Management of surgical infection in patients with critical limb ischemia (State of Art)

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Abstract
Critical limb ischemia (CLI) is a crucial medical and socio-economic problem. Infectious complications in patients after reconstructive surgery significantly aggravate survival and lead to increased incidence of extremity loss.

Keywords: infection, critical limb ischemia, surgical patients

Introduction
Critical lower limb ischemia (CLI) is the most severe manifestation of peripheral arterial disease (PAD) of the lower extremities. According to Engelhardt M et al., prevalence of CLI in Europe is 50-100 per 100 thousand people [1, 2]. Every year about 171,000 patients with CLI are operated in Europe due to chronic CLI [3]. In Ukraine, official information on the prevalence of CLI is currently unavailable.

Despite advancements (high performance of restorative vascular surgeries) postoperative complications remain as an open problem. According to current data there is an increase in the total number of surgical patients with suppurative infectious complications from 35 to 40%, including patients with CLI where it accounts for up to 85% [4].

Most of postoperative complications arise after surgery in the area of abdominal aorta and its branches. Surgery (amputation, reconstruction or angioplasty) is a method of choice in patients with CLI [3, 5, 6].

The course of surgical intervention in patients with CLI is affected by many factors and directly depends on general condition of the patient and the presence of tissue damage. Trophic changes of ischemic areas can become a potential source of chronic infection, and a feasible source of surgical site infections (SSI).

Center for Disease Control and Prevention in the US defines SSI as an infection of the site of surgical incision, organ or cavity that occurs during the first 30 postoperative days (in case of the transplant – during the year). SSI incidence varies from 0.5 to 15% (an average of 5.3%) depending on the type of surgery and condition of the patient [7, 8]. About 2/3 of infections are located in the area of incision and only 1/3 affects an organ or body cavity in the area of surgery [9].

SSI is considered neither nosocomial in case of complication nor continuing infection, which occurred in a patient before surgery, unless new species of microorganism has been identified, or change in the nature of clinical symptoms, allowing a high degree of probability to suspect acquiring of new infection.

Overall risk of SSI in the particular patient can be calculated based on three factors: operation class by the degree of bacterial contamination, the extent of surgical risk - ASA (according to the criteria of the American Society of Anesthesiologists), duration of surgery [10].

Microflora of inflammatory source in patients with CLIs is usually polymicrobial. According to recommendations for treatment of diabetic foot (USA) the most common pathogen of PNL is a pathogenic staphylococcus - Staphylococcus aureus [11, 12], which is often accompanied by saprophytic Staphylococcus epidermidis Coccal flora is detected with microorganisms of different species of Enterobacteriaceae - Escherichia coli, Klebsiella pneumoniae, Proteus species and anaerobic microorganisms [12]. The microflora of chronic PNL commonly has an acquired resistance to most antibiotics, and it is multiresistant. The rate of resistant strains of Staphylococcus aureus is 30% [13].
Surgical treatment of patients with occlusive-stenotic lesions in arteries of the lower extremities is complicated by infection in 4-25% of operated patients [14-16]. The percentage of purulent-septic complications after surgery in patients with IV stage of arterial insufficiency ranges from 56.9 to 64.7% and is independent of systemic antibiotic prophylaxis [17].

Infection of postoperative wounds is associated with decreased regenerative capacity of tissues and their stability, lower adaptive capacity of the body to infection, presence of chronic venous disorders in most patients due to long history of the disease [4]. So according to many data [18-21] infectious component is a major cause of complications following lower limb amputations in patients with obliterator atherosclerosis, and chronic arterial insufficiency creates favourable conditions for the reproduction of microorganisms.

According to the Center of Disease Control surgical wound complications after reconstructive surgery can be divided into 3 categories: superficial (affecting only the skin and subcutaneous tissue), deep (involving fascia and muscles), graft lesions [22, 23]. Surgical wound infection can cause graft festering that is especially dangerous when using implants as a material for reconstruction. Such complication can result in arrosive haemorrhage being an indication to remove the material for reconstruction. Such complication can result in duration of surgery for more than 220 minutes and factors of transfusion of two or more blood bags of preserved blood, infection of postoperative wounds is associated with decreased defenses [27, 28]. In most cases SSI is developed under the influence of endogenous microflora of the skin and mucous membranes of the patient. Wound infection usually occurs during surgery [29].

Our studies of patients with different kinds of trophic lesions of the lower extremities revealed a significant role of staphylococci and mixed microflora in the occurrence of infectious complications. In 88% of cases these microorganisms are typical representatives of opportunistic microflora of the skin and gastrointestinal tract [30].

Much attention has always been paid to assessing the role of microbial factor in the development of infection as it is well known that the particularity of the course of infection and peculiarities of morphological changes in organs depend on the type of microorganism.

Current information on the structure of SSI pathogens was obtained in the course of large-scale research conducted from 1997 to 2005 in the UK at 247 hospitals. The study includes data of 240,231 surgeries, 7,194 (3%) of which were complicated with the occurrence of SSI. The observed spectrum of pathogens echoes previous studies. In 53% of cases S. aureus became the agent of SSI, and in 27% infection was caused by association of microorganisms [31].

References
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