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Peritonitis Generalisata, A Life-Threatening Infection Pathophysiology: A Review Article

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1. Abstract

1.1. Introduction: Peritonitis is inflammation caused by infection of the lining of the abdominal organs (peritonieum). The peritonieum is a thin, clear membrane that encloses the abdominal organs and inner walls of the stomach. The site of peritonitis can be localized or diffuse, history is acute or chronic and the pathogenesis is either infectious or aseptic. Peritonitis is an emergency which is usually accompanied by bacterecemia or sepsis. The peritoneum is a membrane consisting of one layer of mesothelic cells which is separated from the vascular connective tissue underneath by the basement membrane. It forms a closed pouch where the visera can move freely inside. Therefore, it is very important to ensure proper spontaneous breathing so that peritoneal bacterial clearance can take place. If infection spread induce peritonitis generalisata, doctor must be aware a life threatning disease. Aims of this article is to review peritonitis generalisata, a life threatning disease.

1.2. Discussion: The initial reaction of the peritoneum to invasion by bacteria is the release of fibrinous exudate. Pockets of pus (abscess) form between the fibrinous attachments, which stick together with the surrounding surface thereby limiting the infection. The adhesions usually disappear when the infection disappears, but can remain as fibrous bands, which can lead to intestinal obstruction. Inflammation causes fluid accumulation because capillaries and membranes leak. If the fluid deficit is not corrected quickly and aggressively, it can lead to cell death. The release of various mediators, such as interleukins, can initiate a hyper inflammatory response, leading to the subsequent development of multiple organ failure. As the body tries to compensate by means of retention of fluids and electrolytes by the kidneys, waste products also build up. Tachycardia initially increases cardiac output, but this soon fails once hypovolemia occurs.

1.3. Conclusion: Fluid trapping in the peritoneal cavity and intestinal lumen, further increases intra-abdominal pressure, making full breathing efforts difficult and leading to decreased perfusion and hard to self-cleansing. When the infectious material is widespread over the peritoneal surface or if the infection spreads, generalized peritonitis may develop.

2. Introduction

Peritonitis is inflammation caused by infection of the lining of the abdominal organs (peritonieum). The peritonieum is a thin, clear membrane that encloses the abdominal organs and inner walls of the stomach. The site of peritonitis can be localized or diffuse, history is acute or chronic and the pathogenesis is either infectious or aseptic. Peritonitis is an emergency which is usually accompanied by bacterecemia or sepsis [1].

The peritoneum is a membrane consisting of one layer of mesothelic cells which is separated from the vascular connective tissue underneath by the basement membrane. It forms a closed pouch where the visera can move freely inside. The peritoneum includes the abdominal cavity as the parietal peritoneum and bends to the organs as the visceral peritoneum. Its surface area is close to the body surface

area which in adults reaches 1.7m^2. It functions as a semipermeable membrane for 2-way diffusion of fluids and particles [2].

Relaxation of the diaphragm creates negative pressure so that fluids and particles, including bacteria, are sucked into the stomata, which is the gap in the mesothel of the diaphragm that is connected to the lymph lacunae to move the sub-external lymph nodes. Diaphragm contractions close the stomata and push lymph into the mediastinum. Therefore, it is very important to ensure proper spontaneous breathing so that peritoneal bacterial clearance can take place [3]. If infection spread induce peritonitis generalisata, doctor must be aware a life threatning disease. Aims of this article is to review peritonitis generalisata, a life threatning disease (Figure 1).

3. Discussion

Peritoneal cavity of healthy adults there is ± 100cc of peritoneal fluid containing 3 g / dl of protein. Most of it is in the form of albumin. The normal cell count is 33 / mm3 consisting of 45% macrophages, 45% T cells, the remaining 8% consists of NK, B cells, eosinophils, and mast cells and their secretions, especially prostacyclin and PGE2. If there is inflammation the number of PMN can increase to> 3000 / mm3 [4].

Under normal circumstances, 1/3 of the fluid in the peritoneum is drained through the diaphragmatic lymphatics while the rest is through the parietal peritoneum. Relaxation of the diaphragm creates negative pressure so that fluids and particles, including bacteria, are sucked into the stomata, which is the gap in the mesothel of the diaphragm that is connected to the lymph lacunae to move the sub-external lymph nodes (Figure 2). Diaphragm contractions close the stomata and push lymph into the mediastinum[1]. Under normal circumstances, the peritoneum can carry out fibrinolysis and prevent adhesions. The peritoneum treats infection in 3 ways:

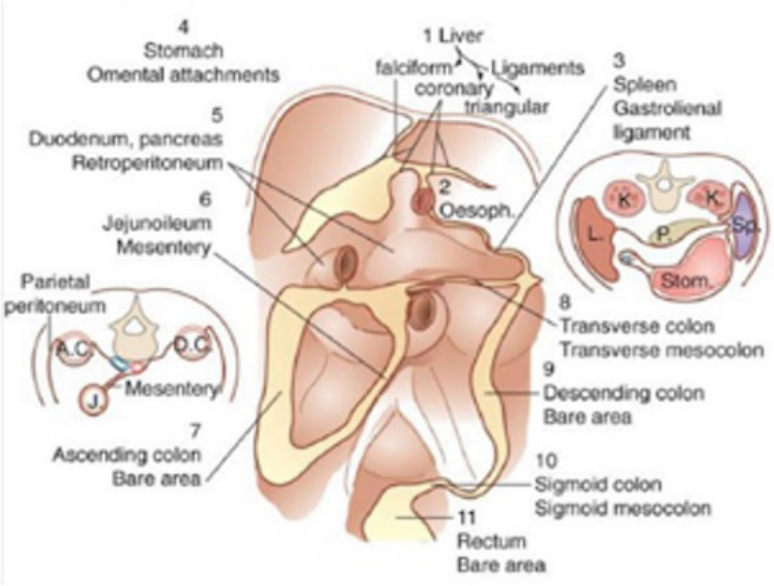

Figure 1: Ligament and Mesentrik from Peritoneum [5]

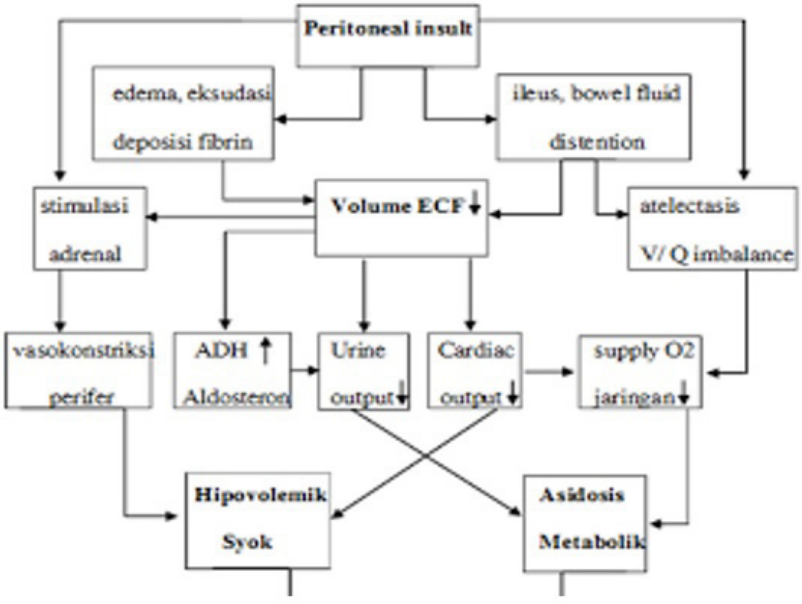

Figure 2: Pathophysiology inflammation of Peritoneum [5]

3.1. Rapid Absorption of Bacteria Through the Stomata of the Diaphragm

The diaphragm pump will attract fluids and particles including bacteria towards the stomata. Therefore, if there is an infection in the lower peritoneum, the bacteria that participate in the flow can nest in the upper part and can cause Fitz-Hugh-Curtis syndrome, which is upper abdominal pain caused by perihepatitis that accompanies fallopian tube infection.

3.2. Destruction of bacteria by immune cells

Bacteria or their products will activate mesothel cells, neutrophils, macrophages, mast cells, and lymphocytes to cause an inflammatory reaction. In addition to releasing inflammatory mediators, it can degranulate vasoactive substances containing histamine and prostaglandins. Histamine and prostaglandins released by mast cells and macrophages cause vasodilation and increased peritoneal vessel permeability, causing exudation of complement-rich fluids, immunoglobulins, clotting factors, and fibrin [2].

It is well known that for tissue healing, a pro-inflammatory mediator response is required in the area of pain until healing occurs where anti-inflammatory mediators begin to emerge that stop the pro-inflammatory process. This situation indicates a functional balance between pro- and anti-inflammatory responses. But in certain circumstances an imbalance can occur where one of them is: pro-inflammatory or anti-inflammatory or even both at the same time increases greatly beyond the patient's needs. In this situation the two conflicting mediators can cause massive organ damage resulting in organ failure [6].

3.3. Localization of The Infection as an Abscess

In increasing venular permeability, there is exudation of protein-rich fluid containing fibrinogen. Damaged cells secrete thromboplastin which converts prothrombin to thrombin and fibrinogen to fibrin. Fibrin will capture the bacteria and process them until an abscess forms. This is intended to stop the spread of bacteria in the peritoneum and prevent their entry into the systemic. Under normal circumstances fibrin can be destroyed antifibrinolytics, but in inflammation this mechanism does not function [4].

4. Etiology of Peritonitis Generalisata

Peritoneal infections can be classified as [4]:

4.1. Primary peritonitis (Spontaneus)

Caused by the hematogenous invasion of the peritoneal organs directly from the peritoneal cavity. The most common cause of primary peritonitis is spontaneous bacterial peritonitis (SBP) due to chronic liver disease. Approximately 10-30% of patients with hepatic cirrhosis with ascites will develop bacterial peritonitis (Table 1).

Table 1: Causes of Secondary Peritonitis [7]

Region	Etiology
Esophagus	Boerhaave syndrome
	Malignancy
	Trauma (mostly penetrating)
	Iatrogenic*
Stomach	Peptic ulcer perforation
	Malignancy (eg, adenocarcinoma, lymphoma, gastrointestinal stromal tumor)
	Trauma (mostly penetrating)
	Iatrogenic*
Duodenum	Peptic ulcer perforation
	Trauma (blunt and penetrating)
	Iatrogenic*
Biliary tract	Cholecystitis
	Stone perforation from gallbladder (ie, gallstone ileus) or common duct
	Malignancy
	Choledochal cyst (rare)
	Trauma (mostly penetrating)
	Iatrogenic*
Pancreas	Pancreatitis (eg, alcohol, drugs, gallstones)
	Trauma (blunt and penetrating)
	Iatrogenic*

Small bowel	Ischemic bowel
	Incarcerated hernia (internal and external)
	Closed loop obstruction
	Crohn disease
	Malignancy (rare)
	Meckel diverticulum
	Trauma (mostly penetrating)
Large bowel and appendix	Ischemic bowel
	Diverticulitis
	Malignancy
	Ulcerative colitis and Crohn disease
	Appendicitis
	Colonic volvulus
	Trauma (mostly penetrating)
	Iatrogenic
Uterus, salpinx, and ovaries	Pelvic inflammatory disease (eg, salpingo-oophoritis, tubo-ovarian abscess, ovarian cyst)
	Malignancy (rare)
	Trauma (uncommon)

4.2. Secondary peritonitis

The most common causes of secondary peritonitis are appendicitis perforation, gastric perforation and duodenal ulcer disease, colon perforation (most commonly the sigmoid colon) due to diverticulitis, volvulus, cancer and small intestine strangulation [7].

4.3. Tertiary peritonitis

Peritonitis that received inadequate therapy, superinfection of germs, and the result of previous surgery Meanwhile, intra-abdominal infections are usually divided into generalized (peritonitis) and localized (intra-abdominal abscess)[7].

5. Pathophysiology of Peritonitis Generalisata

The initial reaction of the peritoneum to invasion by bacteria is the release of fibrinous exudate. Pockets of pus (abscess) form between the fibrinous attachments, which stick together with the surrounding surface thereby limiting the infection. The adhesions usually disappear when the infection disappears, but can remain as fibrous bands, which can lead to intestinal obstruction [1, 4].

Inflammation causes fluid accumulation because capillaries and membranes leak. If the fluid deficit is not corrected quickly and aggressively, it can lead to cell death. The release of various mediators, such as interleukins, can initiate a hyper inflammatory response, leading to the subsequent development of multiple organ failure. As the body tries to compensate by means of retention of fluids and electrolytes by the kidneys, waste products also build up. Tachycardia initially increases cardiac output, but this soon fails once hypovolemia occurs [7].

The organs in the peritoneal cavity including the abdominal wall experience edema. Edema is caused by the increased permeability of the capillary vessels of these organs. Collection of fluid in the peritoneal cavity and intestinal lumen and edema of all intra-peritoneal organs and abdominal wall edema including retroperitoneal tissue causes hypovolemia. Hypovolemia increases with an increase in temperature, absent intake, and vomiting. Fluid trapping in the peritoneal cavity and intestinal lumen, further increases intra-abdominal pressure, making full breathing efforts difficult and leading to decreased perfusion [1, 4].

When the infectious material is widespread over the peritoneal surface or if the infection spreads, generalized peritonitis may develop. With the development of generalized peritonitis, peristaltic activity is reduced until a paralytic ileus develops; the intestines then atony and stretch. Fluid and electrolytes are lost into the intestinal lumen, resulting in dehydration, shock, circulatory disorders and oliguria. Adhesions can form between stretched bowel loops and can interfere with restoration of bowel movements and result in intestinal obstruction [8].

Prolonged intestinal obstruction or intestinal obstruction can cause ileus due to mechanical disturbance (blockage), thus increasing intestinal peristalsis in an attempt to overcome the obstruction. This ileus can be in the form of simple ileus, which is intestinal obstruction that is not accompanied by pinched blood vessels and can be total or partial, in stangulation ileus the obstruction is accompanied by pinched blood vessels resulting in ischemia which will end in necrosis or gangrene and eventually intestinal perforation and due to the spread of

bacteria in abdominal cavity so that peritonitis can occur [1].

Abdominal typhus is an acute infection of the small intestine caused by S. Typhi bacteria that enter the human body through the mouth from contaminated food and water. Some of the germs are destroyed by gastric acid, some enter the small intestine and reach the lymphoid tissue of the painful plaque in the terminal ileum, which is hypertrophied in this place, complications of bleeding and intestinal perforation can occur, ileum perforation in typhus usually occurs in patients with fever for approximately 2 weeks. accompanied by headache, cough and malaise followed by abdominal pain, tenderness, defansmuscular, and a general condition that deteriorates due to toxemia [4].

Perforation of a peptic ulcer is characterized by peritoneal stimulation that begins in the epigastrium and extends throughout the peritonium due to generalized peritonitis. Perforation of the stomach and anterior duodenum causes acute peritonitis. Patients who experience this perforation look in great pain like being stabbed in the stomach. This pain arises suddenly, especially in the epigastric region due to peritonium stimulation by gastric acid, bile and / or pancreatic enzymes. Then it spreads throughout the stomach causing pain throughout the stomach at the beginning of the perforation, there has been no bacterial infection, sometimes this phase is called the chemical peritonitis phase, the pain in the shoulder indicates peritoneal stimulation in the form of diluting stimulating acid salts, this will temporarily reduce complaints until then bacterial peritonitis occurs [1, 9].

In appendicitis it is usually caused by blockage of the lumen of the appendix by hyperplasia of lymphoid follicles, fecalites, foreign bodies, strictures due to fibrosis and neoplasms. This obstruction causes the mucus produced by the mucosa to experience a duct, the longer the mucus is getting more, but the elasticity of the appendix wall has limitations so that does not cause an increase in intraluminal pressure and inhibits lymph flow which results in edema, bacterial diapedesis, mucosal ulceration, and venous obstruction so that edema increases and then arterial flow is interrupted, an appendix wall infarction will occur followed by necrosis or gangrene of the appendix wall, causing perforation and ultimately resulting in good peritonitis local and general [10].

In abdominal trauma, both penetrating abdominal trauma and blunt abdominal trauma can result in peritonitis up to sepsis when intra-peritonial hollow organs are involved. Peritonial stimuli that arise in accordance with the contents of the hollow organ, ranging from the chemical gastric to the colon containing feces. Chemical stimuli have the fastest onset and slowest feces. If the perforation occurs in the upper part, for example in the stomach area, there will be stimulation immediately after the trauma and there will be symptoms of severe peritonitis, whereas if the lower part is like the colon, initially there are no symptoms because the microorganisms need time to multiply and then after 24 hours develop acute abdominal symptoms because peritoneal stimulation [1,10].

6. Conclusion

Fluid trapping in the peritoneal cavity and intestinal lumen, further increases intra-abdominal pressure, making full breathing efforts difficult and leading to decreased perfusion and hard to self-cleansing. When the infectious material is widespread over the peritoneal surface or if the infection spreads, generalized peritonitis may develop.

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