

## **GASTROINTESTINAL DISORDERS IN PATIENTS WITH ACUTE DISTURBANCE OF CEREBRAL BLOOD CIRCULATION**

**V. Drenski, D. Minchev**

*Department of Neurology, Medical University, Varna*

*The authors performed parallel investigations between the nature of gastrointestinal complications and the nature and localization of vascular accidents in 40 patients hospitalized in the Division of Intensive Care of the Clinic of Neurology, Medical University, Varna, with a proved diagnosis of acute disturbance of cerebral blood circulation. The pathophysiological mechanisms leading to these complications were discussed. The data obtained were of practical value with a view to prevention and management of these complications in cases with acute cerebral circulatory disorders.*

**Key-words:** Stroke, brain haemorrhage, brain ischemia, acute gastrointestinal mucosa lesions, peptic ulcer, diagnosis

### **INTRODUCTION**

Sometimes, in progress of stroke and particularly in its acute stage, along with the most common pulmonary and cardiac complications different gastrointestinal disorders involving esophagus, stomach and intestines designated as "acute mucosal lesions" can be observed (3, 17, 21). They are manifested in the form of hyperemia, erosion or even acute ulcer of the stomach mucosa as well as of ulcerous or pseudomembranous enteritis (12, 18, 19). Gastrointestinal complications may appear even in the early phase or in the first weeks

of stroke. Acute necrotic mucosal lesions of the stomach and more rarely of the duodenum and jejunum in the form of erosions or ulcerations very often cause massive haemorrhages aggravating stroke development.

Our aim was to analyze the characteristics of gastrointestinal disturbances in patients with stroke of different localization.

### **MATERIAL AND METHODS**

The object of our investigations were the clinical manifestations and autopsy results in 40 patients (30 men and 10 women) with acute disturbance of cerebral blood circulation treated in the Division of Intensive Care, Department of Neurology, Medical University of Varna, during a 3-

**Table 1**  
**Patients' distribution according to sex, age, localization and type of stroke**

Type of stroke	Patients n	Localization			Sex		Age range
		LMCA	RMCA	VBS	male	female	
Ischaemic	26	17	8	1	20	6	52 - 94
Haemorrhagic	14	9	3	2	10	4	25 - 64

Legend: LMCA - left medial cerebral artery; RMCA - right medial cerebral artery;  
 VBS - vertebro-basilar system

year period (from 1987 till 1989). Brain infarction was diagnosed in 26 cases aged between 52 and 94 years but cerebral haemorrhage was found out in the rest 14 cases aged between 25 and 64 years. The gastrointestinal pathology in these patients was analyzed according to the type of mucosal lesions in single organs.

## RESULTS

Patients' distribution according to stroke localization was demonstrated on

Table 1. The frequency distribution of pathologically proved gastrointestinal disorders was presented on Table 2.

Anamnestic data and x-ray proofs of stomach ulcer ante mortem were available in 2 patients only. Duodenal ulcer was preliminarily proved in 4 cases only. No symptoms of peptic ulceration were reported by the rest patients prior to stroke accident. The adequate information was collected from the hospital documentation service, personal ambulatory card in the polyclinic as well as from patient's family.

**Table 2**  
**Localization and frequency of gastrointestinal lesions in stroke patients**

Localization	Type of mucosal lesion			
	hyperemia	erosion	acute bleeding ulcer	chronic exacerbated ulcer
oesophagus	1	1	-	-
stomach	-	9	7	4
stomach and duodenum	-	3	-	-
duodenum	-	6	12	5
small intestine	6	7	-	-
stomach and small intestine	-	4	-	4
<b>Total</b>	<b>7</b>	<b>30</b>	<b>19</b>	<b>9</b>

Prior to death, duodenal ulcer was proved in 2 cases and new bleeding ulcers were pathologo-anatomically established. At autopsy, hyperaemia, multiple erosions and multiple bleeding ulcers of stomach and duodenum were observed in 3 patients with haemorrhagic and in 2 patients with ischemic stroke. No corticosteroid therapy was administered before or in the development of stroke at all. Three patients with recurrent stroke only were permanently given aspirin in a low dose as an antiaggregant whilst all the rest patients were not administered any anticoagulant drugs.

It stressed that melena was ante mortem diagnosed in two patients but haematemesis in another one patient only. Gastrointestinal bleeding was established at autopsy in the rest 37 cases. Gastrointestinal complications were clinically diagnosed only in two patients with ischemic stroke: the first had chronic exacerbated pyloric ulcer with perforation and haemorrhage in the stomach, intestine and abdominal cavity resulting in secondary anaemia and surgical shock but the second had bleeding duodenal ulcer. Two days prior to death, sudden circulatory collapse presented by pale skin, aggravating neurological symptoms and worsening of the common status was observed. As in most cases there were no clinical data of haemorrhage, melena, haematemesis or sudden collapse the hypostatic bronchopneumonia, pulmonary thromboembolism, acute heart and respiratory insufficiency, etc. could be considered a direct reason for death. Aphasia was diagnosed in 7 patients who were not able to inform eventually the physician about their complaints.

The time interval between the onset of haemorrhagic stroke and gastrointestinal bleeding was 1-10 days long but that between the ischemic stroke and gastrointestinal haemorrhage was 3-21 days long (mean duration of 5,1 and 8,2 days, respectively).

We failed to establish any statistically significant correlation between the gastrointestinal complications and the localization of brain lesion in haemorrhagic and ischemic stroke. It was noteworthy that in 6 out of 8 cases with a focal lesion in the diencephalon (in the thalamus and hypothalamus) as well as in the cases with lesions in the brain stem a large gastrointestinal haemorrhage was observed in the course of stroke. No significant correlation between the distribution of ulceration types and the type of stroke and brain damage localization as well could be found out.

## DISCUSSION

The acute lesions of the gastrointestinal tract in stroke patients are secondary findings due to different etiological factors. Most commonly, both stomach and duodenum but, more seldom, oesophagus are affected. Clinically, these lesions are designated as erosive or haemorrhagic gastritis, acute peptic ulcer, Curling's ulcer, Cushing's ulcer, or Mallory-Weiss' syndrome (3, 17). Some authors report cases with pseudomembranous enteritis as well as with ulcerations and erosions of intestines and colon (10, 12, 18, 19).

The pathogenesis of stress ulcers remains unclear. Certain authors explain them with circulatory disturbances in the gastrointestinal tract wall (6) while others

relate them to enhanced digestion capacity of gastric acid (9). Recently, such ulcers were associated with the sudden disorder of the metabolic processes due to deficit of macroergic compounds in the gastrointestinal mucosal cells resulting in necrosis of these cells. The neurogenic ulceration in cases with head injuries, brain tumours, sepsis or as a consequence of neurosurgical interventions present a well-known phenomenon (1, 4 - 6, 8, 15, 16). Most likely, during the first hours and days after stroke the severe brain oedema stimulates the hypothalamo-hypophyseal-adrenal system manifested by elevated glucocorticosteroid production. The glucocorticosteroids enhance the gastric acid secretion and thus damage the mucous barrier that may cause acute erosions and ulcerations of stomach and duodenal mucosa (14). According to literature data available, gastric erosions are the most common and dangerous lesions of the gastrointestinal tract which provoke gastric ulcer (13). Dalggaard (7) accepts that acute peptic ulcer can occur even in the first 12 hours after brain accident and, most commonly, several days after stroke. According to French and Porter

(15), gastrointestinal haemorrhage occurs on the 8<sup>th</sup> day after brain accident but according to Dalggaard (7) - up to 12 days after it.

Our results are in agreement with these of numerous authors (7, 11, 15). We establish a larger number of males with gastrointestinal complications similarly to other authors' data (2, 7, 15, 20). However, like many investigators (7, 11, etc.) we fail to detect any regulatory dependence between the cerebral lesion topics and the nature of gastrointestinal damage.

## CONCLUSION

Getting acquainted with the problem of gastrointestinal lesions in stroke patients is rather important in the neurological practice. Being familiar with the fact that the gastrointestinal bleeding can occur even in the first days after the cerebral stroke can help the organization of an appropriate preventive and therapeutic behaviour with this patients' contingent. We must have in mind the considerable number of cases without any symptoms of gastrointestinal mucosa damage detected at autopsy only.

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## **Gastrointestinale Schädigungen bei Kranken mit akuter Störung des Gehirnbloodkreislaufes**

**V. Drenski, D. Mintshev**

*Lehrstuhl für Neurologie, Medizinische Universität Varna*

**Zusammenfassung:** Die Autoren führten parallele Untersuchungen über den Charakter der gastrointestinalen Komplikationen und den Charakter und die Lokalisation der vaskulären Schädigungen bei 40 Kranken in der Intensivabteilung der Neurologischen Klinik der Medizinischen Universität Varna mit nachgewiesener Diagnose einer akuten Störung des Gehirnbloodkreislaufes durch. Die pathophysiologischen Mechanismen, die zu diesen Komplikationen führten, wurden besprochen. Die erhaltenen Angaben hatten einen praktischen Wert für die Vorbeugung und Behandlung dieser Komplikationen bei den Fällen mit akuten Störungen des Gehirnbloodkreislaufes.

## **Troubles gastro-intestinales chez des patients avec des désordres aigues de la circulation vasculaire cérébrale**

**V. Drenski, D. Mintchev**

*Chaire de neurologie, Université de médecine à Varna*

**Résumé:** Les auteurs ont effectué des recherches parallèles d'un côté sur le caractère des complications gastro-intestinales et d'autre sur le caractère et la localisation des troubles vasculaires chez 40 patients, qui ont été hospitalisés dans le secteur intensif de la Clinique de Neurologie, Université de médecine à Varna, avec une diagnose prouvée de troubles aigus de la circulation cérébrale. On discute les mécanismes pathophysiologiques, qui ont amené à ces complications. Les données reçues ont une valeur pratique en vue de la prophylaxie et le traitement de ces complications des troubles aigus de la circulation vasculaire cérébrale.