POSTOPERATIVE TOXIC SHOCK SYNDROME: REPORT OF A CASE

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국문초록

Toxic Shock Syndrome의 증례보고

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Toxic Shock Syndrome(TSS)은 1978년 Todd등에 의해 처음 보고된 이래 젊은 여성에서 월경과 연관된 질환으로 생각되어 왔으나 최근에는 코수술을 비롯한 두경부수술 및 기타 소수술후 합병증으로 많은 증례가 발표되고 있다. 이의 진단은 주로 임상적 진단, 즉 갑작스런 술후 고열, 설사, 저혈압 및 홍피증과 혈액배양등에서 검출되는 포도상구균등으로 가능하다. TSS은 대부분 수액공급 및 혈압의 유지, 전신적인 항생제 투여등으로 쉽게 치료가 가능하지만, 경과가 좋지 않은 경우 면역글로불린의 사용도 시도되어 좋은 결과를 얻고 있으며 조기 발견 및 처치가 가장 중요하다고 할 수 있다. 이에 저자는 상악골 및 비골, 안와근심벽 골절을 지닌 48세 남자환자에서 골절부에 대한 관혈적 정복고정술후 발생된 TSS의 처치에 대해 문헌고찰과 함께 보고하는 바이다.

주요어 : Toxic Shock Syndrome, 포도상구균, 조기발견 및 처치

Ⅰ. INTRODUCTION

Toxic shock syndrome(TSS) is an acute multisystem disease that has recently been recognized to occur in a variety of clinical settings. It was first reported in 1978 as a rare complication of staphylococcal infection. Previously, at first it was thought to be a childhood disease and a menses-related clinical setting as an illness of menstruating women using intravaginal tampons, recently, however, cases have been described in nonmenses-related clinical settings, in particular, in association with postoperative infections, as a complication of minor surgery, burns and minimal skin infections[1,2].

The purpose of this article is to report a case of TSS treated by the open reduction and internal fixation of maxilla fracture, nasoorbitoethmoidal fracture with nasal packing with a review of literatures.

Ⅱ. CASE REPORT

A 48-year-old man was admitted to our hospital, he was injured on the multiple facial bones due to an industrial accident. After careful study, we could make a diagnosis of basal skull fracture, frontal skull fracture, maxilla fracture(Lefort II with midpalatal fracture), NOE fracture(Fig. 1,2). The patient showed the pain and swelling on the entire face, anterior open bite, mouth opening limitation(2.5 FB), epistaxis, and oral bleeding. Routine laboratory results were within normal limits.

He was performed the open reduction under the general anesthesia through the intraoral circumvestibular incision. After the fractured maxilla was mobilized with disimpaction forceps, closed reduction with arch bars and intermaxillary fixation was done and internal fixation was done with 3 miniplates and 1 microplate and screws(Fig. 3). After closed reduction of NOE fracture with the periosteal elevator via both nostrils, nasal packing with vaseline gauze on the both nostrils was done and nasal splint was applied for stability. Postoperative hospital course was uneventful and nasal packing was removed on the third postoperatve day, but next day the patient showed the sudden onset of fever(39.4℃), diarrhea, hypotension(70/40 mmHg), erythroderma on the face and upper extremities, and altered mentality.

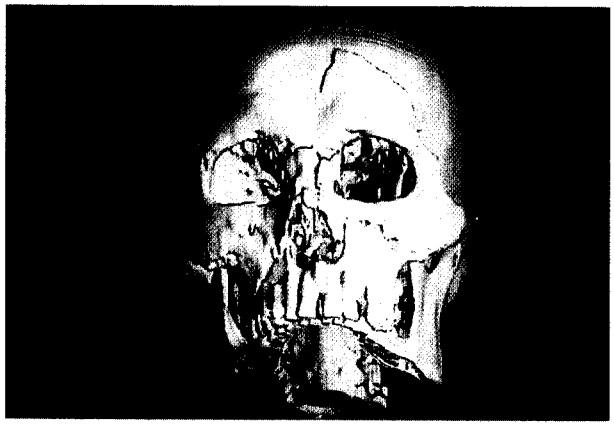

Fig. 1. Preoperative 3D CT finding

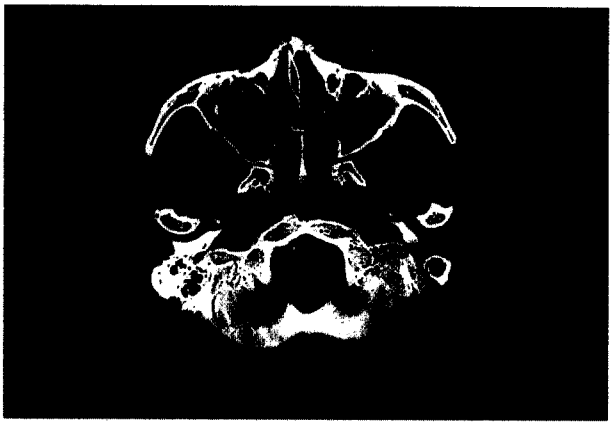

Fig. 2. Preoperative CT shows nasal bone fracture with maxilla fracture

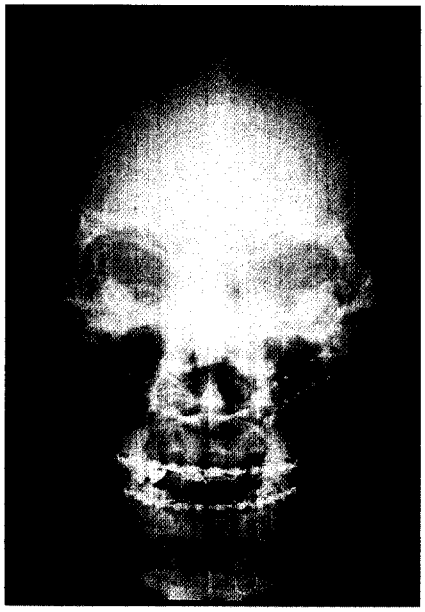

Fig. 3. Postoperative skull P-A

Laboratory tests revealed that Staphylococcus aureus was found on the sputum culture, white blood cell count was 28.4×10^3, mild anemia, and mild thrombocytopenia, but another laboratory results including blood culture, cerebrospinal fluid culture were within normal limits. Under the diagnosis of TSS, the patient was transfered to the intensive care unit, and cared with intravenous fluid resusitation, antibiotics(cefotazole), and dopamine. 3 days after the onset of TSS symptoms, patient's blood pressure and body temperature returned to normal. Hospital course afterwards was uneventful and the patient was discharged on the postoperative 46th day.

Ⅲ. DISCUSSION

Toxic shock syndrome(TSS) is a severe toxin-mediated multisystem disease which is a rare, rapidly progressive, and potentially fatal syndrome associated with postoperative wound infections[3-6]. It was first reported by Todd et al. in 1978, and as a rare complication of staphylococcal infection related with the use of tampons during menstruation[2,7,8]. But recently TSS continues to be encountered with nonmenses-related clinical settings, and the reported number of menstrually related cases of TSS has dropped substantially and it can occur in males as well as females and is not necessarily related to menstruation[2,7,8]. More than 2800 cases have been reported at the Centers for Disease Control(CDC) in Atlanta, and Jacobson and Kasworm(1986) estimated the incidence after nasal surgery to be 16.5 per 100,000, which in fact is higher than the incidence in women of menstrual age using intravaginal tampons[1]. There was a report that among the 1700 functional endonasal sinus surgery(FESS) procedures, 3 cases were complicated by classic TSS, with 2 additional patients having a postsurgical course compromised by a milder degree of TSS[9]. Rabb MG et al. conducted a retrospective review of all cases of postoperative TSS(PTSS) occuring in 2 community hospitals from 1981 to 1993, following 390,000 surgical procedures, there were 12 cases of PTSS among the procedures reviewed(0.003%): all cultures yielded Staphylococcus aureus, and all tested isolates were susceptible to methicillin or cephalothin, mean time from surgery to onset of symptoms was 4 days, and all patients had sudden onset of fever(mean maximal temperature was 40 degrees C), and all patients displayed a rash, most in a truncal sunburn pattern, 11 of 12 patients desquamated. All patients required vigorous fluid

resuscitation, and all patients survived, no correlation could be demonstrated between PTSS and patient age, sex, preoperative skin preparation or antibiotics, members of surgical team, or duration of procedure, so no risk factors for PTSS have been identified[10]. TSS cases following nasal surgery have been associated with nasal packing, mucosal barrier violation, prior Staphylococcus aureus phage 1 colonization, as well as low antitoxin antibody levels, but TSS continues to be encountered more frequently with the head and neck areas as sources of the toxin[7], in head and neck surgery practice it is most commonly noted following nasal packing, and it has been thought that nasal packing is in some ways analogous to the use of tampons for menstrual hygiene[1,3,11-13], and topical or systemic antibiotics doesn't have a demonstrable protective effect. In addition, TSS is reported with increasing frequency in plastic surgical procedures such as rhinoplasty[14], augmentation mammoplasty, liposuction[15], and chemical peeling[2], reconstructive breast surgery with musculocutaneous flaps have also been reported[16], and there were some unusual case reports with minor skin cut[17], using external fixator[4], odontogenic origin[18], and some recurrent cases[16].

It is an acute illness with four major criteria: involvement of multiple organ systems, fever greater than 38.9 degrees C, hypotension or shock, and rash with subsequent desquamation. TSS usually occurs in the immediate postoperative period within 24-48 hours, and presenting symptoms include high fever, sore throat, diarrhea, nausea, and vomitting progressing to hypotension, oliguria with renal dysfunction, conjuntival hyperemia, and an erythematous rash over the trunk, abdomen, and extremities. But there was a report of unusual cases of delayed onset where TSS developed 5 days to 5 weeks postoperatively[8,19]. The clinical characteristics of non-menstrual TSS are identical to those of menstrual TSS. The diagnosis is based mainly on clinical signs and the isolation of toxin-producing Staphylococcus aureus strains[20].

TSS is not a septicemia, but a toxemia, and is known to be a complication of infections by group A beta-hemolytic streptococci(GABHS) and Staphylococcus aureus, and the most extensively described pathogenesis involves a focus of specific Staphylococcus aureus strains capable of producing an exotoxin(toxic shock syndrome toxin-1:TSST-1). Vaginal Staphlococcus aureus infections, particularly those occuring during menstruation, account for most cases of TSS in women of reproductive age, among those patients with onset during menstruation, tampon use has been identified as the most important risk factor, the use of selected superabsorbent brands and styles has been associated with an increased risk of TSS[9]. Strains of group A streptococci isolated from patients with invasive disease have been predominantly M types 1 and 3 that produce pyrogenic exotoxin A or B or both. Staphylococcus aureus strains can also produce various other enterotoxins that may be involved in the pathogenesis of TSS, and the pathogenetic importance of the toxins is supported by the antibody titers in TSS patients: more than 80% of healthy adults show high levels of antibody titers, whereas 90% of TSS patients exhibit low levels in the acute phase followed by a significant increase during convalescence. It is not clear whether the toxins cause TSS by a direct effect or by release of mediators due to their function as superantigens. The three-dimensional structure of a Staphylococcus aureus superantigen(TSST-1), complexed with a human class II major histocompatibility molecule(DR1), was determined by x-ray crystallography. The TSST-1 binding site on DR1 overlaps that of the superantigen Staphylococcus aureus enterotoxin B(SEB), but the two binding modes differ, whereas SEB binds primarily off one edge of the peptide binding site of DR1, TSST-1 extends over almost one-half of the binding site and contacts both the flanking alpha helices of the histocompatibility antigen and the bound peptide, and this difference suggests that the T cell receptor(TCR) would bind to TSST-1:DR1 very differently than to DR1:peptide or SEB:DR1. It also suggests that TSST-1 binding may be dependent on the peptide, though less so than TCR binding, providing a possible explanation for the inability of TSST-1 to competitively block SEB binding to all DR1 molecules on cells(even though the binding sites of TSST-1 and SEB on DR1 overlap almost completely) and suggesting the possibility that T cell activation by superantigen could be directed by peptide antigen[21].

TSS had high levels of tumor necrosis factor alpha and interleukin-6 but no interleukin-1 or interleukin-2 in the serum, intravenous administration of gamma-globulin coincided with clinical improvement and with reduction of the levels of tumor necrosis factor alpha and interleukin-6, and it suggests that streptococcal pyrogenic exotoxins trigger synthesis of tumor necrosis factor alpha and interleukin-6 in vivo, and intravenously administered gamma-globulin may down-regulate the cytokine response[22].

It was suggested that an association between the use of nonsteroidal antiinflammatory drugs(NSAIDS) and the progression of invasive group A streptococcal infections to shock and multiorgan failure, and there is a biochemical rationale that could support this theory. Though NSAIDS are frequently used to relieve pain or reduce fever, they also attenuate granulocyte functions such as chemotaxis, phagocytosis, and bacterial killing. In addi-

tion, findings in recent studies involving human volunteers injected with endotoxin suggest that pretreatment with NSAIDs enhances production of tumor necrosis factor, which leads to higher blood levels of this cytokine, probably by preventing feedback inhibition by prostaglandin E2. Thus, NSAIDs may contribute to the sudden onset of shock, organ failure, and aggressive infection by inhibiting neutrophil function, augmenting cytokine production, and attenuating the cardinal manifestations of inflammation[23].

Treatment of TSS is eradication of the infective focus, aggressive support of vital functions with intravenous fluids and catecholamines, and parenteral antistaphylococcal antibiotics, and continuous hemodiafiltration can be used in case of oliguria, and the prophylactic antibiotics are not necessarily recommended. Other therapeutic measures such as the elimination of toxins by plasma separation or the administration of antobodies or gamma-globulins are subjects of investigation with no general recommendations at this time[20], but intravenous immunoglobulin can be administered when signs and symptoms are worsening while the patient is receiving conventional therapy, within hours of the administration of the intravenous immunoglobulin, the patient may experience dramatic clinical improvement, and this response suggests a possible therapeutic benefit of intravenous immunoglobulin in TSS[8].

TSS is a rapidly developing disease, which may be lethal if not recognized and treated early, although complete recovery is often the case. So early recognition is the hallmark of successful treatment, and an overlooked TSS may lead to a fatal outcome[24]. The clinical course depends on the extent of the organ failure due to decreased tissue perfusion during hypotension. Severe cases are accompanied by multiple organ-system failure including impaired renal function, which is reversible in nearly all cases. Respiratory failure ranges from interstitial and alveolar edema to adult respiratory distress syndrome(ARDS) in 10% of cases: severe DIC(disseminated intravascular coagulopathy) is seen in 10-15%, another severe clinical complication is cardiac insufficiency[20]. Nonmenstrual TSS is apparently associated with a higher mortality rate than TSS associated with menstruation[16], and familiarity with the pathogenesis and clinical presentation of TSS may help achieve early diagnosis and prompt appropriate intervention.

Ⅳ. SUMMARY

Toxic shock syndrome is an acute disease involving multiple organs. I described here a case of TSS associated with intranasal packing. Four major criteria, which are involvement of multiple organ systems, fever, hypotension or shock, and rash with subsequent desquamation should be fulfilled for the diagnosis. The exact pathogenesis is not well understood, however it is thought to be due to the effects of an enterotoxin produced by certain strains of staphylococcus aureus.

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