RISK FACTORS FOR SURGICAL SITE INFECTION AND FEBRILE MORBIDITY FOLLOWING CESAREAN SECTION:

A PROSPECTIVE STUDY

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Précis

Post-Cesarean infection complications compare favorably with those of other reports. Host susceptibility and existing infections are the most important predictors of surgical site infection and febrile morbidity following Cesarean section.

Abstract

Objective: to determine post-Cesarean infection complications and to identify the independent risk factors for surgical site infections.

Methods: a cohort of 969 Cesarean sections from May to August 1997 were prospectively studied. Infections were identified by ward rounds, review of laboratory results and patient follow-up until 30 days after hospital discharge. Risk factors were identified via unconditional multiple logistic regression.

Results: Surgical complication was rare. Febrile morbidity and infection complications were documented in 16.2% and 12.4% of subjects, respectively. Eighty-five subjects developed 95 surgical site infection (9.8%). Eight risk factors are independently associated with post-Cesarean surgical site infections. Identified here are preoperative remote infections (adjusted OR =16.1, 95%CI = 2.1-125.2); chorioamnionitis (aOR = 9.1, 95%CI = 1.8-45.2); preoperative condition of patient (aOR = 5.1 for ASA score ≥ 3, 95%CI = 1.1-23.1); rupture of membrane (aOR= 2.5 for ROM ≥ 24 hours, 95%CI= 1.1-3.1); pre-eclampsia (aOR = 2.2, 95%CI = 1.03-4.7); higher body mass index (aOR = 2.0 for every 5-unit increment, 95%CI = 1.3-3.0); nulliparity (aOR = 1.8, 95%CI = 1.1-3.2); and increased volume of surgical blood loss (aOR = 1.3 for every 100-ml increment, 95%CI = 1.1-1.5).

*{Other five factors were documented as independent predictors of febrile morbidity following Cesareans section. They are chorioamnionitis (aOR = 16.0, 95%CI = 3.1-83.3); preoperative condition of patient (aOR = 13.6 for ASA≥ 3, 95%CI = 2.8-65.7); preoperative fever (aOR = 9.5, 95%CI = 3.8-23.7); preeclampsia (aOR = 2.4, 95%CI = 1.2-4.6); and blood loss (aOR = 1.3 for every 100-ml increment, 95%CI = 1.1-1.5)

Conclusion: Post-Cesarean complications compare favorably with those of other reports. Host susceptibility and exiting infections are important predictors of surgical site infection and febrile morbidity after Cesarean section. High-risk patients should be more carefully monitored and preoperative infection should be properly intensively treated as soon as possible.

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Introduction

The number of abdominal deliveries has been steadily increasing worldwide, making Cesarean section one of the most common major operations nowadays[1]. Maternal morbidity related to infections after Cesarean delivery was eight-fold higher than that after vaginal delivery[2]. Surgical site infection (SSI), accounting for almost one-third of nosocomial infections[3], results in discomfort for obstetric patients with many quantifiable consequences in terms of morbidity, time and money. Total cost, including indirect expenses related to SSI, in the United States may exceed 10 billion dollars annually[4].

Reported rates of post-cesarean SSI vary greatly from 0.3% in Turkey[5] to 18.3% in Saudi Arabia[6]. Rates of only superficial and deep SSI in some settings were surprisingly as high as 25.6% in New Zealand[7] and 36.6% in Canada[8]. Despite numerous investigations, there has been a lack of agreement about the risk factors of SSI. A wide variety of factors affects infection rate differently in different settings. Moreover, confounding variables were not sufficiently controlled in many of those reports. Therefore, we conducted this prospective study to determine the postoperative infection complications and to identify the risk factors for surgical site infection after Cesarean section in the context of multivariate analysis. A better understanding of the predictors may improve our infection control program in reducing the clinical impact of SSI.

Materials & Methods

We carried out a prospective study of a cohort of 969 Cesarean sections in Hungvuong obstetric and gynecological (Ob/Gyn) Hospital in HoChiMinh City, Vietnam. It is a 450-bed tertiary-care Ob/Gyn hospital with an average of 1,300 deliveries and 350 major operations per month. It serves not only the population of 2.5 million women in HoChiMinh city but as a tertiary-level supervisory center for 18 district hospitals and the Ob/Gyn departments of all other hospitals in HoChiMinh city as well.

From May to August 1997, all Cesarean deliveries were recruited into the study. The principal investigator visited each postoperative ward twice weekly and collected all pertinent data. Demographic, putative factors and surgical indications were recorded. The host-related variables included age, residence, parity, body mass index, preoperative stay, existing co-morbidity, prior amniocentesis, rupture of membrane (ROM) duration, and preoperative condition of patient. Surgery-related variables included emergency, Cesarean hysterectomy, surgical duration, wound class, type of anesthesia, type of abdominal incision, experience of surgeon, volume of blood loss, and timing of prophylactic administration of antibiotics. The variable residence was defined as urban or rural. Body mass index was calculated using postpartum weight and height, which were measured at the third-postoperative day by the research assistants. Preoperative stay was the interval in days between hospital admission and

surgery. The existing co-morbidities investigated were diabetes mellitus, remote infection, pre-eclampsia, anemia, and chorioamnionitis. Preoperative condition of patient was assessed by the American Society of Anesthesiologists preoperative assessment score (ASA score)[9]. We modified the classical wound classification to make it more appropriate for obstetric operation. A Cesarean section was classified as class I if there was no ROM nor labor; class II if there was less than 2 hours of ROM without labor or labor of any length with no ROM; class III for ROM greater than 2 hours and class IV for purulent amniotic fluid[10]. ROM duration was the interval in hours between the recorded timing of ROM and the onset of surgical incision. The variable anesthesia was defined as general or epidural. An emergency Cesarean delivery was a section performed for immediate and compelling reasons, which had not been planned well in advance. In contrast, an elective Cesarean section was defined as an operation which had been planned in advance and performed at the scheduled time or sooner if the onset of labor accelerated the time of delivery[11]. We classified experience of surgeon into three levels: supervisor, attending physician, and resident. The volume of blood loss was first calculated by subtracting total irrigation fluid used and amount of amniotic fluid from the total amount of fluid in the suction container at the end of surgery, then adding the amount of blood on the sponges, determined by weight. The true volume of blood loss was finally recorded after subtracting volume of possible blood replacement. The timing of antibiotic

prophylaxis was classified as early (2 to 24 hours before the surgical incision), preoperative (0 to 2 hours before the incision), perioperative (within 3 hours after the incision) and postoperative (more than 3 hours after the incision)[13].

The postoperative patients were monitored for signs of infections. Temperature measurement was done every four, six and twelve hours for the first, second and following postoperative days, respectively. The temperature of patients who manifested fever (temperature ≥ 38.0°C) was obtained every four hours until it was less than 37.5°C in two consecutive measurements. Leukocyte count was routinely performed when a patient's temperature was over 38.5°C. Further laboratory tests such as urine analysis, urine culture, and chest X-ray or wound culture were not performed routinely unless infection was suspected. The surgical sites were re-examined by the research assistants, when the patient returned to the outpatient clinic as scheduled after discharge from the hospital.

Surgical complications included intraoperative hemorrhage necessitating blood transfusion; postoperative hemorrhage; and injury to adjacent organs. Any bleeding event requiring intervention postoperatively was known as postoperative hemorrhage complication. The standard criterion of postoperative febrile morbidity was the presence of an oral temperature higher than or equal to 38.0°C on any two of the first ten days postpartum, excluding the first 24 hours[15]. Postoperative infections were

diagnosed using the Centers for Disease Control and Prevention (CDC) definitions[14,15]. Surgical site infections comprised superficial, deep and space/organ SSI. Endometritis and vaginal cuff infection constituted organ SSI[15]. SSI was identified when there was the presence of either purulent discharge, positive culture, deliberate reopening of the surgical wound, any evidence of abscess, or diagnosis of the attending physician[15].

Data management and analysis were performed using the statistical software EPI INFO version 6.04b (CDC 1997 Atlanta) and STATA version 5.0 (StataCorp. 1997 USA). Potential risk factors of SSI were tested first by univariate analysis. Chi-squared test or Fisher's Exact Test and Student's t test or Mann-Whitney test were used for discrete and continuous variables, where appropriate. A P-value less than 0.05 was considered as a statistically significant level. Odds ratios (OR) and their corresponding 95% confidence interval (95% CI) were computed to indicate the association between putative factors and postoperative SSI. Multiple logistic regression analysis was then performed to obtain an adjusted estimate of the ORs and to identify risk factors independently associated with SSI. Variables which were likely to be associated with the outcome (P value $\leq$ 0.2 from univariate analysis) or those which were considered to be potential confounders were included in the multiple logistic regression model.

Results

During the four-month study period, there were 969 Cesarean sections among 5,181 deliveries, yielding the Cesarean delivery rate of 18.7%. There were five obstetric hysterectomies, two of which were selectively indicated for different extent of placenta increta. More than four-fifths of the sample were free from any co-morbidity (Table 1). Ovarian carcinoma was histologically confirmed in a Cesarean section accompanying oophorectomy. Radical hysterectomy was then performed one month later. Although 19.4% of our subjects experienced repeated Cesarean section, in only 15.4% was previous operation the only indication for the current Cesarean delivery (Table 2). Placenta previa rather than previous Cesarean section was recorded as the indication if this placental abnormality resulted in the need for the current operation. Fetal distress or umbilical cord compression was diagnosed using the evidence of external cardiotocography.

The rate of surgical complications was low. Estimated volume of blood loss over 1000 ml occurred in 15 cases, in which 12 were associated with abnormal implantation of placenta; and blood replacement was indicated for 27 cases (2.8%). There was only one postoperative hemorrhage, which was diagnosed four hours after surgery. Partial bladder injury occurred in two cases of repeated cesarean section; one was complicated by placenta increta.

We documented 157 patients suffering febrile morbidity and identified 120 infections among post-Cesarean patients, yielding rates of 16.2% and 12.4%, respectively (Table 3). Surgical site infections contributed 79.0% of all postoperative infections. Eighty-five patients developed 95 SSI among 969 abdominal deliveries, yielding the rate of 9.8%. One vaginal cuff infection was identified after a Cesarean hysterectomy. Ten SSI were documented after discharge from the hospital.

We identified nine factors with some evidence of association with SSI in univariate analysis ($P< 0.05$). Table 4 outlines all variables likely to be associated with this study outcome and thus included into the multivariate modeling progress. Despite the fact all Cesarean deliveries received parenteral antibiotics for prophylaxis, timing of administration appeared not to be closely associated with subsequent infections ($P=0.28$). Similarly, the association with patient age less than 20, even though it increased the risk of infection 1.7 times did not achieve the significant level ($P=0.27$).

Finally the multiple logistic regression revealed eight predictors independently associated with the occurrence of post-cesarean SSI (Table 5). Host susceptibility plays an essential role in the prediction of SSI.

*{The association between some putative factors and post-Cesarean section are listed in Tables 6 and 7.}

Discussion

The present prospective study emphasized the importance of post-Cesarean infection complications. The prospective nature of the study accompanied by a follow-up component enabled us to address precisely the magnitude and to identify the independent predictors of SSI after Cesarean delivery. Moreover, the short duration of the study as well as the application of the CDC definitions allowed uniformity in the criteria for diagnosis of various sources of post-Cesarean infections.

Our rather high rate of Cesarean section reflects the common phenomenon in the modern obstetrics[1,15-18]. It is hard to justify this trend in current medicine since the increasing rate of abdominal delivery has not been independently linked with the improvement of neonatal mortality and morbidity[19]. Medical professionals should focus more efforts to optimize Cesarean birth rate and consequently reduce maternal morbidity.

The surgical complications were rare in our data. Our blood transfusion rate is within the limit of those other reports which varied from 1.2% to 6.3%[16-18,20]. Most blood transfusion was given to patients with different extents of abnormal placental implantation. The incidence of reoperation because of intra-abdominal hemorrhage in Nielsen's study (0.3%)[20] was triple that in ours. The likelihood of bladder injury in our data is identical with the finding of Nielsen et al[18] and Eisenkop et al[22]. There was

no ureter injury in our study, whilst Eisenkop reported the rate of 0.09%[22].

We identified 16.2% of study subjects complicated by postoperative febrile morbidity. Although Pothinam et al[23] documented a significantly low rate (5.5%), recent reports in different settings around the world indicated post-Cesarean febrile morbidity rates ranging from 15.5% to 25.0%[16,17,24,25]. In a large meta-analysis study, recruiting 6,760 Cesarean deliveries, Hirsch et al[26] identified rates of 18% and 44% among patients with and without antibiotic prophylaxis, respectively. Differences in febrile morbidity definition, population studied and settings may bring about the different rates between others and ours.

The overall rate of postoperative infection was 12.4%, which is consistent with the rate of 13.9% reported from a prospective study of 1,319 Cesarean sections in Denmark[21]. In addition, our SSI rate, contributing four-fifths of all postoperative infections, was 9.8%, falling within the extremes reported by others also applying CDC definition of SSI[5,6,8,27,28]. However, neither Yacin's rate[5] nor Elhatawy's rate[6] was detected from a large-scale study, and so may be distorted to either side. Comparable rates to that found in our study have been documented from Brazil (11.6%)[27], Canada (8.8%[28] and 9.6%[16]), and the United States (10.8%)[30].

Risk factors for post-Cesarean surgical site infections

Multiple logistic regression revealed eight variables independently associated with the occurrence of post-Cesarean SSI.

We have corroborated the independent risk of preoperative remote infection and chorioamnionitis on the development of subsequent post-Cesarean SSI[11,29-32]. Remote infection not only compromises immune status of the patient but can increase the innoculum of microorganisms contaminating the surgical site as well. Further, the presence of a remote infection reflects heavy abnormal bacterial colonization that can readily contaminate the surgical site. Suonio et al[29], Garibaldi et al[30] and Hagglund et al[11] have shown that the presence of distant infections carried risks 8.7, 2.8 and 2.6 fold, respectively. Similarly, the presence of either intrauterine infection[11] or pathological[31] or clinical intra-amniotic infection[32] as risk factors of subsequent endometritis were demonstrated. This association remained valid when potential confounding variables were eliminated by multivariate[29-31] or stratified analysis[32]. In current medicine, prompt and aggressive antibiotic therapy should be started as soon as the suspected infection is established to reduce subsequent postoperative infections[11].

The ASA physical status classification is a standardized, reproducible numerical determination, which is used routinely to stratify severity of illness for surgical patients and known to be a good indicator of host susceptibility[9]. Surprisingly, no study to

date has investigated the potential association of ASA score and post-Cesarean infection. A concept of antepartum risk factors, a complex proxy of host susceptibility, was proven to be closely correlated with post-Cesarean endometritis[2]. Unfortunately, this association was not examined in the light of multivariate analysis. Our data supported Garibaldi's finding[30] in which the association between preoperative health status of patient and the development of wound infection remained valid even after multivariate modeling analysis. Severe systemic disease (ASA class of 3 or more) can increase the risk of infection by five times.

Similarly, we substantiated ROM duration as the independent predictor for post-Cesarean surgical site infections[2,10,21,33,34]. ROM lasting 24 hour or more carried 2.5- fold increased risk of infection after controlled for possible confounding effects. Further, despite the fact that ROM duration and level of wound contamination was closely related (P-value of Spearman's test for independence <0.0001), the former independently affected the outcome. Prolonged ROM duration increased the risk of bacterial contamination into the uterine cavity through vagina. Since ROM complicates 10%[35] to 18%[36] of term pregnancies, a proper intervention for this group of patients would result in large clinical implications.

Pre-eclampsia is also not uncommon since it is identified in approximately 6.1% of pregnancies[37]. This co-morbidity increases the risk of post-cesarean SSI by a factor of two. The immune

status of pre-eclampsia woman is compromised by several mechanisms. The generalized maternal endothelial cell dysfunction, a part of abnormally excessive systematic inflammatory response in pre-eclampsia, can result in a deterioration of function in a number of organs and systems[32]. Further, a woman with severe pre-eclampsia, who consequently lacks normal pregnancy hypervolemia, is much less tolerant of blood loss than is the normotensive pregnant women. At last, the abnormal vasospasm temporarily reduces blood perfusion of the incision site and thereby renders it liable to invasion by micro-organisms[33].

The role of obesity as a risk of post-Cesarean wound infection has been established for years[1,2,3,4,5,6]. Furthermore, multivariate analysis indicated that obesity remained a risk factor independently of other confounding variables[1,3]. However, the use of either prepregnancy[34] or antenatal[7] or delivery data[21] limited their findings. The postpartum data enabled us to more precisely investigate this association. Rather than using a dichotomous variable as obesity, a controversial term, we attempted to identify the correlation between the increment of body mass index and the occurrence of SSI. The risk of SSI doubled for every 5-unit increment of body mass index. There are many explanations for this finding. Specially included are a relative avascularity in adipose tissue or technical difficulties of handling adipose tissue that are associated with longer operation and can result in more trauma to the abdominal wall or the difficulties to obliterate dead space in the fat abdominal wall. Additionally, a

greatly increased wound area found in obese patients also contributes to the increasing incidence of SSI among obese patients.

We have confirmed the adverse effect of nulliparity on post-Cesarean wound infection[34,40]. The risk of wound infection was reduced by 39% and 60% when the patient had one and more than one children, respectively. The precise reason for the increased risk of SSI in a nullipara has not been fully understood. It has been postulated that the adaptation of nulliparous patient is not as good as that of multipara. Besides, a nullipara may have little experience of postpartum self-care.

The role of high volume of blood loss in increasing the risk of SSI[11,41] was reaffirmed in the present data. In addition, Ott et al[2] identified a significant association between postoperative anemia, a proxy of intra-operative blood loss, and postoperative endometritis. Risk of SSI increased 30% for every 100 ml-increment of blood loss. A high volume of blood loss is usually associated with poor control of bleeding, an increase in tissue damage from prolonged retraction and manipulation, and an increase in amount of suture. Suture, a foreign body, can promote the contamination and reduce local resistance mechanisms. Consequently, high volume of blood loss results not only in an immuno-compromised status of the patient but also in a directly increased possibility of wound contamination.

Finally, we could not reaffirm some variables, which had been demonstrated as the predictors of post-cesarean SSI, namely surgical duration[11,43,44] and experience of surgeon[43]. Unfortunately, confounding effects were sufficiently controlled by multiple logistic regression in some studies[29,42]. Prolonged surgical duration and resident as a lead surgeon carried the risk of 2.4 and 2.1, respectively. The significance however was lost when the confounding effects were controlled in multiple logistic regression.

In conclusion, eight factors were documented to be independently associated with post-Cesarean SSI. High-risk nulliparous patients with higher ASA score, pre-eclampsia, ROM, or increased body mass index should be more carefully managed. Moreover, the proper treatment of preoperative infection prior to Cesarean delivery should be more rigorous. A further intervention would target high-risk group of patients whose fetal membrane was ruptured prior to labor.

***{ Risk factor for post-Cesarean febrile morbidity**

Factors related independently to post-Cesarean febrile morbidity by multiple logistic regression analysis include chorioamnionitis, severe systematic diseases, preoperative fever, pre-eclampsia, and volume of blood loss.

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Whilst pre-Cesarean chorioamnionitis remained the strongest predictor of febrile morbidity after controlling for confounding effects, none of four cases experiencing remote infections developed fever postoperatively. Suonio et al.[29] also documented a strong association between the presence of amnionitis and post-Cesarean fever (RR=3.1, P=0.04). This association, however, was no longer valid when controlling for confounding effects in multivariate analysis[29]. In contrast, our data indicate that the presence of chorioamnionitis appears to be the important predictor of febrile morbidity after Cesarean delivery. The unadjusted odds ratio for the development of post-Cesarean FM associated with chorioamionitis (OR= 16.1, 95%CI= 3.2-80.5) remained the same in the multivariate analysis (OR= 16.0, 95CI= 3.1-83.3) after adjusting for all other putative factors. Chorioamnionitis increased the contamination of the uterine cavity and surgical field, thereby increasing the risk of postoperative febrile morbidity.

Despite the fact that obstetric patients are usually healthy, a patient with severe systematic disease (ASA ≥ 3) was placed at a very high risk of developing both SSI and PFM, perhaps owing to decreased immunocompetence. Further, the preoperative condition of patient was an independent predictor after controlling for underlying co-morbidity, such as severe pre-eclampsia, anemia, and heart diseases. The preoperative assessment would be widely used to indicate the group of high-risk patients for whom intensive

monitoring during labor should be required and the administration of antibiotics for prophylaxis should be considered.

In addition to chorioamnionitis, other co-morbidities, namely preoperative fever and pre-eclampsia turned out to be risk factors independently associated with post-Cesarean FM. The strength of the crude association between either fever or pre-eclampsia and PFM in univariate analysis was not appreciably changed in the multiple logistic regression analysis. Patients who manifested fever preoperatively could have infection etiology which remained the reason for the development of fever in the postoperative period. Further, like other co-morbidities, fever and pre-eclampsia could compromise the immune status, thereby increasing the risk for postoperative FM.

Volume of blood loss during Cesarean section has been identified as a predictor of PFM for years[23,25,29]. Pothinam et al[23], directly measuring estimated volume of blood loss, documented that the risk of PFM paralleled the increase in volume of blood loss during Cesarean section. However, the association lost its significance in the multivariate logistic regression analysis (adjusted OR=0.27, 95%CI= 0.02-5.54 for blood lost from 500 to 1000ml, compared with more than 1000ml). Similarly, using postoperative anemia as a proxy for intra-operative hemorrhage, both Guldholt[25] and Suonio[29] substantiated the risk of excessive blood loss for the subsequent development of PFM. Approximately one third of

postoperative anemia patients manifested fever, whereas the rate of PFM was only around 12.5% among patients without anemia identified after the operation[45]. Further, the multivariate analysis corroborated the independent prognostic value for intra-operative blood loss[25,29]. Like other co-morbidities, excessive blood lost intra-operatively could decrease the host defense, consequently increasing the risk of PFM.

Some factors documented as the independent determinants of post-Cesarean FM were not reaffirmed in our study. Specially included here are duration of ROM[29,43], preoperative anemia[29,44], obesity[44] and general anesthesia[44]. In fact, there was marginal evidence of an association between ROM duration and the development of PFM in the multivariate analysis (P=0.08).

In conclusion, host susceptibility was demonstrated to be the strongest independent predictor of post-Cesarean febrile morbidity. Chorioamnionitis should be extensively treated prior to the operation, minimizing the risk for post-Cesarean infection complications. The commonness and the considerable risk of ROM duration on the development of SSI and PFM could have large implication in both clinical and research aspects.)

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Table 1 *Demographic characteristics and co-morbidity of study subjects*

Characteristics	N=969
Age* (years)	29.7 ± 5.7
Nulliparous	44.7%
Body mass index*	22.6 ± 2.9
Residence (% rural)	31.4%
Repeated Cesarean section (%)	19.4%
Number patients having co-morbidity (%)	
None	85.2%
Pre-eclampsia	6.0%
Hypertension	3.5%
Preoperative fever	2.3%
Chorioamnionitis	0.8%
Anemia	0.6%
Diabetes mellitus	0.6%
Preoperative infection	0.5%
Heart diseases	0.3%
Others†	0.4%

* Mean ± standard deviation

† Comprised ovarian carcinoma(1), asthma(2), and hyperthyroidism(1).

Table 2 *Surgical indications for Cesarean section*

Surgical indications	Percent
Dystocia	35.2
Repeated Cesarean section*	15.4
Breech and malpresentation	13.8
Cephalopelvic disproportion	13.3
Fetal distress†	9.8
Fetal umbilical cord compression	4.5
Placenta previa	4.9
Placenta aruptio	1.4
Placenta increta	0.2
Others‡	1.5

* Indicated purely because of previous operation.

† Included acute fetal distress and intrauterine growth retardation.

‡ Included maternal diseases, failed forceps trial, failed labor induction, twin pregnancy, malformation of reproductive tract.

Table 3 Rate of post-cesarean febrile morbidity and infection complications*

Type of Cesarean section	Number	Febrile	Surgical Site Infection					UTI†	Others‡
		Morbidity	Superficial	Deep	Vaginal cuff	Endometritis	Organ/space		
Cesarean section	955	16.2	6.6	0.1	-	2.7	0.2	2.0	0.5
Cesarean section+ Gyn operation	14	14.3	-	7.1	7.1	-	-	-	-
Total	969	16.2	6.5	0.3	0.1	2.7	0.2	2.0	0.5

* Calculated as number of infections per 100 operations.

† Urinary tract infection

‡ Comprised pneumonia(2) and gastro-enteritis(3).

Table 4 *Crude association between selected variables and SSI*

Variables	SSI (n=85)	No SSI (n=884)	OR	95% CI	P-value
Nulliparity	56	480	1.6	1.02-2.6	0.04
Residence (rural)	32	272	1.4	0.9-2.1	0.20
Body mass index*	23.9± 3.7	22.5± 2.9	2.0†	1.4-2.9	<0.0001
Pre-eclampsia	13	43	3.5	1.8-6.9	<0.0001
Remote infection‡	2	2	10.6	1.5-76.5	0.003
Chorioamnionitis	4	4	10.9	2.7-44.3	0.001
Prior hospitalization (d)*	2.4± 6.9	1.5± 2.8	1.1§	1.0-1.1	0.04
ROM-operation interval (h)	7.9± 10.3	5.9± 8.5	1.2‖	1.0-1.3	0.04
ASA score					<0.0001
1	67	802	1	1	
2	14	76	2.2	1.2-4.1	
≥ 3	4	6	9.6	2.5-36.6	
Level of surgeon					0.08
Supervisor	14	187	1	1	
Attending physician	54	589	1.2	0.7-2.3	
Resident	17	108	2.1	1.1-4.1	
Cesarean hysterectomy	2	3	7.1	1.2-43.0	0.01
Abdominal incision¶	16	122	1.5	0.8-2.6	0.20
Wound class					0.12
Clean	18	279	1	1	
Clean-contaminated	15	152	1.5	0.7-3.1	
Contaminated	52	453	1.8	1.01-3.1	
Surgical duration ≥ 1 hour	5	22	2.4	0.9-6.6	0.07
Blood loss (ml)*	421.9±160.7	372.5±126.3	1.3**	1.1-1.4	0.001

* Mean ± standard deviation

† OR for every 5-unit increment of BMI; ‡ Preoperative infections other than chorioamnionitis; § OR for every 1-day increment. ‖ OR for every 6-hour increment ROM duration. ¶ Vertical incision versus others; ** OR for every 100-ml increment

Table 5 Multiple logistic regression analysis and independent risk factors for surgical site infections following Cesarean section

Variables	Coefficient	OR	95% CI	P-value
Constant	-7.7			
Preoperative remote infection	2.6	12.9	1.6-102.0	0.015
Chorioamnionitis	2.1	8.2	1.7-40.3	0.010
ASA score ≥ 3	1.7	5.5	1.2-25.4	0.029
PROM ≥ 24 hours	0.9	2.5	1.1-5.4	0.021
Pre-eclampsia	0.8	2.2	1.02-4.7	0.044
Body mass index (5-unit increment)	0.7	2.0	1.4-3.0	0.001
Nulliparity	0.6	1.8	1.03-3.2	0.04
Blood loss (every 100-ml increment)	0.2	1.3	1.1-1.5	0.003

Table 6 *Crude association between selected variables and PFM*

Variables	PM (n=157)	No PM (n=912)	OR	95% CI	P-value
Body mass index*	23.0± 2.7	22.6± 3.0	1.3†	1.0-1.7	0.06
Pre-eclampsia	19	37	2.9	1.6-5.2	<0.0001
Preoperative fever	13	9	8.1	3.4-19.2	<0.0001
Placental abnormality‡	18	48	2.1	1.2-3.6	0.013
Chorioamnionitis	6	2	16.1	3.2-80.5	0.001
Prior hospitalization (d)*	2.3± 4.9	1.4± 2.6	1.6§	1.1-2.5	0.021
Preoperative amniocentesis	13	14	1.7	0.9-3.2	0.110
ROM-operation interval (h)	8.1± 12.1	5.7± 7.8	1.2‖	1.1-1.3	0.002
ASA score					<0.0001
1	128	741	1	1	
2	22	68	1.9	1.1-3.1	
≥ 3	7	3	13.5	13.4-52.9	
Antibiotic administration					0.013
Early	13	27	2.4	1.1-5.0	
Preoperative	42	207	1	1	
Perioperative	102	578	0.9	0.6-1.3	
Level of surgeon					0.09
Supervisor	33	168	1	1	
Attending physician	96	547	1.2	0.8-1.8	
Resident	28	97	1.8	1.1-3.0	
Cesarean hysterectomy	2	3	3.5	0.6-21.0	0.17
Wound class ≥ 3	88	417	1.2	0.9-1.7	0.28
Surgical duration ≥ 1 hour	10	17	3.2	1.4-7.1	0.003
Blood loss (ml)*	422.2±182.7	368.1±115.7	11.3¶	1.21-1.5	<0.0001

* Mean ± standard deviation

† OR for every 5-unit increment of BMI; ‡ Placenta previa, placenta aruptio, placenta increta; § OR for every 3-day increment; ‖ OR for every 6h increment of ROM duration; ¶ OR for every 100ml increment.

Table 7 *Multiple logistic regression and independent predictors of post-Cesarean febrile morbidity (PFM)*

Variables	Coefficient	OR	95% CI	P-value
Constant	-4.1			
Chorioamnionitis	2.8	16.0	3.1-83.3	0.001
ASA score ≥ 3	2.6	13.6	2.8-65.7	0.001
Preoperative fever	2.2	9.5	3.8-23.7	<0.0001
Pre-eclampsia	0.9	2.4	1.2-4.6	0.012
Blood loss (every 100-ml increment)	0.2	1.3	1.1-1.5	0.003