**Name of Journal:** *World Journal of Clinical Cases*

**Manuscript NO:** 65640

**Manuscript Type:** CASE REPORT

**Streptococcal toxic shock syndrome after hemorrhoidectomy: a case report**

Lee CY *et al*. STSS after hemorrhoidectomy

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**Author contributions:** Lee CY wrote the manuscript; Lee YJ and Chen CC participated in patient care; Chen CC collected the data; Kuo LJ reviewed and edited the manuscript.

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**Received:** March 12, 2021

**Revised:** April 22, 2021

**Accepted: October 14, 2021**

**Published online:**

**Abstract**

BACKGROUND

Streptococcal toxic-shock syndrome after hemorrhoidectomy is rare but may be catastrophic. Group A streptococci have produced various surface proteins and exotoxins due to genetic changes to fight the human body’s immune response. Though life threatening infection after hemorrhoidectomy rarely occurs, all surgeons should be aware of the potential complications of severe sepsis after hemorrhoidectomy and keep in mind their clinical presenting features in order to diagnose early and administer appropriate and effective therapeutic drugs early.

CASE SUMMARY

Here, we present a case of a 56-year-old man with a painful thrombotic external hemorrhoid who presented to our outpatient department for management. There was no history of systemic diseases or recent disease infection. Hemorrhoidectomy was suggested and performed. After surgery, the patient developed hypotension, tachycardia, fever with chills and renal function impairment on day 2 post-operation. The clinical condition progressed to severe septic shock and metabolic acidosis. The patient responded poorly to treatment and expired after 1 d even with use of extracorporeal membrane oxygenation. The results of the blood and wound cultures showed group A streptococcus pyogenes.

CONCLUSION

Although extremely uncommon, all surgeons should be aware of these potential life-threatening septic complications and alert to the presenting features for patients receiving hemorrhoidectomy.

**Key Words:** Hemorrhoid; Sepsis; Streptococcus pyogenes; Streptococcal toxic shock syndrome; Case report

Lee CY, Lee YJ, Chen CC, Kuo LJ. Streptococcal toxic shock syndrome after hemorrhoidectomy: a case report. *World J Clin Cases* 2021; 0(0): 0000-0000 URL: https://www.wjgnet.com/2307-8960/full/v0/i0/0000.htm

DOI: https://dx.doi.org/10.12998/wjcc.v0.i0.0000

**Core Tip:** Group A *Streptococcus* (GAS; *Streptococcus pyogenes*) causes a broad spectrum of infections, including skin and soft tissue infections, tonsillitis, postpartum endometritis, puerperal sepsis, necrotizing soft tissue infection, and toxic shock syndrome (TSS). Though GAS infection and streptococcal TSS rarely happen after hemorrhoid treatment, all surgeons should be aware of the potential complications of severe sepsis after hemorrhoidectomy and keep in mind their clinical presenting features in order to diagnose early and administer appropriate and effective therapeutic drugs early.

**INTRODUCTION**

Streptococcal toxic shock syndrome (STSS) occurs as a serious complication of invasive group A streptococcus (GAS) and 30%-70% of patients die in spite of aggressive treatments[1-3]. The criteria to define STSS include the isolation of GAS from a normally sterile site, hypotension, and involvement of at least two organ systems (renal impairment, coagulopathy, abnormal liver function, acute respiratory distress syndrome, skin rash, or soft tissue necrosis)[4]. Though GAS infection and STSS rarely happen after hemorrhoid treatment, catastrophic complications indeed do occur. All surgeons should be aware of the potential complications of severe sepsis after hemorrhoidectomy. The GAS infection following hemorrhoidectomy should be considered even when there is little to find on examination and the presenting features of STSS should be kept in mind.

**CASE PRESENTATION**

***Chief complaints***

The 56-year-old man was seen in our outpatient department because of sudden onset severe anal pain.

***History of present illness***

The patient had a history of external hemorrhoids for 20 years and denied any systemic diseases. This time, he visited our outpatient department because of sudden onset severe anal pain and bleeding.

***History of past illness***

The patient had a free previous medical history.

***Personal and family history***

No significant personal or family history was identified.

***Physical examination***

Rectal examination showed a thrombosed external protruding hemorrhoid and surgery was suggested because of acute pain. Preoperative blood pressure was 108/96 mmHg, the pulse was 59 beats per minute, the oxygen saturation was 100% under ambient air at rest and other examination results were normal.

***Laboratory examinations***

Routine laboratory examinations were within normal limits.

***Imaging examinations***

Routine chest x-ray examination was normal.

**FINAL DIAGNOSIS**

Acute thrombotic hemorrhoids with bleeding and severe anal pain.

**TREATMENT**

The patient received hemorrhoidectomy immediately after his outpatient department visit.

**OUTCOME AND FOLLOW-UP**

The patient received hemorrhoidectomy immediately after his outpatient department visit. Hemorrhoidectomy was performed smoothly. After the operation, the patient was sent back to the ward of general surgery and vital signs were similar to those from preoperative examination. On the morning of day 1 post-operation, his temperature was 36.4 oC, blood pressure was 85/50 mmHg, and pulse was 83 beats per minute. On examination, the patient had good spirits and fair activity without any discomfort except for moderate wound pain (VAS = 5). The wound showed mild swelling and no pus or bloody discharge. Mefenamic acid 250mg QID PO and Pethidine 50mg PRN were prescribed for pain relief. Increased pulse rates to 108 beats per minute and persistent hypotension (76/54 mmHg) were noted on day 2 post-operation. The patient appeared well and denied having dizziness, chills, weakness, poor appetite or low urine output. Sepsis, stress ulcer induced gastrointestinal bleeding and dehydration were first considered but the patient denied tarry stool and epigastric discomfort. Due to the hypotension, we planned to give intravenous fluid, but the patient refused to establish an intravenous line because of fear of pain; thus, water intake was encouraged and vital signs were closely monitored. On the morning of day 3 post-operation, the patient had fever to 38.6 oC with mention of chills. His blood pressure was 70/42 mmHg, his pulse was 124 beats per minute, and his oxygen saturation was 97% under ambient air. Two sets of blood cultures and laboratory tests were immediately obtained. The laboratory result revealed leukocytosis (white blood cell, 13100/µL), elevated C-reactive protein (33.12 mg/dL), blood urea nitrogen (40.6 mg/dL), creatinine (2.6 mg/dL) and decreased platelets (81000/µL). Intravenous fluid and antibiotics (Cefmetazole, 1g, Q8H) were given due to suspected sepsis. We rechecked vital signs after 2 h, and found his blood pressure was 155/110 mmHg, his pulse was 88 beats per minute, and his oxygen saturation was 95% under ambient air. The patient started to complain of general soreness and discomfort. After 6 h, the patient underwent a consciousness change, as noted by his family. On examination, we found a body temperature of 36.1 oC, blood pressure of 68/51 mmHg, pulse of 144 beats per minute, respiratory rate of 27 per minute and oxygen saturation of 95% under ambient air. Immediate intravenous fluid resuscitation was performed and artery blood gas analysis revealed pH 7.32, pCO2 16.9 mmHg, pO2 118.9 mmHg, and HCO3 8.5 mmol/L. The patient was sent to the intensive care unit and an endotracheal tube was put in place because of low oxygen saturation and tachypnea. Sodium bicarbonate was given and due to persistent metabolic acidosis, continuous venous-venous hemofiltration was arranged. Sudden cardiac arrest happened after continuous venous-venous hemofiltration. Cardiopulmonary resuscitation was performed and emergent extracorporeal membrane oxygenation (ECMO) was applied to sustain circulation and tissue perfusion. Although there was neither significant swelling nor pus discharge of the anal wound, a swab culture from the deep wound was obtained. The patient experienced cardiac arrest again 2 h after ECMO placement and expired. The blood and wounds culture both yielded *Streptococcus pyogenes.*

**DISCUSSION**

Hemorrhoids are a common disease with the prevalence of 4.4%-11% throughout the population[5,6]. Hemorrhoidectomy is an efficient and advantageous way to cure hemorrhoids, especially when patients fail to respond to conservative measures[7]. The postoperative complications of hemorrhoidectomy include fecal impaction, infection, urinary retention, bleeding and anus stenosis. The overall postoperative complications rate is approximately 3% and septic complication following treatment of hemorrhoids is rare[8-10]. The predominant organisms isolated in those patients with septic complications are Escherichia coli and Bacteroides[9,10]. Only one study to date has reported Streptococcus pyogenes induced necrotizing fasciitis and toxic shock syndrome after hemorrhoidectomy similar to the case we presented[11].

Group A *Streptococcus* (GAS; *Streptococcus pyogenes*) causes a broad spectrum of infections, including skin and soft tissue infections, tonsillitis, postpartum endometritis, puerperal sepsis, necrotizing soft tissue infection, and toxic shock syndrome (TSS)[12]. Invasive group A streptococcal (invasive GAS) disease is relatively rare but is often complicated by shock and multiorgan failure and is associated with high mortality and morbidity[1-3]. The incidence of invasive GAS diseases is high in adults > 50 years of age and young children and most patients are not immunocompromised[2,3,13]. Streptococcal TSS (STSS) occurs as a serious complication of invasive GAS disease in approximately one-third of cases and 30% to 70% of patients die in spite of aggressive treatments[14,15]. The criteria to define STSS includes the isolation of GAS from a normally sterile site, hypotension, and involvement of at least two organ systems (renal impairment, coagulopathy, abnormal liver function, acute respiratory distress syndrome, skin rash, or soft tissue necrosis) (Table 1)[4]. Our patient fulfilled the diagnostic criteria of confirmed STSS, without the presentation of necrotizing fasciitis. The pathogenic mechanisms of STSS are not completely understood because each is the culmination of complex interactions between the defense abilities of the human host and specific virulence factors of GAS[16]. Streptococcal pyrogenic exotoxins and other proteins act as superantigens and trigger excessive T cell response and secretion of massive inflammatory cytokines producing capillary leakage and arterial hypotension[17]. Predisposing factors for invasive GAS are minor trauma, including injuries resulting in hematoma, bruising, muscle strain, recent surgery, viral infection (*e.g.*, influenza, varicella, *etc.*), alcohol abuse, immunosuppression, chronic lung disease, intravenous drug use, heart disease, diabetes, cancer, and recent child birth[18]. Risk factors identified in our patient included thrombosed hemorrhoid, recent surgery (hemorrhoidectomy) and age > 50 years.

Bacteria do colonize anal wounds following open hemorrhoidectomy[19]. *E. coli,* followed by *Staphylococcus aureus* and *Staphylococcus epidermidis* are the most dominant organisms[19]. However, overt wound infection after routine hemorrhoid surgery is rare (1.4%) and routine prophylactic antibiotic use is unnecessary[20,21]. In thrombosed hemorrhoid and septic complications after hemorrhoid treatment, *E. coli* and anaerobes are the predominant pathogens[10,11,22]. In our patient, a thrombosed hemorrhoid and open hemorrhoidectomy provided a portal of entry for GAS. This could explain local or indeed distant sepsis.

The systemic review of McCloud *et al*[9] reported 38 patients with life threatening sepsis following treatment for hemorrhoids. Of these, all were well prior to surgery with the exception of two (one was a case of human immunodeficiency virus infection and the other had drug-induced agranulocytosis). The predominant organisms isolated in these patients were *Escherichia coli*, *Bacteroides fragilis*, and *Staphylococcus aureus.* Only one study to date reported *Streptococcus pyogenes* induced STSS after hemorrhoidectomy[11], similar to the case presented here. In the literature reviewed by McCloud *et al*[9], 10 patients died and seven of them had initial presentations of septic shock; conversely, only 2 of the 28 survival cases developed septic shock at initial presentation. In our case, the most important presentation was septic shock without local wound necrosis. The fierce progression of GAS infection related to TSS calls for early aggressive intervention due to the high mortality and morbidity rate[14,15].

**CONCLUSION**

Though GAS infection and STSS rarely happen after hemorrhoid treatment, catastrophic complications indeed do occur. All surgeons should be aware of the potential complications of severe sepsis after hemorrhoidectomy. The GAS infection following hemorrhoidectomy should be considered even when there is little to find on examination and the presenting features of STSS should be kept in mind.

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**Footnotes**

**Informed consent statement:** Informed written consent was obtained from the patient for publication of this report and any accompanying images.

**Conflict-of-interest statement:** The authors declare that they have no conflict of interest.

**CARE Checklist (2016) statement:** The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

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**Manuscript source:** Unsolicited manuscript

**Peer-review started:** March 12, 2021

**First decision:** April 13, 2021

**Article in press:**

**Specialty type:** Surgery

**Country/Territory of origin:** Taiwan

**Peer-review report’s scientific quality classification**

Grade A (Excellent): 0

Grade B (Very good): B

Grade C (Good): 0

Grade D (Fair): 0

Grade E (Poor): 0

**P-Reviewer:** Xavier-Elsas P **S-Editor:** Gong ZM **L-Editor:** Filipodia **P-Editor:** Gong ZM

**Table 1 Clinical criteria for streptococcal toxic-shock syndrome**

|  |
| --- |
| **Hypotension defined by a systolic blood pressure less than or equal to 90 mmHg for adults or less than the fifth percentile by age for children aged less than 16 years** |
| Multiple organ involvement characterized by two or more of the following: |
| Renal impairment: creatinine ≥ 2 mg/dL (≥ 177 μmol/L) for adults or ≥ wice the upper limit of normal for age. In patients with preexisting renal disease, > twofold elevation baseline creatinine levels |
| Coagulopathy: platelets ≤ 100000/mm3 (≤ 100 × 106/L) and/or disseminated intravascular coagulation, defined by prolonged clotting times, low fibrinogen level, and the presence of fibrin degradation products |
| Liver abnormalities: alanine aminotransferase, aspartate aminotransferase, or total bilirubin levels ≥ twice the upper limit of normal for the patient’s age. In patients with preexisting liver disease, a > twofold increase over baseline levels |
| Acute respiratory distress syndrome: defined by acute onset of diffuse pulmonary infiltrates and hypoxemia in the absence of cardiac failure or by evidence of diffuse capillary leak manifested by cute onset of generalized edema, or pleural or peritoneal effusions with hypoalbuminemia |
| A generalized erythematous macular rash that may desquamate |
| Soft tissue necrosis, including necrotizing fasciitis or myositis, or gangrene |
| Laboratory criteria for diagnosis: |
| Isolation of group A streptococcus |