POST-GASTRECTOMY SYNDROMES

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Abstract: *Nowadays, there are a significant number of patients who have in their personal history a gastrectomy, a very common procedure in the past decades in the treatment of peptic ulcer. In addition, there is also another group of patients who are long-term survivors after gastrectomy made for gastric cancer. The awareness regarding the late complications after gastric resections must be raised not only for surgeons but also for gastroenterologists and general practitioners. This article describes the post-gastrectomy syndromes elaborating on the symptoms, the pathophysiological mechanisms involved and the possible treatment.*

Cuvinte cheie: *sindroamele postgastrectomie, gastrectomie, rezecţie gastrică, complicaţii tardive*

Rezumat: *În prezent există un număr mare de pacienţi ce au în antecedentele chirurgicale o gastrectomie, intervenţie chirurgicală practicată pe scală largă în decadele trecute pentru boala ulceroasă. De asemenea, acestora li se adaugă şi un grup de pacienţi care supravieţuiesc pe termen lung după o gastrectomie efectuată pentru neoplasm gastric. Cunoaşterea complicaţiilor tardive după rezecţiile gastrice, grupate sub denumirea de sindroame postgastrectomie este importantă atât pentru chirurgi dar şi pentru gastroenterologi sau medicii de familie. Acest articol prezintă sindroamele post-gastrectomie detaliind simptomatologia, mecanismele fiziopatologice implicate şi tratamentul posibil.*

Under the name of post-gastrectomy syndromes we classify a group of symptoms which occur after gastric resection and are caused by the complex anatomical and physiological changes induced by gastrectomy. The stomach has a multitude of functions which are lost after a total or partial resection. The abnormal transit, the disturbed metabolism and the digestion and absorption deficiencies are the most common problems.

CLASSIFICATION, SYMPTOMATOLOGY, PATHOPHYSIOLOGICAL MECHANISMS AND TREATMENT

1. ABNORMAL TRANSIT. The most common postoperative problems are related to the abnormal transit in the upper gastrointestinal tract – the transit can be either accelerated or delayed or it can occur in the wrong direction.

1.1. The dumping syndrome is the result of the gastric incontinence which appears after the distal resection, the term being used for the first time in 1922 by Mix.(1) The dumping can be early or late. **The early dumping** is defined as a complex of gastrointestinal and cardiovascular symptoms which are triggered 30 minutes after the meal, especially after the ingestion of food with a high amount of carbohydrates and fats. The symptoms include nausea, bloating, diarrhoea, vomiting, abdominal pain, excessive sweating, tachycardia and hypotension. Two different pathophysiological mechanisms are involved in the early dumping syndrome: the sudden release of the hypertonic content of the stomach in the jejunum causing the reduction of the plasmatic volume with approximately 400-800 ml resulting in tachycardia and hypotension and the release of a variety of intestinal hormones (neurotensin, serotonin, bradikinin, intestinal vasoactive peptide) who trigger the digestive symptoms.(2,3) **Late dumping** is a syndrome which appears 1-3 hours after the meal and it is characterized by the symptomatology of hypoglycaemia with perspiration, dizziness, hunger. In comparison with the early dumping, it lacks the cardiovascular symptoms and the diarrhoea. The rapid absorption of the glucose in the proximal jejunum causes not only a hyperglycaemia but also an excess secretion of the glucagon-like hormone GLP-1 in the L-cells of the digestive tract. These two factors cause a disproportionate release of insulin which finally leads to hypoglycaemia.(4) The incidence and the severity of the dumping syndrome is higher after the Billroth II reconstruction as compared with the Billroth I. The recent publications show that depending on the type of reconstruction, the incidence of the dumping syndrome ranges between 1.1 and 9.2%. The treatment of the dumping syndrome consists of dietary measures: dividing the daily intake into 6-8 meals of smaller quantity, avoiding liquids intake during meals, avoid simple carbohydrates as well as the administration of drugs which inhibit the gastric emptying. In severe, refractory cases the treatment with somatostatin analogues like Octreotid is indicated.(5) Surgery is reserved for severe cases showing no or low response to the conservative treatment and the therapeutic choices include the narrowing of the gastrojejunostomy, the reintegration of the duodenum in the digestive passage (when this option is technically possible) or the interposition of a reversed jejunal segment between the stomach and the duodenum.

1.2. Reflux esophagitis has an incidence of about 80% after total gastrectomies and a bit lower after distal gastrectomies (20-50%). The reconstruction using jejunal pouches and the Roux-Y loop is associated with a significantly lower incidence of the esophagitis.(6). The pathophysiological mechanism is the reflux of the duodenal content in the distal esophagus, the bile acids and the lyzolecithine causing the inflammation of the mucosa.(7) The conservative treatment includes bile acids binding resins and sucralfate. In the refractory cases, the

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degastro-gastrectomy and reconstruction with a Roux-Y loop is indicated.

1.3. Atrophic and reflux gastritis is another possible complication following gastrectomy, the pathophysiological mechanism in this case being similar to the reflux esophagitis, the reflux of the enteral content in the remnant stomach. Approximately 10 years after a gastric resection, 80-90% of the patients develop an atrophic gastritis but the great majority of these patients remain asymptomatic.(8) Only about 10% of the gastrectomised patients develop significant symptoms like epigastric pain, bloating and bile vomiting, which require treatment.(9) Characteristic is the immediate relief of the symptoms after vomiting and their exacerbation after the stimulation of biliary and pancreatic secretion. In the cases with severe symptomatology caused by the reflux gastritis, the surgical treatment consists of the interposition of an isoperistaltic jejunal loop or the conversion in a Roux-Y gastrojejunostomy.(9,10)

1.4. Afferent loop syndrome occurs due to the alimentary passage in the afferent loop or the disturbed outflow of the biliary and pancreatic secretion into the small bowel. This complication is generally caused by technical issues (overlong loop, asymmetric or too narrow anastomosis). The symptoms include postprandial dull or colicky pain which culminate with explosive biliary or alimentary vomiting (and a relief of the pain) and malnutrition. The treatment is in most of the cases surgical and it consists in the reconstruction using the Roux-Y loop or, in the cases where the local dissection is technically difficult, the Braun jejunojejunostomy can be used.(11)

1.5. Gastric stasis. The delayed gastric emptying associated with gastric stasis which interferes with the food intake is rare after the gastrectomy. Still, food retention in the gastric remnant is frequently seen in the postoperative endoscopy after the distal gastrectomy (in over 20% of the patients, 2 years after the gastrectomy).(12) This incidence was higher after the Billroth I reconstruction compared to the Billroth II or Roux-Y.(13) Despite these endoscopic findings, none of these patients had a gastric emptying which was delayed significantly at a clinical level and there was no association between the gastric stasis and the symptomatology or the weight loss.

1.6. Diarrhoea is a possible complication after the gastric resection but it is caused and hence appears only in the cases where a vagotomy is associated. The incidence of the postvagotomy diarrhoea is about 15% after a selective vagotomy with antrectomy and between 3 and 11 % after a highly selective vagotomy.(14,15)

1.7. Roux syndrome or the Roux loop syndrome is characterized by abdominal pain, nausea and vomiting and postprandial bloating. The incidence of this complication is different according to the different existing articles, going up to 30% in some studies, while others attribute no clinical significance to this syndrome.(16) The pathophysiology of this syndrome is not fully understood. It is known that the small bowel has a pacemaker activity which generates cyclical electrical potential with the highest frequency in the duodenum. These potentials induce a muscle contraction that drives the bowel content towards the terminal ileum. The transmission of these potentials is interrupted once the jejunum is transected causing the appearance of new ectopic pacemakers which generate retrograde potential towards the stomach.(17)

2. ABNORMAL NUTRITION – WEIGHT LOSS. Almost all patients who undergo a gastrectomy show a weight loss in the first 6 months after the surgery. On average, this loss is about 10% of their initial weight. In many patients, there is an insufficient nutritive intake. The causes are the lack of appetite and the symptoms caused by the abnormal transit. The meals are in too smaller portions and usually interrupted once the symptoms occur and also their composition is altered in order to cope with the dumping syndrome.(18) The malnutrition which requires treatment is mainly observed after a Billroth II gastrectomy. A study published in 1982 proves that the patients who underwent a Billroth II gastrectomy had at the age of 75 in average 11 kg less bodyweight compared to a similar group of healthy, non-operated patients.(19) Most of nutrition disturbances are prevalent in the first month after the surgery (when also the symptoms associated with the abnormal transit are more severe) and have a tendency to improve over time, the weight of the patients usually stabilizing at about 6 months after gastrectomy. The reconstruction using a jejunal pouch has shown a benefic effect regarding this evolution.(20)

3. ABNORMAL DIGESTION/ABSORPTION. The maldigestion and the malabsorption are frequent complications after the gastrectomy but these complications are usually mild and rarely cause clinical problems. The pathophysiological mechanisms causing the syndromes are intricate. The most important mechanism is the asynchrony in the activation of the pancreatic enzymes due to the lack of timely mixing with the gastric secretion and the ingested aliments.(21) This asynchrony contributes to the deficit in the absorption of fats. In addition, due to the excessive colonization with bacteria caused by reduced gastric acid output, a rapid deconjugation of the bile acids occurs, which also contributes to the maldigestion of fats. As a result, most of the fat-soluble vitamins (A, D, E and K) are affected by this maldigestion. The iron metabolism is also disturbed by the modifications present after a gastrectomy, as most of the ingested iron is absorbed in the duodenum which is excluded from the digestive passage after the Billroth II and Roux-Y reconstructions. Also, in order to be absorbed, the iron must be reduced in the acid environment of the stomach, the lack of this environment further causing a low absorption of iron. The B12 vitamin requires the presence of the intrinsic factor produced in the proximal stomach in order to be absorbed in the small bowel.

4. METABOLIC CHANGES

4.1. Anaemia. Almost 50% of the patients having a history of gastric resection have a certain degree of anaemia 20 years after the surgery.(22) The cause of this anaemia is a combination between the deficit of iron, B12 vitamin and folic acid. The postgastrectomy anaemia as a cause of the mentioned deficits is easily treatable with enteral and parenteral substitution.

4.2. Osteopathy. The appearance of osteopathy after a gastric resection is multifactorially conditioned. The reduction in the absorption of the D vitamin due to lipid malabsorption and the exclusion of the duodenum which is the main area where the calcium absorption occurs, are some of the causes.(23) The alterations in the bone structure like osteoporosis and osteomalacia have been described in approximately 40% of the patients.(24)

5. OTHER COMPLICATIONS

5.1. Cholelitiasis. The increase in the risk of the development of gallstones is due to the lack of vagal innervation of the gallbladder and decreased motility after the radical gastrectomies, but is also the consequence of the low release of cholecystokinin from the duodenum after the bypass procedures like the Roux-Y gastrectomy. Wu et al. have studied the incidence of cholelitiasis on a number of 463 patients who underwent a gastrectomy. Gallstones were observed in 85 of 281 patients with radical gastrectomies and in 9 of 182 patients with simple gastrectomies. Based on these results, the authors recommend the prophylactic cholecystectomy in the patients who undergo a radical gastrectomy.(25)

5.2. Recurrent/anastomotic ulcer. The appearance of a

recurrent or anastomotic ulcer after the gastric resection for peptic ulcer is most often the result of an incomplete vagotomy or a treatment with nonsteroidal anti-inflammatory drugs.(26) This type of ulcer is usually located in the peripyloric, duodenal or perianastomotic areas and the role of Helicobacter pylori in its etiology is less clear as in the primary ulcer.(27) The treatment consists of proton pump inhibitors and in the cases of incomplete vagotomy, the transthoracic truncal vagotomy is required.(28)

5.3. Gastric remnant cancer. The development of an intestinal metaplasia over a gastric atrophy in the mucosa of the gastric remnant is considered a precancerous lesion. The incidence of the gastric remnant carcinoma has suffered a dramatic increase over the last decades. 15 years after a gastric resection, the incidence of the remnant cancer is of approximately 3%.(29) The recommended treatment for this condition is the degastro-total gastrectomy with regional lymphadenectomy.(30)

CONCLUSION. The postgastrectomy syndromes are still a current topic in the gastric surgery as they are one of the main factors contributing to a low quality of life for that percent of the affected patients. The knowledge of the pathophysiological mechanisms involved allows an adequate etiopahtogenic treatment, which in many cases is surgical.

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