7.13.4 Pathophysiological principles of ileus prevention

Intestinal obstruction is curable until ileus develops. Several positive feedbacks which progressively intensify both intestinal and overall impairments, disable the successful therapy of ileus. The therapy, or prevention of intestinal obstruction include therefore the measures disabling ileus to develop. Preventive antiileous algorithms are based on the pathogenesis of ileus and are aimed at the procurement of luminal ventilation, removal of digestive juices from the intestinal lumen, and the procurement of sufficient intestinal perfusion by oxygenated blood. In a majority of cases, the **mechanical intestinal obstruction** requires surgical therapy (assessment of the localisation and cause of obstruction, resection of the necrotic intestine). The significance of the preventive measures resides in the fact that an entirely resuscitated patient is able to tolerate better the state of anaesthesia and surgical intervention per se.

In some cases, especially if partial obstruction of the small intestine is involved, the intestinal transport can be renewed solely by means of conservative therapy. **Intestinal pseudoobstruction** often requires a long-term conservative therapy lasting even for several weeks, and its supplementation by parenteral nutrition. An exclusively conservative therapy of intestinal obstruction, however, increases the risk of the development of intestinal strangulation. Therefore it should be applied solely in cases when the risk of intestinal strangulation is low. Inversely, if the assumed cause of complete obstruction resides in intususception, volvulus or incarcerated hernia, it will be necessary to operate immediately, with no regard to the clinical state.

Non-standard, although frequent is the use of various antimicrobic interventions. Administration of wide-spectrum antibiotics can restrict the growth of intestinal bacteria. An analogue of somatostatin – octreotide is administered with the aim to restrict the secretion of intestinal mediators of inflammatory reaction induced by bacteria, or endotoxins.

A **primary significance of prevention** is ascribed to those techniques of abdominal operations which reduce the risk of the development of adhesions. A pronounced protective effect resides in the preservation of the omentum, inhibition of both contamination and the drying out of the peritoneum, and avoidance of extensive peritoneal ligatures.

Alterations within the internal environment evoked by dehydration or circulatory shock quickly succumb to changes; therefore it is necessary to monitor continuously the serum electrolytes, acid-base balance and haemogram. Azotaemia and a pronounced increase in haematocrit give evidence of a severe decrease in intravascular volume. Metabolic acidosis and leukocytosis are frequently present in coincidence with intestinal strangulation and advanced ileus. No biochemical parameters can be however used for unambiquous assessment of the diagnosis of intestinal strangulation.

OVERVIEW OF THE ILEUS PREVENTION

A. Procurement of luminal ventilation
   1. Gastrointestinal aspiration
   2. Intraluminal insufflation of oxygen

B. Removal of digestive juices
   1. Gastrointestinal aspiration
   2. Fasting prior to operation
   3. Parenteral nutrition
   4. Fasting after operation until the reappearance of intestinal motility

C. Procurement of intestinal perfusion by oxygenated blood
   1. Infusion therapy – maintenace of the volume and capacity of blood to transport oxygen. Correction of the deficit in fluids and electrolytes
   2. Optimalisation of the cardiopulmonal and renal systems
   3. Anaesthesia without hypoxaemia
   4. Spinal anaesthesia

7.14 Inflammation and other intestinal diseases

Inflammatory bowel diseases represent two clinically distinct diseases, namely ulcerative colitis and

Crohn's disease. They are severe chronic diseases of unknown etiology. Both these diseases will be analysed mutually in order to emphasise their distinctions.

The cause of inflammatory bowel diseases is not known. Etiological considerations can involve several factors. The incidence of **ulcerative colitis** yields familial occurrence in 20 % of cases, and the familial incidence of **Crohn's disease** takes place in 40 % of cases. Genetic impact may determine the type of immune reaction. Genetic predisposition is proved by the occurrence of inflammatory bowel diseases in monozygotic twins. Until now, no evidence has been acquired proving the infectious etiology of inflammatory bowel diseases. The theory that an immune mechanism may be involved is based on the presence of antibodies against the colonic epithelial cells, histological changes, and the recovery of the clinical state after the immunosuppressive therapy. It is believed that the cause of inflammation resides in epithelial immunoregulatory defects. Intestinal mucosa contains an increased amount of T helper cells and a decreased amount of T supressor cells. Psychological factors are assumedly applied albeit incurring deterioration of the clinical state by assisting the maintainance of the disease. Various external factors, such as toxins that are present in the living and working environment, can participate as the triggering factors in the development of the disease.

In ulcerative colitis and Crohn's disease **the inflammatory process is involved having many common signs**, however it differs in its clinical manifestation and histological findings.

7.14.1 Ulcerative colitis

The **acute stage** of ulcerative colitis **is characterized by a diffuse superficial inflammation** of the colonic mucosa and submucosa. In addition to the large intestine, the rectum is usually involved (95 % of cases). Inflammatory reaction may involve the entire large intestine, as well as the terminal ileum. Inflammatory foci are infiltrated by neutrophils. In a majority of cases, the mucosa is afflicted by a diffuse inflammation. In addition to the latter, the superficial defects are present in the mucosa. Sometimes more profound linear ulcerations develop. The surrounding of crypts is covered by multiple microabscesses. Intestine with noticeable inflammatory changes is significantly distended (more than 6 cm in diameter). The intestinal distension brings about attenuation of its wall. In this manner the picture of toxic megacolon develops. In such cases danger implies from intestinal perforation.

If ulcerative colitis lasts for a long period, hyperplasia muscularis mucosae develops. It is a result of long lasting inflammation and processes of reparation. Finally, the large intestine alters to such an extent that the typical haustra disappear. Regenerating islands of mucosa surrounded by areas of ulcerations can be so pronounced that the condition leads to the formation of pseudopolyps lacking the signs of a neoplastic process. If, however, the duration of the inflammatory process is prolonged, dysplastic changes with signs of malignity may take place in epithelial cells. However, carcinoma rarely appears in coincidence with ulcerative colitis. More frequent are the strictures or fistulae.

The acute ulcerative colitis **is manifestant by diarrhea, rectal bleeding, fever, weight loss and abdominal pain**. These signs are considered to represent the cardinal symptoms. However, the clinical manifestation of the disease can be modest residing only in an increased frequency of stool per day, especially when localised in the rectum (ulcerative proctitis).

The state of a patient principally depends on the extent of diarrhea. Ulcerations, extreme hypokalaemia, anaemia, leukocytosis and fever can develop. The signs of toxicity appear – weakness, anorexia, tachycardia and hypotension. The abdomen can be diffusedly distended, although demarkated resistances are not present. In approximately 10–15 % of patients the disease begins very abruptly and requires immediate hospitalisation. The patients are at risk to develop severe complications such as megacolon, shock, hypokaliaemia, hypoalbuminaemia, or perforation of the colon.

7.14.2 Crohn's disease

Crohn's disease is sometimes referred to as regional enteritis. The **inflammatory process involves all intestinal layers including the serous surface**. The inflammatory process is often discontinuous; severely involved segments of bowel are separated from each other by areas of apparently normal bowel. In approximately 90 % of Crohn's disease, the rectum may be spared. In addition to the intestine, there may be diffuse ulceration of the other parts of GIT (oral cavity, oesophagus, stomach and duodenum). Ap-

proximately one third of the patients is constituted by those with the involvement of the ileum, one third with involvement of the large intestine and one third of patients is afflicted by lesions in the ileum and large intestine. Inflammation of the intestinal serous surface incurs the development of adhesions between intestinal loops, or adhesions of intestines with other organs. Strictures and fistulae are frequently present, foremostly in the perirectal area. Various pathological structures are detectable by palpation. The macroscopical appearance of the mucosa may be normal. Profound linear ulcerations are present along the longitudinal axis of the intestine. In addition to neutrophils, the inflammatory infiltration contains also lymphocytes and macrophages. Granulomas develop in 50 % of cases. Fibrosis and formation of stenoses occur in conseqence of an excessive production of collagen. Epithelial dysplasia is not observed.

The **clinical manifestation** in Crohn's disease greatly resembles that of ulcerative colitis but pathological changes are sparing rectum. Only in 10 % of cases are inflammation in rectum and even more often as pararectal – perianal fistulae. According to ileum involvement (site of B_{12} vitamin absorption) macrocytic anaemia can occur and as well protein malabsorption. However, all symptoms are much more moderate. With involvement of ileum, the symptoms reflecting variable degrees of intestinal stenosis may occur. A colicky pain sometimes arises following the food intake. Less frequently, the right lower quadrant can display a palpably detectable resistance. Marked rectal bleeding is rare, the occult bleeding however, is frequent. The beginning of Crohn's disease in young people can simulate acute appendicitis. With involvement of the large intestine, diarrhea is present together with perirectal abscesses, fistulae and fissures. In such case (large intestine involvement), the manifestation includes extraintestinal symptoms. Toxic megacolon can even develop. In contrast to ulcerative colitis, the patients with Crohn's disease more frequently develop strictures and fistulae, and adversely, perforation and haemorrhage develop less frequently.

7.14.3 Ischaemic colitis

In elderly people the ischaemic colitis can simulate an inflammatory intestinal disease. **The cause of ischaemic colitis resides in extensive atherosclerosis**. Ischaemic colitis is less frequently brought about by other vascular disorders, surgical interventions, dissecting aortic aneurysm, marked decrease in the heart output, vasculitis, or hypercoagulation. Ischaemia is mostly marked in the interface between the chief inflowing arteries – in the spleenic flexure and rectosigmoid area (flexura lienalis, area rectosigmoidea). Ischaemic colitis is a consequence of hypoperfusion, however, not in coincidence with a complete obstruction. The rectum is usually spared because of rich collateral blood flow. The clinical course of disease may be acute or chronic with all transitory stages between those two extreme conditions. **Acute ischaemic colitis** usually begins very abruptly with abdominal pains and bloody stools. Fever may be present, as well as hypotension, tachycardia and symptoms of peritoneal irritation. It is very difficult to differentiate the acute stage of ischaemic colitis from ulcerative colitis. Angiographic, or X-ray examinations with contrast enema would represent an unreasonable hazard. Surgical intervention may be required when signs of perforation are present.

Subacute and subchronic colitis begin inconspicuously. Patients develop abdominal pain and diarrhea. Anxiety arises in coincidence with food intake resulting in evident weight loss. Chronic states result in the narrowing of some segments of the large intestine.

7.14.4 Diverticulitis

Diverticula in the large intestine occur especially in the aged. Diverticula are, in fact, pouches in form of small sacks which maintain all components of the intestinal wall. **Diverticula develop most frequently in the site of the entrance of arteriole into the intestinal wall**. The intestinal wall in these sites is weakened. The development of diverticula is enhanced by several factors, foremostly by a chronically increased intraluminal pressure frequently in coincidence with a diet deficient in fiber or roughage. Stool in such cases is low in volume. The majority of diverticula develop in the sigmoid colon. They are usually asymptomatic. Diverticula may, however, be the cause of infection. The vessels surrounding the diverticula can rupture easily thus causing bleeding, in most cases moderate.

Diverticulitis is a state, in which **infection develops inside and around the diverticula**. Such a state can result in diverticular micro- or macro-perforation. The inflammation is incurred by colonic bacteria.

Bacterial overgrowth in chronic states can lead to B_{12} vitamin consumption as it is in all blind loops and could be a reason of macrocytic anaemia development. Gradually, microabscesses can develop. Microabscesses are inclined to perforate spontaneously, thus causing localised, or less frequent generalised peritonitis. If the perforation results in peritonitis, there is a risk of the development of a large abscess and its penetration into adjacent organs. Acute diverticulitis can be clinically manifestant as appendicitis localised to the left. The symptoms frequently appear during defecation. In addition to the intestinal symptoms, the overall clinical picture includes fever, leukocytosis, and palpable resistance. Bleeding is not massive. Repeated attacks of diverticulitis result in the development of fistulae.

7.14.5 Extraintestinal manifestation of inflammatory intestinal diseases

Extraintestinal manifestation of ulcerative colitis and Crohn's disease greatly resemble each other. Nutritional deficiency appears in consequence of anorexia, fever, blood loss and malabsorption. This state results in an abrupt **weight loss** which at a young age brings about developmental retardation. The inflammatory bowel disease can be associated with two forms of athritis, often referred to as enteropathic arthritis. The first form is so-called **non-deformating acute arthritis** of big joints. The second form is sacroiliitis and ankylosing spondylitis which appear in patients with HLA B27. The articular affliction can precede the clinical manifestation of intestinal inflammation even months or years prior to the latter. The characteristic symptoms include mono or oligoarthritis and asymmetrical synovitis of the knee or ankle joints. Sacroiliitis and ankylosing spondylitis persist also in cases where resection was performed due to Crohn's disease.

Extraintestinal manifestation of inflammatory intestinal diseases can include also hepatobiliary changes. Many patients have moderate **pericholangitis** which is manifestant by an increase in alkaline phosphatase. The occurrence of extra and intrahepatic sclerotising cholangiitis manifestant by obstructive jaundice, is less frequent. Slower progression brings about cirrhosis. Sclerotising cholangiitis is more often associated with ulcerative colitis than with the Crohn's disease. Patients with Crohn's disease more frequently develop cholelithiasis. Cholelithiasis is especially enhanced in cases where ileum is involved, namely in consequence of decreased reabsorption of bile acids.

The symptoms of inflammatory intestinal disease more frequently include **episcleritis, iritis and uveitis**. Approximately 5 % of patients develop erythema nodosum. Renal disorders are observable as urolithiasis with the formation of calcium-oxalate stones. Further symptoms include obstructive uropathy, kaliopenic nephropathy and amyloidosis. The patients with inflammatory intestinal diseases are inclined to the development of thrombophlebitis. Inflammatory intestinal diseases can be complicated by osteoporosis and osteomalacia.

Extraintestinal manifestation can, in special cases, precede the development of intestinal symptoms. It is not simple to assess the correct diagnosis on the basis of clinical manifestations. Ulcerative colitis and Crohn's disease must be differentiated especially from bacillary dysentery, amoebiasis, pseudomembranous colitis, ischeamic colitis, neoplastic processes, angiodysplasia and specific forms of colitis.

7.15 Tumours of the GIT

Neoplastic processes of the gastrointestinal tract are the most frequent malign tumours. This chapter will be devoted to tumours of GIT, in addition to the tumours of pancreas and liver.

7.15.1 Carcinoma of the oesophagus

The oesophageal tumours are usually malign. Solely 10 % of tumours localised in the oesophagus are benign. Oesophageal tumours occur approximately 4 times more frequently in black people and as many as 30 times more frequently in the northern China and the surroundings of the Kaspian Sea. The exact cause of this phenomenon is unknown. They often occur in smokers, alcoholics, and in persons with gastro-oesophageal reflux. The incidence of oesophageal carcinoma is higer in patients with gluten-