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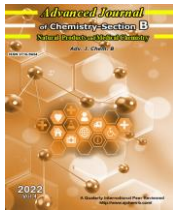

Review Article

A Review of Systemic Inflammatory Response Syndrome in Patients with General Anesthesia Candidates for Rhinoplasty

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ABSTRACT

Introduction: Since rhinoplasty surgery is performed under general anesthesia and the prevalence of this type of surgery in the Iranian population is increasing at a very fast rate, and on the other hand, because there is no accurate information about the systemic inflammatory response caused by general anesthesia in candidate patients. Rhinoplasty is not available. We decided to conduct the present study with the aim of reviewing the systemic inflammatory response syndrome in patients with general anesthesia who are candidates for rhinoplasty.

Method: Keywords such as General Anesthesia, rhinoplasty, surgical stress, systemic inflammatory response syndrome, pro-inflammatory cytokines, and anti-inflammatory cytokines in the Iranian and international databases by both authors of the present study with the help of Boolean operators (AND, OR, and NOT) were searched and the obtained studies that met the criteria for entering this study were evaluated.

Results: The most important topics discussed by examining the number of articles include surgical stress and systemic inflammatory response syndrome, the role of immune cells in systemic inflammatory response syndrome, the potential effects of cytokine release during surgery and general anesthesia, and the role of promoting cytokines. Inflammatory in systemic inflammatory response syndrome caused by rhinoplasty.

Conclusion: During surgical stress, neuro-endocrine, metabolic, inflammatory, and immune systems can be activated rapidly and potentially harmful consequences such as cardiac dysfunction, cardiovascular instability, endothelial activation, inflammation, vascular dysfunction, and possibly immunosuppression. Enhancing endothelium function may reduce the incidence of excessive inflammation, immune system, and complement, as well as complications such as systemic inflammatory response syndrome, multiple organ dysfunction syndrome, and multiple organ/organ failure.

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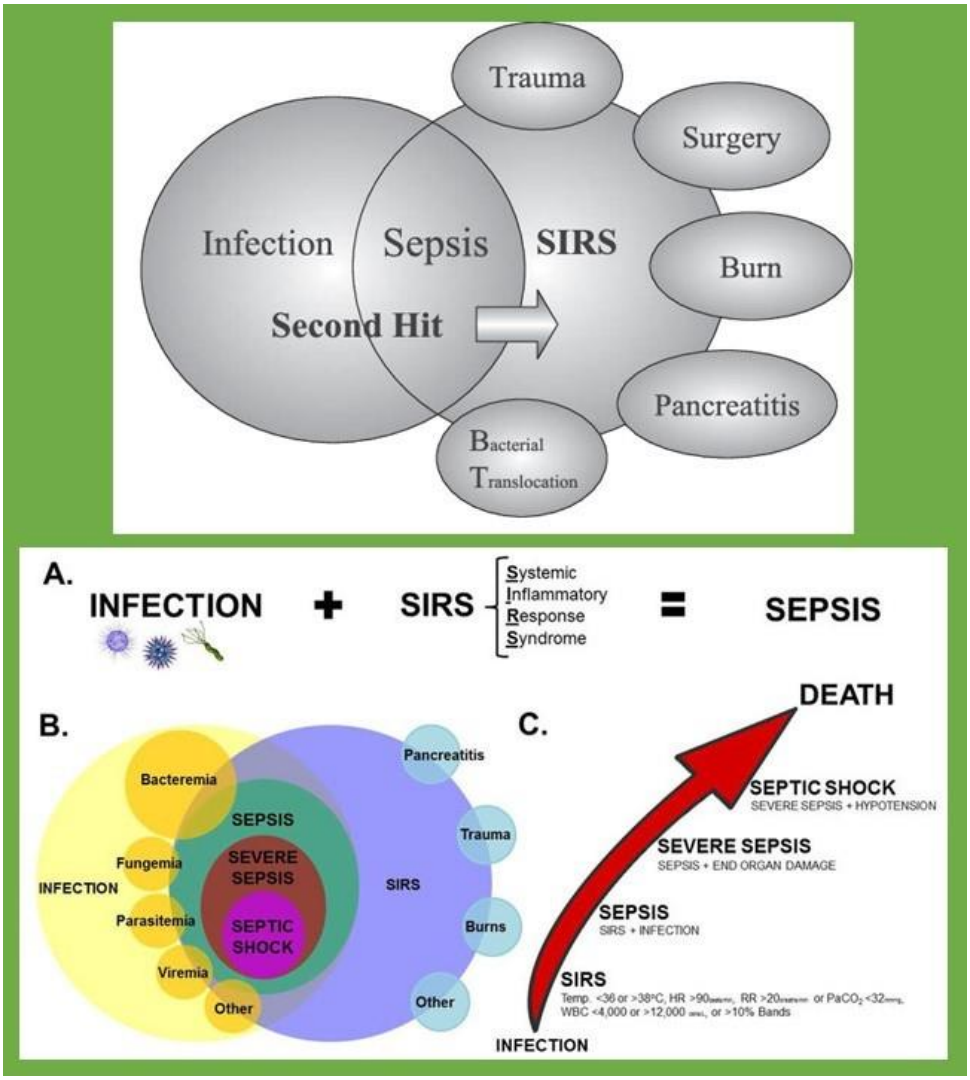
GRAPHICAL ABSTRACT



1. Introduction

Major surgery refers to any type of intervention performed in a hospital environment that requires cutting, splitting, manipulation, tissue sutures, local anesthesia, general anesthesia, or providing sedation for pain control [1]. Any injury caused by or caused by major surgery is referred to as surgical trauma [2]. The extent of damage caused by surgery depends on the type and duration of surgery and anesthesia, the presence of cardiopulmonary bypass, and patient-related factors such as age, sex, health status of the patient before surgery, treatment profile, and pain after surgery. The surgical stress response can be defined as the host body's response to a stressor (which may include a serious injury, bleeding, infection, or burn) [3]. This response is a physiological reaction that is very important in medicine. One of the basic stages of surgical stress response results from the interaction between injury, inflammation, infection, and organ dysfunction [4] (Figure 1).

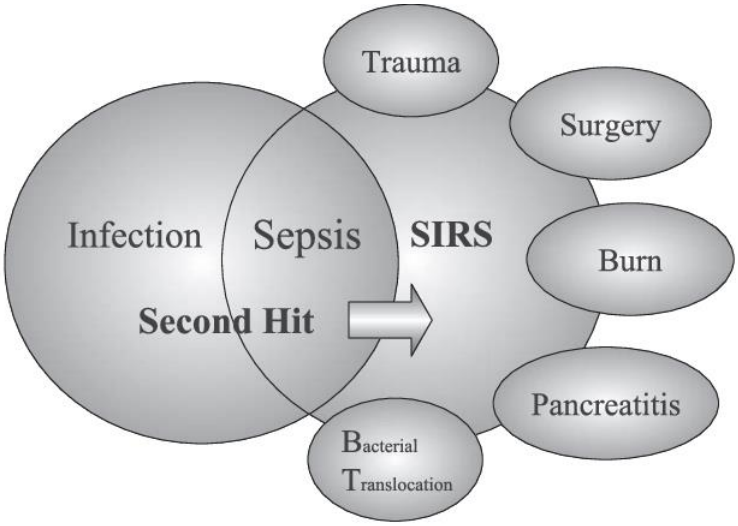


Figure 1. Systemic inflammatory response syndrome and infection

In response to injury, immune cells are activated and blood-derived immune cells are activated in the injury area. Likewise, immune cells initiate the sensitivity of peripheral pain receptors. Inflammation is known by its five characteristics, i.e. swelling, redness, heat, pain, and organ dysfunction [5]. When the inflammatory response is not controlled, the systemic inflammatory response syndrome occurs. Systemic inflammatory response syndrome is considered as an adverse event after surgery, which is associated with delayed immunosuppression [6]. As with mild stress, mild immunosuppression is generally not harmful, but its progression and spread can lead to secondary infection, which eventually leads to multiple organ dysfunction and even death [7].

Despite this, the process of disruption in several organs is still considered as the biggest destructive factor in delayed trauma in terms of prevalence and mortality rates. Meanwhile, a regular complex of inflammatory polypeptide molecules participates in the occurrence of this inflammatory response which is known as cytokines [8]. Lack of local control in the release of these cytokines causes systemic inflammation and potentially devastating complications such as systemic inflammatory response syndrome, multiple organ dysfunction syndrome, shock, and death [9].

Since rhinoplasty surgery is performed under general anesthesia and the prevalence of this type of surgery in the Iranian population is increasing at a very fast rate, and on the other hand, because there is no accurate information about the systemic inflammatory response caused by general anesthesia in rhinoplasty candidate patients who decided to participate in the present study with the aim of reviewing the systemic inflammatory response syndrome in patients with general anesthesia who are candidates for rhinoplasty.

Method

The present study is a civilian review conducted by checking the databases of Google Scholar, PubMed, Scopus and Medline, SID, and Magiran without any time limit among the articles published in Persian and English languages. The inclusion criteria of the studies in this present study were full-text articles, articles with keywords general anesthesia, rhinoplasty, surgical stress, systemic inflammatory response syndrome, pro-inflammatory cytokines, anti-inflammatory cytokines, and articles in the form of case reports, case series and presented in the

conference were excluded from the review process. The keywords mentioned in Iranian and international databases were searched by both authors of the present study with the help of Boolean operators (AND, OR, and NOT) and the studies that met the review criteria in terms of the title and abstract of the article were included in this study. In the next step, the text of the articles was read and those in line with the objectives of the present study were included in this civilian review.

Results

In total, the number of articles were evaluated and the headings presented in the following and will be discussed were considered as important and basic materials and were evaluated so that their information can be used for further studies.

Surgical stress and systemic inflammatory response syndrome

Among the indicators of surgical stress, we can mention the type of surgical approach, duration of surgery, and the amount of blood lost by the patient during surgery, which are closely related to the occurrence of destructive factors after surgery [10]. In patients who have undergone major abdominal surgery for the treatment of gastrointestinal malignancies, the production of IL-1ra and Il-10 increases more significantly than in the conditions of sepsis after surgery [11].

Acute pain caused by trauma, surgery, or some diagnostic processes such as bone marrow aspiration stimulates pain receptors and increases the release of catecholamine's and other hormones [12]. On the other hand, the pain caused by inflammation is a chronic and natural pain associated with tissue damage and the release of inflammatory mediators from the damaged tissue. Low monocyte HLA-DR expression is associated with increased risk of systemic inflammatory response syndrome and sepsis following pediatric cardiothoracic surgery [13]. In a murine model of sepsis following ligated cecal puncture, both pro-inflammatory and anti-inflammatory cytokines were markedly elevated in mice that died within 24 hours [14].

However, the interaction between pro-inflammatory and anti-inflammatory pathways can lead to sepsis. It has been stated that systemic inflammatory syndrome is a reflection of the degree of surgical stress and as an evaluation system; it shows the intensity of stress after surgery [15].

Response to the surgical stress is determined by complex interactions between endocrine, immune, and hemopoietic systems [16]. Tissue damage leads to hypovolemia and neuro-hormonal reflex pain. This event is regulated by the release of corticotrophin, endorphins, growth hormones, vasopressin, and prolactin [17].

The activation of the sympathetic system increases the plasma level of catecholamine's, while the increase in cortisol and aldosterone levels is caused by the activation of the renin-angiotensin system [18]. The results of these processes are: catabolism event, insulin resistance, increased metabolism rate, and water and sodium reabsorption. The response severity depends on the severity of the injury, so surgical techniques and different anesthetic drugs can affect these responses. Pain control seems to play an important role in reducing these responses [19]. It has been found that the stress response after surgery is related to the creation of nerve messages in the surgical wound and of course the release of cytokines during and after surgery. The other molecules such as p55, p75, and phospholipase A2 have been introduced as indicators of the degree of surgical stress [20].

After surgery, fever also occurs due to a regulated increase in body temperature. The clinical manifestation of this syndrome includes disturbances in body temperature, breathing rate, heart rate, and white blood cell count [21]. The simultaneous occurrence of this syndrome with infection is called sepsis, and severe sepsis has been mentioned as the cause of organ failure,

deficiency in perfusion, and blood pressure drop, while septic shock, restoring body fluids, leads to blood pressure drop caused by sepsis and reduced perfusion [22]. Although the occurrence of this syndrome after surgery is variable, this unpleasant event is often reported. Systemic inflammatory response syndrome has a direct relationship with mortality and organ failure [23].

Surgical stress may occur as a result of the lack of proper hemostasis and is considered as a factor to increase various devastating complications after surgery. Therefore, the category of surgical stress is an important issue in post-surgical management [24]. However, various physiological responses suggest that these factors are insufficient in predicting the incidence of postoperative complications. It has been reported that changes in the response of the neuroendocrine system, such as changes in the amount of cortisol and catecholamine's, or immune responses such as cytokines, reflect the degree of surgical stress [25]. The development of clinically useful markers to prevent and detect the early and rapid complications caused by inflammation are necessary and are concerned as highly important goals in post-surgical management [26].

Systemic inflammatory response syndrome is a non-specific systemic inflammatory response characterized by disturbances in more than two of the following four criteria: (i) temperature, (ii) heart rate, (iii) the number of breaths per minute, and (iv) the number of white blood cells. It has been reported that systemic inflammatory response syndrome is a preliminary stage in the progression of pathological disorders such as sepsis, dysfunction syndrome in multiple organs, and multiple organ failure [27]. The said pathology is caused by the increase of cytokines in the blood including the activation of the cytokine network in response to initial stress in the body [28].

In some physical responses, cytokines are alternately evaluated as an indicator to determine the degree of surgical stress [29]. In particular, it has been reported that the serum level of interleukin-6 is a sensitive indicator of pathological response, which also reflects the level of surgical stress. Patients with sepsis and severe trauma, following bypass of the cardiovascular system or patients undergoing major surgery show the acute phase of the inflammatory response, which is characterized by clinical symptoms including fever, drowsiness, and anorexia [30].

Center proteins of the acute phase of the liver, activation of the complement system, increase in the number of white blood cells, decrease in lymphocytes in the peripheral blood, and the disorders occurrence in metabolism are the biochemical characteristics of this inflammation [31]. When the inflammatory response is not controlled, the systemic inflammatory response syndrome occurs. Systemic inflammatory response syndrome is considered as an adverse event after surgery. This concept was proposed for the first time in 1991 at the conference of the American Society of Chest Physicians and the Society of Critical Care Medicine, and a set of clinical symptoms was proposed by which the systemic inflammatory response syndrome is known [32].

These symptoms include: Tachypnea, fever, or increased body temperature, decreased heart rate, and leukocytosis, or leukopenia with a change in the number of white blood cells (increase in immature polymorph nuclear cells) [33]. It is well confirmed that in an extensive surgical intervention, the occurrence of systemic inflammatory response caused by sepsis or extensive trauma is associated with immunosuppression (cell-mediated and humoral systems) [34].

The role of immune cells in systemic inflammatory response syndrome

Innate immune cells (such as monocytes and macrophages) initiate the primary inflammatory response in patients with systemic inflammatory response syndrome. In human studies, a change in the expression of HLA-DR antigen of luxate monocytes has been seen. Increased expression of HLA-DR on monocytes has been seen in elderly patients undergoing major abdominal surgeries [35]. The systemic inflammatory response syndrome is initiated by the binding of Toll-like receptors (TLRs) to their ligands. In the early stages of the inflammatory process, large amounts of TNF-α and IL-1 are released systemically and are involved in the release of stress hormones such as adrenaline and noradrenaline, the occurrence of fever, and the IL-1release [36].

Interleukin-6 plays a role in the production of acute phase proteins, including C-reactive protein (CRP) and pro-calcitonin. It was mentioned before that the increase of IL-1 concentration is correlated with the worse prognosis of major traumas and surgery. It is interesting to note that IL-6 plays a role in the anti-inflammatory response in trauma patients through the production of prostaglandin E2 and the reduction of IL-10 [37].

Potential effects of cytokine release during surgery and general anesthesia

Pro-inflammatory cytokines and anti-inflammatory cytokines are released by an initial stimulation and participate in the inflammation process. Anti-inflammatory cytokines act to localize and prevent the occurrence of excessive inflammation, and the lack of control of this local inflammation leads to systemic inflammation and its harmful effects, including systemic inflammatory response syndrome, multiple organ dysfunction syndrome, shock, and death [38] (Figure 2).

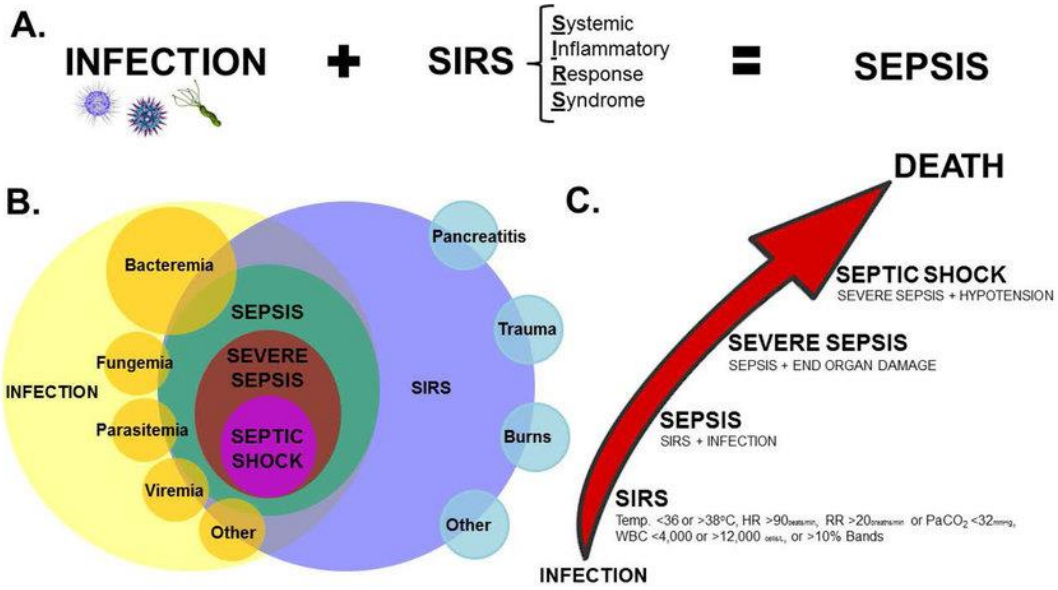


Figure 2. Systemic inflammatory response syndrome and infection

Initiating factors of systemic inflammatory response syndrome include the following:

1. Endotoxin/lipopolysaccharide (LPS) is a fragment of the cell wall protein of gram-negative bacteria. LPS is one of the primary factors in the stimulation and development of the systemic inflammatory response syndrome. In contrast, TNF-α and interleukin-8 are associated with

bypass duration, systemic inflammatory response syndrome, and multi organ dysfunction syndrome [39].

2. Complement system: The part of the immune system that strengthens the ability of antibodies and xenophagous cells to clear microorganisms and damages cells, attacks the cell membrane of the pathogen, and encourages the inflammatory process is called the complement system [40]. The complement system is a family of plasma proteases related to the innate immune system. When this system is activated, it has the ability to destroy many proteins (by piercing their coat) and activate cytokines. C3a and C3d proteins of the complement system are increased in sepsis, which are directly related to PAI-1 (plasminogen activator inhibitor 1) and inversely related to AT-3 (anti thrombin 3). However, the levels of C3a and C3d have no relationship with the levels of TNF-α and interleukin-6, which are reduced following the treatment of sepsis [41].

It has been shown that the levels of classical and alternative complement system proteins are increased in different proportions in septic, as compared with non-infected patients with systemic inflammatory response syndrome. Therefore, the amount of these proteins increases and can be identified at least 3 days before the appearance of clinical symptoms of systemic inflammatory response syndrome. Many complement system proteins may be used as the dependent biomarkers for the early diagnosis and future therapeutic targets [42].

3. Ischemic injury-reperfusion: It has been reported that the plasma levels of TNF-α in rats' increase more after 3 hours of bilateral hind limb ischemia compared to one hour after reperfusion. Likewise, the levels of interleukin-6 following reperfusion increase increasingly [43]. In the studies performed on patients undergoing sub vaginal artery reconstruction surgery, it was shown that the serum level of TNF-α and the permeability of the intestinal mucosa are higher than in the case of ischemia of the posterior motor organ. In addition, it was shown that the degree of intestinal permeability depends on the time course of the arterial clamp. These studies suggest that ischemic-reperfusion injury is a potential cause of systemic inflammatory response syndrome [44].

4. Oxidative stress: Measurement of plasma sulfhydryl (thiol) groups (for example, glutathione (GSH)), and alpha-tocopherol in 26 trauma patients in the ICU showed that the redox status was increasingly worsened, with a significant increase in the oxidation of plasma glutathione (due to the worsening of the redox status reduction), and the scoring of disorder syndrome in several organs is higher on the 10^{th} day after the injury. In addition, a decrease in total plasma glutathione levels was observed in some of these patients, which indicates the collapse of the glutathione-dependent antioxidant system. According to the mentioned information, it is possible to consider the possible role of oxidative stress in the development of systemic inflammatory response syndrome [45].

Role of pro-inflammatory cytokines in rhinoplasty-induced systemic inflammatory response syndrome

Systemic inflammatory response syndrome is a pro-inflammatory stage that affects the whole body. The occurrence of this response following an infectious agent is called sepsis. The other causes of this syndrome include complications after surgery, pancreatitis, trauma, and burns. The systemic inflammatory response syndrome is initiated by the usual inflammatory pathways and is defined as a three-stage process [46]. In the first stage, following the injury, local swelling begins due to the production of the local cytokines. These local reactions lead to the activation of the reticular-endothelial system and, of course, stimulation of wound healing. In the second stage, cytokines are introduced into the general blood stream, which cause the calling of macrophages and platelets. Following the

decrease in the release of pro-inflammatory mediators, the return of homeostasis is observed [47]. If the normal homeostasis of the body does not occur and the release of pro-inflammatory mediators continues, the third stage of this syndrome is seen, which causes general damage, activation of humoral processes, disruption of blood supply continuity, and finally organ failure [48].

Three important cytokines in this syndrome are TNF-α, IL-1, and IL-6. Cytokines are released as a cascade. Pro-inflammatory cytokines also play a role in the production of nitric oxide and leukotrienes. The leukotrienes production increases in patients with systemic inflammatory syndrome. MK-886, a leukotriene biosynthesis inhibitor, has been shown to reduce acute lung injury following hemorrhagic shock by reducing pro-inflammatory cytokine production [49]. Cytokines that are initially released include TNF-α and IL-1β, which stimulate the production of the other pro-inflammatory proteins. TNF-α, IL-1, IL-6, IL-8, and macrophage inflammatory protein 1-alpha (MIP-1α) are among the most important cytokines promoting inflammation. It has been shown that these cytokines are consistently associated with mortality following severe injury, and also TNF-α and IL-6 levels are associated with poor outcome from sepsis [50].

TNF-α: TNF-α is a 17 kDa protein produced mainly by monocytes. It has been reported that injection of recombinant TNF-α in humans has led to systemic inflammatory response syndrome with fever, hemodynamic disorder, leukopenia, increased liver enzymes, and coagulation disorders. The role of TNF-α in burn-related systemic inflammatory response syndrome have stated the opposite. Sometimes there is no change and sometimes a significant increase in the production of this cytokine has been reported [51]. However, an increase in the concentration of TNF-α is seen after trauma and severe bleeding. It has been observed that the high concentration of TNF-α in patients with severe pancreatitis is a sign of lack of recovery and bad prognosis. It has been stated that higher concentrations of pro-inflammatory cytokines are a sign of a more severe disease process. The precise role of TNF-α in systemic inflammatory response syndrome after surgery is worth evaluating [52].

IL-1: Interleukin 1 includes two types of IL-1α and IL-1β proteins, both of which act on the same IL-1 receptor. IL-1 is secreted by monocytes, neutrophils, and other cell types. It has been shown that the injection of IL-1 in humans leads to fever, hemodynamic disorders, anorexia, lethargy, joint pain, and neutrophilia [53]. It has also been reported that the injection of endotoxin in humans increases the interleukin level. From the viewpoint that interleukin-1 has pro-inflammatory activity, its abnormally low levels may also play a role in the development of systemic inflammatory response syndrome [54].

IL-6: Several mediators are involved in this matter, but interleukin 6 is considered a cytokine and an important mediator in this regard. The level of interleukin 6 is correlated with various parameters, including the nature of surgery (type of surgery, duration and volume of blood loss, and surgical complications), increase in body metabolism, stress hormone levels, and C-reactive protein concentration [55]. However, the other cytokines are also involved in the pathophysiology of this syndrome. It is known that minor traumas also lead to an increase in IL-6. When sepsis occurs after selective surgeries, IL-6 production increases significantly on days 1 to 5 after surgery. It has been seen that the serum concentration of this cytokine rises during the occurrence of systemic inflammatory response syndrome after hepatectomy surgery. A sharp increase in the serum concentration of this cytokine in acute pancreatitis has been mentioned as a predictive factor. Observing the decreasing trend of this factor is considered as the recovery of the disease. In this way, IL-6 is

called a biological index in the evaluation of systemic inflammatory response syndrome [56].

Other interleukins: Interleukins 8 and 17 are also pro-inflammatory cytokines [56].

Anti-inflammatory cytokines

Simultaneously with the release of pro-inflammatory mediators, compensatory compounds are released to cause the anti-inflammatory response syndrome. Anti-inflammatory cytokines are produced simultaneously with pro-inflammatory cytokines, which balance and control inflammation [57]. The most important anti-inflammatory cytokines include IL-10 and IL-13. It has been shown that anti-inflammatory cytokines play a role in the pathogenesis of systemic inflammatory response syndrome in sepsis and it has been shown that the serum levels of pro-inflammatory cytokines (TNF-α, IL-6, and IL-8) along with the anti-inflammatory cytokine IL-10 are also are increasing significantly. There is a strong correlation between IL-10 and TNF-α in patients with fatal outcomes [58].

IL-10: IL-10 is an 18 kDa anti-inflammatory cytokine produced by monocytes and lymphocytes. IL-10 has poly-tropic effects in the regulation of the immune system, which include the regulation of T helper type 1 cytokines (TNF-α, IL-2, IL-3, and interferon gamma), decreasing the expression of MHC class 2 antigen, increasing the survival of B cells and the block of some NF-kβ production pathways indicated [59]. A comparative study on 12 healthy volunteers and 12 patients with systemic inflammatory response syndrome showed that the levels of TNF-α and IL-10 in patients with systemic inflammatory response syndrome and disorders in several organs/organs were higher than healthy volunteer patients. It has been reported that intraperitoneal loading of IL-10 in a mouse model reduces serum TNF-α response to the inflammatory stimuli. This study suggests that IL-10 is an antagonist of TNF-α in the pathogenesis of systemic inflammatory response syndrome [60].

IL-13: Interleukin-13 is an anti-inflammatory cytokine with a weight of 17 kDa, which is mainly secreted by T-helper cells. This interleukin induces the secretion of immunoglobulin E from B cells, has an upregulation effect on matrix metalloproteinase (MMPS), which reduces inflammation and stimulates the proliferation of lymphocytes [61]. Interleukin 13 increases compared to TNF-α in systemic inflammatory response syndrome and also causes a degree of leukopenia. These studies suggest that IL-13 plays an important role in the pathogenesis of systemic inflammatory response syndrome and is also considered as a regulator of TNF-α and leukocyte response [62].

Discussion

Surgery causes changes in neuro-endocrine, metabolic, inflammatory, and immune systems and finally causes the response to surgical stress [11]. Naturally, the surgical stress response is self-limiting and resolves. The complications of the created damage and the subsequent inflammatory cascade have led to significant problems in advancing effective therapeutic approaches in the treatment of systemic inflammatory response syndrome and disorder syndrome in several organs [25]. Regarding therapeutic strategies with release targets, primary inflammatory mediators, and even physiological responses to inflammation, there are many data indicating that their use has not been successful [26]. There are still many unanswered questions about the mentioned treatment approaches. Some extensive research based on the primary pro-inflammatory cascade, how they are modulated, methods of pro-inflammatory cytokine extraction, and how genetic polymorphisms may affect the natural history of systemic inflammatory response syndrome in patients [14]. However, there are

some promising data regarding the novel absorbent strategies in reducing hyper cytokinesis caused by systemic inflammatory response syndrome [29]. The need to reduce the clinical and rapid levels of pro-inflammatory cytokines has led to the extensive research on the selection of the type of compound and absorbing solution of these substances [31]. The use of activated charcoal and polymyxin B hemofiltration systems has had encouraging results in the discussion of absorbent materials in the treatment of patients with systemic inflammatory response syndrome [39].

Conclusion

During surgical stress, the neuroendocrine, metabolic, inflammatory, and immune systems can be rapidly activated, leading to potentially harmful consequences such as cardiac dysfunction, cardiovascular instability, endothelial activation, inflammation, vascular dysfunction, and possibly immunosuppression. Enhancing endothelium function may reduce the incidence of excessive inflammation, immune system, and complement, as well as complications such as systemic inflammatory response syndrome, multiple organ dysfunction syndromes, and multiple organ/organ failure.

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