7 lung (23.3 %), 8 liver (26.6 %), 8 spleen (26.6 %) and 11 MLN (36.6 %) specimens. Conversely, no bacterial overgrowth was detected in sham groups at any time point (Tabs 2 and 3). The most commonly observed isolate was coagulase-negative staphylococci (n=16) (Tab. 4).

Histopathologic examination by hematoxylin and eosin staining showed intestinal mucosal damage and immunohistochemical examination by immunostaining showed significant ICAM expression in the lamina propria of enlarged villi in MCAO groups (Fig. 1).

Tab. 3. Live bacteria translocation in MCAO and sham groups according to organs.

	Group I (n=9)	Group II (n=11)	Group III (n=10)	Group Sham I (n=6)	Group Sham II (n=6)	**Group Sham III (n=6)**
Lung	0/9	4/11	3/10	0/6	0/6	**0/6**
Liver	3/9	3/11	2/10	0/6	0/6	**0/6**
Spleen	4/9	3/11	1/10	0/6	0/6	**0/6**
MLN	5/9	4/11	2/10	0/6	0/6	**0/6**
Blood	1/9	0/11	0/10	0/6	0/6	**0/6**
Total BT	5/9	5/11	3/10	0/6	0/6	0/6

n - number of animals; BT - bacterial translocation

Tab. 4. Translocating microorganisms.

	MLN	Liver	Spleen	Lung	Blood	Total
E.coli	3	-	1	1	-	5
Brevundimonas diminuta	2	2	2	-	-	6
Coagulase-negative staphylococci	4	4	3	4	1	16
Diphtheroid bacillus	2	2	1	1	-	6
Enteroccocci	-	2	1	2	-	5
Total	11	10	8	8	1	38

The values represent the number of positive cultures for different bacterial species in MLN, liver, spleen, and blood. The total in the right column represents the sum of positive cultures of one species; the total in the bottom row represents the sum of all positive cultures for each organ individually. In some of the rats two types of bacteria is overgrowth is seen.

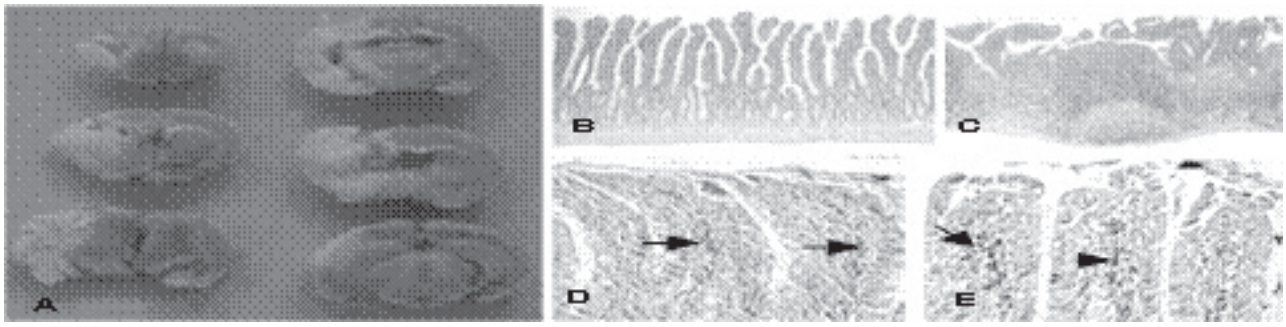

Fig. 1. Gut mucosa damage in stroke but not in Sham animals. Coronal sections of the cerebral infarction area in MCAO group detected by TTC staining method (A). Histopathologic examination of the ileum in sham (B) and MCAO (C) group at 72 h; intestinal mucosal epithelium shows regular villi and few lymphocytes in lamina propria in the sham group (B), while enlargement of Peyer plaques with severe lymphocytic infiltration progressing to submucosa and muscularis propria by eroding muscularis mucosa and prominent distortion of the villi of the ileal mucosa are seen in MCAO group (C). ICAM expression (arrows) was found in only a few cells in the lamina propria of the ileum in the sham group at 72 h (D), while significant ICAM expression was found in the lamina propria of enlarged villi in MCAO group at 72 h (E). Magnification = B and C (H&E; original magnification X 100), D and E (X200).

Morphometric analysis of ileal tissue showed that villous height, middle villous segment diameter, crypt depth and surface area were significantly decreased in the MCAO groups when compared to sham groups ($p<0.01$ for the first three and $p<0.0001$ for villous surface area) (Tab. 5 and Fig. 2A). Moreover, subgroup analysis revealed a statistically significant reduction in villous surface area from the 24th to 72nd h, in both MCAO and sham groups as shown in Figure 2A ($p<0.0001$).

Immunohistochemical analysis demonstrated no statistically significant difference within sham groups ($p>0.05$). Nevertheless, ICAM expression was found to be significantly upregulated in MCAO groups compared with sham groups at 24, 48 and 72 h ($p<0.01$, $p<0.0001$, and $p<0.0001$, respectively) (Fig. 2B).

During the experiment, 4, 2 and 3 rats died in MCAO groups I,II and III, respectively because of anesthesia and surgical complications and were excluded from the analysis.

Discussion

The mechanisms of infections complicating the clinical course of acute stroke have received limited attention which is the root of the problem (8). In the present study, our results showed the occurrence of BT in an animal stroke model. The intestine is the largest immune organ of the body and is a major nidus for inflammation that can spread to the entire body, causing systemic inflammation (9). Although the gut's endogenous microflora has been known to play some role in host homeostasis for years, its profound influence in both healthy and diseased states has recently been increasingly recognized (22–25). The gut is composed of three entities: i) epithelium, ii) mucosal immune system, and iii) commensal bacteria. The epithelium of the gut, a barrier to protect against invasion of potentially harmful antigens, is critical for intestinal integrity (22, 23). Villous height, villous diameter, crypt depth and villous surface area are known to be the specific indices for the evaluation of mucosal damage (9).

In our study, mucosal damage was shown in the MCAO group (Figs 1B and 1C). The distal ileum and more particularly, the villi of the distal ileum appear to be the region of the gastrointestinal tract most vulnerable to injury in stroke. The histologic analysis of intestinal tissues showed that mucosal damage occurred which may be the cause of BT in our stroke model. Villous height, villous diameter, crypt depth and villous surface area were significantly lower in the MCAO group than sham group ($p<0.01$). The loss of barrier function (demonstrated by morphometric analysis of ileum and immunohistochemistry), allowed viable BT. This study demonstrated translocation of live bacteria from the intestine to the MLN, liver, spleen and lung in the

Tab. 5. Histopathological scores of villous height, villous segment diameter, and crypt depth determined using an image analyzing program (mean ± SD).

h	Villous Height			Villous Segment Diameter			Crypt Depth		
	Sham	MCAO	p *	Sham	MCAO	p*	Sham	MCAO	p*
24	274.90±8.78	247.00±3.10	<0,01	60.80±3.01	53.45±1.23	<0,01	70.83±2.45	56.20±1.56	<0,01
48	267.45±4.04	238.70±4.50	<0,01	58.86±1.92	50.60±1.97	<0,01	69.11±2.73	54.09±1.46	<0,01
72	**261.18±2.82**	**233.76±3.27**	**<0,01**	**56.55±0.73**	**47.06±2.69**	**<0,01**	**65.05±1.59**	**51.04±2.27**	**<0,01**

h - hour; * Comparison of Sham and MCAO groups

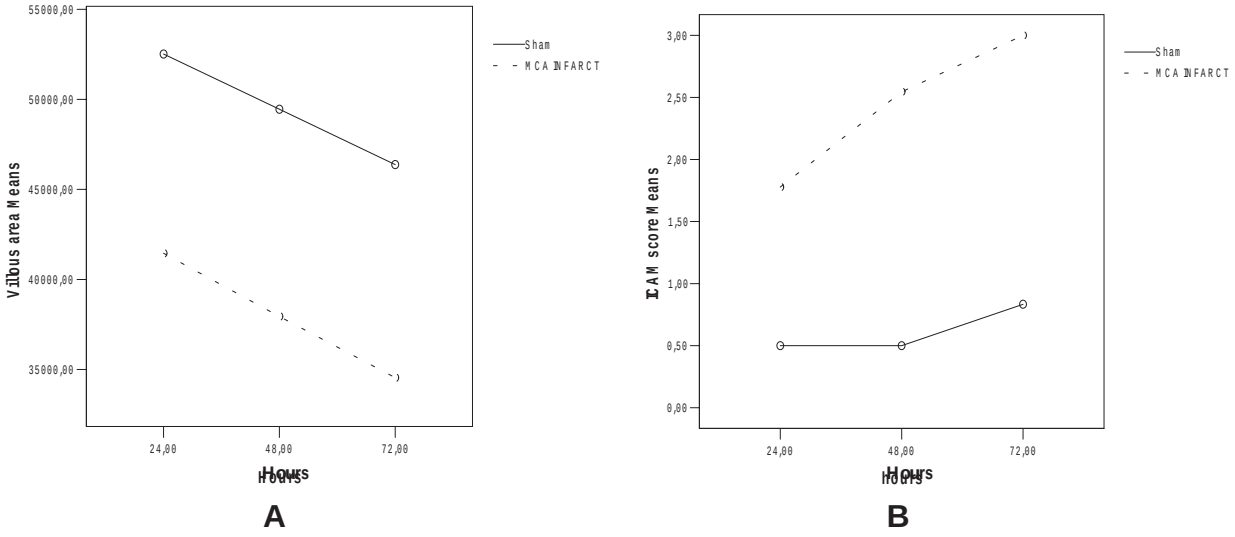

Fig. 2. Temporal course of ileal villous area (A) and ICAM-1 levels (B) in MCAO group as compared with sham group.

MCAO group. By using phenotypic methods based on identification at species level and antibiotyping, it was confirmed that the bacteria detected in the specimens came from the intestinal flora.

During intestinal inflammatory response, release of proinflammatory cytokines and the upregulation of adhesion molecules cause intestinal recruitment of neutrophils and monocytes. These could also lead to gut mucosal injury. ICAM-1, a member of the immunoglobulin super-family of adhesion molecules, is important in leukocyte recruitment during the inflammatory process and could have a profound effect when upregulated after cytokine challenge (9). As was shown in our study, ICAM-1 was significantly higher in MCAO groups than in the sham groups ($p<0.01$) (Figs 1D and 1E). In other words, stroke itself increased the percentage of ICAM-1 cells in the intestine from normal state, which indicates organ injury. This may be the major cause of bacterial translocation.

In some studies, most aspects of stroke-induced immunodepression are the worst on poststroke day 4, and spontaneous development of systemic bacterial infections after stroke occurred within the first three days (6). This was confirmed in our study. Regarding BT, although live bacteria were found to exist in liver, spleen and MLN in the 24 h MCAO group, none was found in the lung. However, in the 48 and 72 h MCAO groups, live bacteria were found in all organs evaluated. Anatomic explanation for BT in the lung is that the MLN flows through the thoracic duct, reaches the systemic circulation through the subclavian vein draining to the superior vena cava, then to the left atrium, and finally to the pulmonary artery and pulmonary vasculature. Thus, the lungs receive the lymph drainage from the gut (11). It has been claimed that BT may represent a significant source of organ abscess, pneumonia and sepsis in the critically ill patient via intestinally derived bacteria/endotoxin (24–27).

In conclusion, our results showed that stroke itself, played an important role in the development of poststroke gut barrier damage and BT. To prevent poststroke infection, it would be wiser to inhibit BT or prevent reasons leading to gut mucosal damage. As a consequence, we propose that when treating patients with poststroke infection in the future, intestinal pathogens should be considered, and a kind of selective decontamination of the digestive tract that also eradicates intestinal pathogens should be used.

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