

Superficial Surgical Site Infections - An Overview of Port - Site Infections in Gynecologic Laparoscopic Surgery

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1. Abstract

Laparoscopic Surgery has brought about a revolution in the approach to various diseases demanding surgical intervention. The paradigm shifts towards laparoscopic surgery compared to conventional surgery has attained acceptance both amongst the patient and operating surgeon. However, this technique is not bereft of its set of complications. Amongst them the least talked about is Port site infection. It's an infrequent complication which cannot be ignored, as this may undermine the benefits of the laparoscopic technique. Moreover, the complication is not life threatening but definitely leaves an err for the surgeon as it affects the postoperative quality of life and spoils the aesthesis of the surgery. Breach in asepsis resulting in bacterial infection and the rapidly emerging multidrug resistant atypical mycobacterium are a constant threat. This article focuses on the diagnosis, current available treatment options and measures that can be taken to prevent its occurrence. The article emphasis the need on more research work and guidelines to be established pertaining to the techniques required for sterilization of laparoscopic instruments.

2. Introduction

The growth in health care technology have opened avenue and empower surgeons to treat surgically with limited invasiveness. The greatest example is minimal access surgery (MAS) or Laparoscopic surgery. This advancement in surgical technique caused a paradigm shift in the approach to modern surgery. Laparoscopic surgery gained popularity both amongst the surgeons and patient. The cosmetically small and almost invisible scar, less pain, ear-

ly ambulation and discharge from the hospital and early return to work, minimizing financial & social burden are the advantages to name a few. The popularity of the laparoscopy is on rise even amongst the surgeons – the magnified view of the operative site and better perception and appreciation of the anatomy, the captivating view of the operation on large screens encourages young surgeons to learn surgery laparoscopically. This is in stark contrast to open surgery where one or two assistants of the main operative surgeons would struggle to catch a view of the operative site, concentrating more on retracting and giving adequate exposure. Philips Mouret reported the first laparoscopic cholecystectomy in 1987, since then approach has been adapted for many surgical procedures including appendectomy, hernioraphy, colonic surgery, gastric, urologic and gynecological surgery [1-5]. This has been due to increased interest among surgeons to learn the technique as a part of advancement in technology and increasing acceptance of MAS by the patients, which has lead to increasing use of laparoscopic surgery.

Laparoscopic surgery comes with its own array of complications which include entry point damage to vital organs, vascular injury, injury due to energy source to name a few. One such complication, which is less talked about, is the port site infection (PSI). PSI erodes all the advantages of laparoscopic surgery in a blink. All may have been correct with the surgical procedure, but a PSI with its indolent and nagging infection loses the confidence of patient on the operating surgeon. This causes a significant increase in the morbidity, hospital stay and financial loss to the patient. The very purpose of laparoscopic surgery to achieve utmost cosmesis,

a PSI turns this into an unsightly wound, and the quality of life of patients is seriously affected. The current article is to emphasize the importance of this issue, review the incidence, clinical presentation, etiopathogenesis, management and methods of prevention of PSI in laparoscopic surgery.

3. Category of Infections

The Centers for Disease Control and Prevention (CDC) defines a SSI as an infection occurring within 30 days of an operation occurring in one of the three locations:

Superficial at the incision site, deep at the incision site or in other organs or spaces opened or manipulated during an operation [6].

a. Superficial Incisional SSI: Infection involves only the skin and subcutaneous tissue of the incision and at least one of the following:

- Purulent discharge with or without laboratory confirmation, from the superficial incision;
- Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision;
- At least one of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat and superficial incision being deliberately opened by surgeon, unless incision is culture negative.

b. Deep Incisional SSI: The infection involves deep soft tissue (e.g fascia, muscle) of the incision and at least one of the following:

- Purulent drainage from the deep incision but not from the organ /space component of the surgical site.
- A deep incision which spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (38°C), localized pain, or tenderness, unless incision is culture negative;
- An abscess or other evidence of infection involving the deep incision being found on direct examination, during reoperation, or by histopathological or radiologic examination.

c. Organ / Space SSI Include Adnexal Infections and Pelvic Abscesses: Infection involves any part of the anatomy (e.g., organs and spaces) other than the incision that was opened or manipulated during an operation and at least one of the following:

- Purulent discharge from a drain that is placed through a stab wound into the organ or space;
- Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ or space;
- An abscess or other evidence of infection involving the organ /space that is found on direct examination, during reoperation, or by histopathologic or radiologic examination

tion

PSI is a type of SSI but limited to Laparoscopic surgery. Wounds are classified as (CDC criteria for SSI 2015) [7] into

- **Clean:** A surgical wound that is neither exposed to any inflamed tissue nor has breached the gastrointestinal, respiratory, genital or uninfected urinary tract;
- **Clean:** Contaminated: Surgical wounds where there is controlled entry into the gastrointestinal, respiratory, genital or uninfected urinary tract with minimal contamination.
- **Contaminated:** Fresh wounds related to trauma, surgical wounds with major breach in sterile technique or gross contamination from the gastrointestinal tract, and incisions through non purulent inflammatory tissues
- **Dirty and Infected:** Old wounds following trauma having devitalized tissue and surgical procedure performed in the presence of active infection or visceral perforation.

Most of the surgical procedures done by laparoscopy belong to class 1 and 2. PSI is a type of SSI (Superficial Surgical Site Infection) but limited to Laparoscopic surgery. There is higher incidence of superficial incisional SSI as compared to that of deep incisional SSI in laparoscopic surgery [8].

3.1. Port site infection

The PSI presents in the form of seropurulent discharge from the port site with inflammation of surrounding skin or symptoms related to organ / space infection. The human body harbors a variety of microbes which can cause infections. When the host systemic immunity is suppressed as a result of any disease, or disruption of the integrity of the skin or mucous membranes or following uptake of immunosuppressant or steroids, patients own commensal microbial flora may cause infection. The surveillance for PSI poses a challenge, as the patients are usually discharged in a day or two, and the port site infections manifest later [9-10]. In the absence of such surveillance, it is estimated that almost one-third of all the SSI go unidentified. No study has been done to see the incidence of PSI in gynecologic surgery but those done in gastrointestinal surgery can be used to ascertain the multitude of the menace. The reported incidence of PSI in various studies is variable. In fact, the actual incidence of PSI is higher than revealed (Table-1).

Table 1: Studies showing frequency of port site infection following laparoscopic Cholecystectomy

Study	Type	Year	Patients	Frequency of infection
Karthik et al (11)	Prospective	2013	570	10 (1.8%)
Mir et al (12)	Prospective	2013	675	45(6.7%)
Yanni et al (13)	Prospective	2013	100	4(4%)
Shindholimath et al (14)	Prospective	2003	113	7(6.3%)

As stated earlier for most SSI, the source of pathogens is the endogenous flora of the patient's skin, which consists of predominantly aerobic gram positive cocci. However gynecologic procedures pose a unique challenge in those potential pathogenic microorganisms may come from the skin or ascend from the vagina and endocervix and gain access to the incision site. The endogenous vaginal flora is a complex and dynamic mix of pathogenic and nonpathogenic bacteria composed of facultative and obligate anaerobic gram positive and gram negative species. Therefore, gynecologic SSI are more likely to be polymicrobial and may include gram negative bacilli, enterococci, group B streptococci, and anaerobes as a result of infection ascending up from vagina and perineum.

4. Risk Factors for PSI

The risk factors for SSI may be applicable to PSI: An increase in preoperative stay in hospital of more than 2 Days for open surgical procedures have been reported by Lilani et al [9]. Same study reported no infection in surgeries of less than 30 minutes' duration. There was a significant increase in SSI for operations of prolonged duration. Obesity, prophylactic antibiotics, and drains have no effect on the rate of SSI following laparoscopic cholecystectomy [15] Factors like emergency / multiprocedure surgery and surgery in acutely inflamed organs adversely affect the rate of SSI [16-17]. The risk of SSI increases in patients with history of drug abuse, diabetes, malnutrition, long hospitalization, preoperative colonization of staphylococcus aureus, or following excessive hemorrhage requiring blood transfusion [18-19]. PSI are more common in the entry or umbilical port [11]; the infection rate may depend upon the port through which the specimen is extracted. The infected specimen may be removed in an end bag in order to prevent wound infection and accidental spillage of contents.

4.1. Microbial flora causing PSI IN LS

PSI occurs due to exposure of surgical wound to infectious agents who may be endogenous or exogenous. The endogenous infections are the result of microbes present in patients' skin, mucous membrane or any other viscera. The source of exogenous flora may be from any contaminated source present in the sterile surgical field including surgeon and team, instruments, room air etc [20]. Clean surgical wounds usually harbor Staphylococcus aureus which may have an exogenous origin or endogenous in origin. Infections in clean contaminated, contaminated, and dirty surgical wounds are polymicrobial, resembling the flora of the target organ [21] Kowhar et al reported Staphylococcus aureus (37 %) as the most common isolate in causing superficial SSI. [22] Klebsella sp being the commonest offending agent in deep SSI [22]. The culprit of PSI is usually the hospital acquired skin flora. Organisms causing deep SSI usually are endogenous in origin or may be the skin commensals which reach the fascia or muscle layers through surgical incision [23]. In a study by Wolcott et al [24] Bacteroids fragilis was the predominant flora (60%) which originate from intraoperative

visceral spillage. Mir et al [12] found pseudomonas (42%) as the commonest offending organism. Several reports have established the role of rapid growing mycobacterium (RGM), particularly *M. fortuitum* and *M. Chelonae* together termed as *m fortuitism - chelonae* complex that infect both humans and animals [25]. The endospores of this non tuberculous mycobacterial (NTM) complex are usually considered saprophytes which colonize in sewage, soil and even tap water. These often cause localized skin infections 3-4 weeks post surgery [26, 27]. The NTM complex can cause disseminated disease in immunosuppressive conditions. These atypical mycobacterium have a predilection to involve the skin and subcutaneous tissue. *M. Chelonae* and *M. abscessus* have similar characteristics, and hence together were called as *M. Chelonae/abscessus* group. Vijayraghav et al [28] reported an outbreak of laparoscopic PSI due to *M. chelonae* in patients operated by them. The contaminating source was water which was used for washing the instruments after chemical disinfection.

5. Types of PSI

PSI are of two types based on the timing. The most common type manifest early, within a week of the surgical procedure. Gram positive or negative bacteria are the usual offending organisms which are found in the skin or infected surgical site. They usually respond well to broad spectrum antibiotics. The other variety is the delayed type which is caused by rapid growing atypical mycobacterium species, and has an incubation period of 3-4 weeks and respond poorly to antimicrobial agents [29]. Based on the agents the PSI may be due to non-mycobacterial isolates or Mycobacterial isolates.

5.1. Clinical presentation of PSI

Wound discharge and erythema around the port site are the most common presentation of non-mycobacterial infection occurring within a week of the surgery. They are usually limited to the skin and subcutaneous tissue [11-14]. There may be surrounding tissue inflammation with pain or tenderness and low grade fever [20]. The delayed type of PSI is usually caused by mycobacteria and manifest nearly a month after surgery, in the form of persistent multiple discharging sinuses or lumps / nodules, not responding to antibiotics. There may be pigmentation and induration at the port site starting in a single port and spreading to others. The delayed type manifest as one of the following are five clinical stages I [29].

First stage: A tender nodule appears near of the port site, and its usual appearance around 4 weeks following the surgery.

Second stage: Increase in the size of the nodule, increase tenderness of the site with signs of inflammation later resulting in the formation of a discharging sinus.

Third stage: Reduced pain sensation due to rupture of nodule with discharge of the purulent material and necrosis of the skin surrounding the port site.

Fourth stage: White or serous discharge which persists for long

time.

Fifth stage: Hyper pigmentation of the skin near the sinus and appearance of multiple nodules at different places.

5.2. Management

Daily dressing, cleaning of the wound and an empirical course of antibiotics started. The early type of PSI usually resolves with antibiotics while for unresolved or late onset PSI gram staining and culture sensitivity of the discharge from wound are taken. Drainage and debriment of the wound required for wound healing. Unresolved and unattended PSI may lead to life threatening necrotizing fasciitis of the abdominal wall following laparoscopic surgery. Significant erythema and wound discharge from port site with fever are features of necrotizing fasciitis. This condition requires aggressive management. In delayed PSI, a study by Chaudhari et al [29] have shown raised markers of acute infection levels without leukocytosis and a normal differential count in patients with atypical mycobacterium infection thus posing confusion in diagnosis and management. Tissue or fluid obtained by biopsy or aspiration needs to be processed for gram staining, culture in Lowenstein–Jensen medium and BACTEC technique. Isolation of the atypical mycobacteria by tissue culture takes time to grow. The

most accurate method of identification of *M. chelonae* is detecting resistance to polymycin B disc [30]. The culture of pus does not grow any bacteria. The diagnosis is based on the clinical signs and symptoms and high level of suspicion [31]. In case of growth of the organism, the organisms are confirmed by either biochemical reactions or the more recent nucleic acid amplification tests. Other investigations like tissue culture, real time PCR, and serology for antitubercular antibodies can support the diagnosis [30]. The histopathological examination at times may show chronic granulomatous inflammation, with features of epithelioid cells and lymphoplasmacytic infiltration.

PSI with atypical mycobacteria is difficult to treat as it responds poorly to anti tubercular treatment. Second line ATT including macrolides, quinolones, tetracycline and aminoglycosides may be used alone or in combination to achieve optimal results [30-32]. Macrolides including clarithromycin are the only group of antimicrobials active against *M. chelonae* and *M. abscessus* [33]. *M. fortium*–*chelonae* complex has shown resistance to antibiotics because of mutation in the porin channels present in the bacterial wall, which is the site of entry of antibiotics for antimicrobial activity [29] (Table 2) shows different drugs used in Mycobacterium infection in port site location.

Table 2: Different antibiotics effectively used against mycobacterial sp. in port site infections

Ref	Type of study	Mycobacterial isolates	Treatment Given
Ramesh et al (32)	Case series	<i>M. tuberculosis</i>	Standard firstline treatment with antitubercular regimen Rifampacin , isonazide , pyrazinamide and ethambutol for 2 months followed by rifampacin and isonazide for 9 months
Chaudhari et al(29)	Case series	atypical mycobacterial	Clinically suspected atypical mycobacterial, Clarithromycin & Ciprofloxacin for 28 days to 3 months . For persistent local nodule
Chaudhari et al(29)	Case report	<i>M. Chelonae</i>	Amikacin 750 mg/day and azithromycin (500 mg BD x 2 weeks followed by Linezolid 500 mg and azithromycin 500mg BD for 6 weeks
Duarte et al (34)	Case series	<i>M . massiliense</i>	Sensitive to amikacin and clarithromycin , but resistant to ciprofloxacin cefotaxim and Doxycycline
Sethi et al (32)	Case report	<i>M. flavescens</i>	Ofloxacin and amikacin for 6 months
Shah et al (37)	Case series	<i>M. fortuitum</i>	Clarithromycin and ciprofloxacin (500 mg each twice daily) for 6-9 months
Rajini et al (36)	Case report	<i>M . chelonae</i>	Clarithromycin 500mg BD & Doxycyclin 100 mg OD for 4 weeks

5.3. Prevention of PSI

Infections are prone in any types of surgical procedure. In clean and clean contaminated wounds of PSI after laparoscopic surgery, the cause may be contamination. The endogenous source of infection can be avoided by meticulous surgical techniques and retrieval of specimen in sterile endobags. The exogenous source of infection is too many and that is the cause which needs to be addressed. Non tuberculous mycobacteria may be present in water or soil which can contaminate hospital equipment's. Inadequate or improper sterilization protocol of laparoscopic instruments is the most common cause of PSI with atypical mycobacteria [29]. This

arises from the fact that most of laparoscopic instruments are not autoclavable because of the heat sensitive outer insulation sheath instruments have multiple joints and crevices, where blood and tissues can collect. Frequent use of instruments without optimal cleaning potentially results in contamination with organisms such as atypical mycobacteria. Endospores in the contaminated instruments get deposited in the subcutaneous tissue, which germinate in 3-4 weeks to produce clinical signs and symptoms [34-36]. Sterilization of laparoscopes for 30 minutes in 2 % alkaline glutaraldehyde solution is recommended. Many a times, paucity of instruments and the patient load may not permit such sterilization for 30

minutes. This may facilitate implantation of organisms leading to infection. Moreover, some of the rapid growing mycobacterium (RGM) can survive in such disinfectant solutions for periods as long as four hours. A study by Lorenaa et al [37] on M massillense BRA 100 strain showed that it is resistant to even higher concentration of glutaraldehyde (GTA, 7%) .Other sterilizing agents like orthohthaldehyde and per acetic acid have been used to substitute GTA for higher level of disinfection . With good efficacy [37].

5.4. Divine rules to be followed to prevent PSI

- Use disposable trocars and instruments and adequately sterilized reusable trocars should be used.
- Use laparoscopic hand instruments which can be autoclaved
- Use of instruments with good ergonomics, limited joints and facility for proper cleaning of debris in crevices.
- Ultrasonic technology is considered best for sterilizing laparoscopic instruments. Use of clean and autoclaved water for cleaning of instruments after dismantling should be encouraged.
- Proper protocols should be followed regarding the concentration, contact time and cycles of use for instrument sterilization with liquid sterilizing agents
- It is desirable to use plasma sterilizers or ethylene oxide in between the consecutive surgery for instrument sterilization.
- Avoiding sharing of instruments with gastic or urologic lap surgeons.
- Avoid spillage of specimen content eg in pyosalpinx, dermoid cyst, tubo ovarian abscess in the operative area.
- Use of non porous sterile retrieval bags or endobags for retrieving the specimen
- Thorough irrigate the port site with normal saline before wound closure.

6. Conclusion

Port site infection can be a frustrating complication in MAS, both for the patient and for the operating surgeon. The rapidly growing multidrug resistant strains are a new threat, which adds fuel to the indolent PSI. Strict asepsis and appropriate sterilization of the laparoscopic instruments is the only answer to this largely overlooked problem of PSI. More research is needed to find out appropriate guidelines for the diagnosis and treatment of this emerging problem.

7. Conflict of Interest

None

8. Sources of Funding

None

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