Wound Outcome Following Primary and Delayed Primary Skin Closure Techniques After Laparotomy for Non-Traumatic Ileal Perforation: a Randomized Clinical Trial

Vivek Agrawal¹ · Mohit Kumar Joshi² · Ashish Kumar Gupta¹ ·
Bhupendra Kumar Jain¹

Received: 21 April 2014 / Accepted: 28 December 2015 / Published online: 15 January 2016
© Association of Surgeons of India 2016

Abstract To study the effect of primary and delayed primary closure of skin incision on wound outcome in patients with non-traumatic ileal perforation, 68 patients of ileal perforation were studied in a prospective randomized clinical trial. Patients fulfilling inclusion criteria were divided into ileostomy and non-ileostomy groups, both of which were then randomized into two subgroups each depending on whether skin was closed primarily or in a delayed primary manner. Wound infection and dehiscence were the main outcome parameters studied. The data collected was analyzed using appropriate statistical tools taking significant *p* value at 5 %. Most patients were 21–30 years of age. Male:female ratio was 3.2:1. The overall incidence of wound infection was 63 %. Wound infection was strongly associated with the incidence of superficial wound dehiscence and total wound dehiscence that were 11.76 and 47 %, respectively. Mortality was 10.3 %. Methodology of wound closure has no significant impact on incidence of wound infection, wound dehiscence, and mortality, although the onset of wound complications is significantly delayed with delayed primary closure of the skin.

Keywords Ileal perforation · Primary skin closure · Delayed primary skin closure · Wound infection · Wound dehiscence

Abbreviations

PSC	Primary skin closure
DPSC	Delayed primary skin closure
MPI score	Mannheim peritonitis index
WI	Wound infection
SWD	Superficial wound dehiscence
TWD	Total wound dehiscence
DOPC	Degree of peritoneal contamination

Introduction

Peritonitis due to ileal perforation is a common surgical emergency worldwide [1, 2]. Enteric fever is one of the commonest causes of ileal perforation, followed by non-specific inflammation, tuberculosis, and trauma [3, 4]. Ileal perforation carries a high morbidity and mortality [3, 5, 6]. Early surgery and either primary closure of the perforation or its exteriorization as ileostomy are considered the best treatment for ileal perforation [3, 7, 8]. During surgery, however, contamination of the surgical wound by intestinal contents may lead to increased incidence of wound infection [9–11]. Wound infection increases the incidence of wound dehiscence and related morbidity, posing a significant impact on health care resources and costs [12]. Risk factors increasing the incidence of wound complications in patients with ileal perforation include advanced age, malnutrition, anemia, hypoproteinemia, uremia, type of suture used, use of drains, surgeon's experience, steroid use, abdominal distension, pulmonary disease, and positive intraoperative cultures [13, 14]. In patients with these factors, measures can be taken at the time of wound closure to reduce the incidence of wound infection. The method of skin closure has been implicated as an important factor influencing postoperative wound complications [9, 15–17], although various studies conducted addressing this issue

✉ Mohit Kumar Joshi
drmohtijoshi@gmail.com

¹ Department of Surgery, University College of Medical Sciences and Guru Teg Bahadur Hospital, Delhi, India

² Department of Surgical Disciplines, All India Institute of Medical Sciences, New Delhi, India 110029

report diverse results [15, 16, 18, 19]. This study was done to investigate the effect of primary skin closure (PSC) and delayed primary skin closure (DPSC) techniques on wound outcome following laparotomy for non-traumatic ileal perforation.

Methods

This prospective randomized trial was conducted on 68 patients of all ages and either sex meeting the entry criteria, undergoing laparotomy through midline incision for non-traumatic ileal perforation, over a period of 19 months.

The patients with duodenal ulcer perforation, perforation secondary to obstruction, volvulus, strangulation, immunocompromised states, diabetes mellitus, uremia, jaundice, chronic pulmonary disease, and history of previous laparotomy were excluded. All eligible patients received same antibiotics in peri-operative period. Mannheim peritonitis index (MPI) score was calculated. Laparotomy was performed in all patients by midline vertical incision. The findings including the degree of peritoneal contamination were recorded. Peritoneal contamination was classified as mild, moderate, or severe if the volume of the peritoneal fluid was <500, 500–1000, or >1000 ml, respectively. Tissue from the edge of the perforated ulcer was taken for histopathological examination in all patients. In some patients, other abnormal or suspicious tissues like lymph node and omentum were also biopsied. The patients were then divided into two groups, depending whether ileostomy was done or not as a part of treatment (group 1 ileostomy group and group 2 non-ileostomy group).

The patients were then randomly allocated to a method of skin closure technique (either PSC or DPSC) by using computer-generated random numbers, thus dividing the two groups into subgroups as subgroup A, PSC, and subgroup B, DPSC. Two boxes were made, one labeled as ileostomy and the other as non-ileostomy containing equal number of slips labeled with numbers of PSC or DPSC. Once the decision on the mode of operative procedure (ileostomy or non-ileostomy) was made, a slip from that particular box was picked up, and accordingly, the patient underwent PSC or DPSC. The number labels were stuck onto the patient's proforma for identification purposes. Thus, we had the following four subgroups: subgroup 1A, ileostomy with primary closure of the skin; subgroup 1B, ileostomy with delayed primary closure of the skin; subgroup 2A, primary closure of ileal perforation and skin; and subgroup 2B, primary closure of ileal perforation and delayed primary closure of the skin.

On conclusion of laparotomy, the abdominal wound was closed in similar manner in both groups till the rectus sheath, using continuous monofilament no. 1 polypropylene suture. After irrigation of the wound with copious amount of saline, in

subgroup A, the skin was closed by interrupted sutures using nylon 2-0 suture. In subgroup B, the skin and subcutaneous layer was left unsutured and the wound was dressed with saline-soaked gauze. All wounds in subgroup B were closed on fourth postoperative day except in cases where the wound exhibited purulent discharge. The technique of skin closure was same as that in subgroup A. The dressing was not opened till second postoperative day in either group unless it was soiled. The wound was examined on second and fourth postoperative days. In cases of wound infection, pus was drained, a sample sent for microbiological investigations, and antibiotic therapy was instituted. In patients with satisfactory wound healing, the sutures were removed on 10th postoperative day. Patients in whom wound could not be sutured because of infection, the wound was left for a further period till decision for delayed suturing/healing by secondary intention was made. Postoperatively patients were observed for

1. Wound infection, purulent discharge, skin erythema, tachycardia, fever, or leukocytosis.
2. Wound dehiscence, superficial (if the gaping was confined to skin and subcutaneous tissue) and total (if the musculo-aponeurotic layer also gave way).

The duration of hospital stay was recorded. The patients were followed for a minimum of 1 month. The data was entered in a predesigned proforma. Univariate analysis was done using Student's *t* test, Pearson's chi-squared test, and Fischer's exact test. Multivariate analysis was done only for significant variables (determined by univariate analysis) using forward stepwise likelihood ratio, taking 5 % as significant level.

Results

Over the study period, 194 patients with non-traumatic small-bowel perforation attended our hospital. Of these, 68 patients met the entry criteria and were included in the study after obtaining formal written consent. Thirty-eight patients underwent ileostomy (group 1), with 19 patients each randomized to PSC (group 1A) and DPSC (group 1B) groups. Thirty patients were treated without ileostomy (group 2), and 15 cases each were randomized to PSC (group 2A) or DPSC (group 2B) groups.

Majority of patients were aged 21–30 years ($n = 23$, 33.8 %). The difference in mean age of patients in subgroups 1A and 1B and that in subgroups 2A and 2B was not statistically significant ($p = 0.1225$ and 0.6081 , respectively). The male to female ratio was 3.2:1. The male:female ratio in groups 1 and 2 were 2.16:1 and 9:1, respectively; the difference was statistically significant ($p = 0.0417$). However, the gender difference among the subgroups could not attain statistical significance.

Commonest cause of perforation was non-specific inflammation (Table 1). The overall mean duration of peritonitis was 67.0 ± 43.85 h (range 27 to 202 h). The mean duration of peritonitis for group 1 (ileostomy group) was 77.5 ± 49.68 h as compared to that of group 2 (non-ileostomy group) 53.8 ± 31.11 h. The difference between the two groups was statistically significant ($p=0.019$). However, this difference among subgroups was not statistically significant.

Mean pre-operative in-hospital time in subgroups 1A and 1B were 8.63 ± 3.131 and 9.26 ± 4.254 h, respectively; the difference between these was not significant ($p=0.6063$). The same scores in subgroups 2A and 2B were 8.60 ± 3.661 and 7.40 ± 4.323 h, respectively; the difference again was not significant ($p=0.4189$). All patients were operated within 24 h of admission. The difference between the degree of peritoneal contamination among the subgroups, i.e., subgroup 1A versus subgroup 1B and subgroup 2A versus subgroup 2B, did not attain statistical significance ($p>0.0562$ and $p>0.4493$, respectively).

Mean MPI score was 31.16 ± 3.87 (range 21–38). The disease-wise MPI is mentioned in Table 2. In subgroups 1A and 1B, mean MPI scores were 32.16 ± 3.078 and 33.32 ± 2.868 , respectively; the difference was insignificant ($p=0.2375$). The same scores in subgroups 2A and 2B were 29.80 ± 3.840 and 28.33 ± 4.030 , respectively; the difference was again insignificant ($p=0.3155$).

Most common organism isolated from pus culture was *Escherichia coli* in 24 patients (35 %) followed by mixed growth in six cases (8.8 %) and *Klebsiella* in three cases (4.4 %) and *Staph aureus*, *Enterobacter*, and *Pseudomonas* in one patient each (1.4 %) and no growth in five cases (7.3 %). However, no significant difference was found regarding the organism isolated in various subgroups ($p=0.906$).

Loop ileostomy was the preferred procedure and was performed in all patients except four patients who underwent resection of bowel for multiple perforations, and either a double barrel or end ileostomy was performed in them. The overall incidence of wound infection was 63 % ($n=43$). In subgroup 1A, incidence of wound infection was 74 % ($n=14$). In subgroup 1B, DPSC could not be done in six patients because of infected wound. Out of remaining 13 patients who underwent DPSC, 8 patients developed wound infection. However, the difference among the two subgroups did not

attain statistical significance ($p=0.7341$). In subgroup 2A, incidence of wound infection was 53 % ($n=8$). In subgroup 2B, three patients (20 %) could not undergo DPSC because of infected wound. Among 12 patients that underwent skin closure, 4 patients developed wound infection. The difference between these two subgroups however was insignificant ($p=0.5160$). The difference between incidence of wound infection in group 1 and group 2 (73.6 and 50 %, respectively) was found to be statistically significant ($p=0.044$). Onset of wound infection, superficial wound dehiscence (SWD), and total wound dehiscence (TWD) were significantly delayed with DPSC in both groups (Table 3). The wound outcome parameters in various subgroups are summarized in Table 4.

Out of 43 patients with wound infection, 32 (74 %) developed TWD. The association between wound infection and TWD was found statistically significant ($p=0.000$). All 32 patients who developed TWD had wound infection. Mean hospital stay was 14.46 ± 10.49 days (range 5–56 days). Mean duration of hospital stay in subgroups 1A and 1B were 12.95 ± 10.35 and 17 ± 10.5 days, the difference being statistically insignificant. Hospital stay in groups 2A and 2B were 13.73 ± 12.2 and 13.93 ± 9.2 days, respectively; the difference again was found insignificant.

The incidence of wound infection increased with increasing peritoneal contamination (Table 5); the association was statistically significant ($p=0.008$). Wound infection also increased with MPI score of >32 , irrespective of the type of skin closure technique. Wound infection was 6.8 times more likely and TWD was 14 times more likely in patients with MPI score of >32 .

There was no incidence of leak. Seven (10.3 %) patients died. All deaths occurred in ileostomy group (18.4 %), and they all had MPI score of more than 32. The difference in mortality between two groups was statistically significant ($p=0.015$).

Discussion

Peritonitis due to ileal perforation deserves attention as it is prevalent in developing countries, primarily affects young patients and carries a high morbidity and mortality. We studied 68 consecutive patients of ileal perforation meeting the entry

Table 1 Etiology of NTPSI and their distribution in various subgroups

Etiology	Subgroup 1A (I-PSC, $n=19$)	Subgroup 1B (I-DPSC, $n=19$)	Subgroup 2A (NI=PSC, $n=15$)	Subgroup 2B (NI-DPSC, $n=15$)
Non-specific	12 (63.15 %)	14 (73.68 %)	13 (86.6 %)	12 (80 %)
Tuberculosis	6 (31.5 %)	4 (21 %)	2 (13.3 %)	3 (20 %)
Enteric	1 (5.2 %)	1 (5.2 %)	0 (0 %)	0 (0 %)

I-PSC ileostomy-primary skin closure, I-DPSC ileostomy-delayed primary skin closure, NI-PSC non-ileostomy-primary skin closure, NI-DPSC non-ileostomy-delayed primary skin closure, n = number of patients

Table 2 Disease-wise Mannheim peritonitis index (MPI) score

MPI score	<27	27–32	>32
Non-specific (<i>n</i> = 51)	2	13	36
Tuberculosis (<i>n</i> = 15)	2	3	10
Enteric (<i>n</i> = 2)	1	1	0
Total (<i>n</i> = 68)	5	17	46

criteria for the study. The majority of patients were in second and third decades of their life. This observation is similar in some other studies also [3, 6, 7, 20]. The male:female ratio of 3.2:1 is also comparable to the study by other authors [4, 6, 20]. Male preponderance has also been reported in various other series [2, 7, 8, 21–23]. More females underwent ileostomy; the reason may be that the women in our society are usually neglected and present late with advanced peritonitis. The increased peritoneal contamination associated with delayed presentation in peritonitis may influence a surgeon's decision to perform ileostomy, explaining the large number of females undergoing ileostomy.

The incidence of typhoid as a cause of bowel perforation was low. Wani et al. [4] reported enteric perforation in 62 % patients in their series, while Jhobta et al. [2] and Afridi et al. [1] have reported frequencies of 8 and 17 %, respectively. In various studies, the typhoid perforation was assumed either on clinical grounds alone, a positive Widal test or operative findings [1, 4, 24]. The low incidence of typhoid as a cause of perforation in this study may be due to the stringent criteria for defining typhoid ileal perforation. We designated a perforation to be caused by typhoid only if mononuclear infiltration and erythrophagocytosis were demonstrated on histopathological examination [24]. The mean duration of peritonitis was significantly more in group 1 ($p=0.019$), indicating that the surgeon's decision of making stoma was justified in most cases. Literature also supports stoma formation as a preferred modality of treatment in advanced peritonitis [7, 8, 24]. The patients in both groups who could not undergo DPSC because of wound infection (WI) and had poor wound outcome later either had MPI>32 and/or severe peritoneal contamination with a few having bowel edema and multiple perforations, suggesting that these factors also play a role in determining the wound outcome.

The pre-operative hospital stay was similar in both groups ($p=0.314$). All patients underwent surgery within 24 h of admission. In various other series also, the patients of typhoid ileal perforation were operated within 24 h [2, 3, 8, 25, 26].

The incidence of severe peritoneal contamination was similar to the findings of Malik et al. [8]. However, this has been reported less in other studies [3, 27]. The severe degree of peritoneal contamination is due to delayed presentation to the hospital and is responsible for the high incidence. WI was more in patients with moderate and severe degree of peritoneal contamination (DOPC) ($p<0.008$). However, the difference in incidence of WI among the four subgroups was not significant, indicating that WI is more likely to depend on DOPC rather than the type of skin closure.

To predict the outcome of patients with peritonitis, several scoring systems have been used. MPI score used in the study has high sensitivity and specificity. In addition, it is simple, effective, and reliable [28, 29]. MPI score was significantly higher in ileostomy group as compared to the non-ileostomy group ($p=0.000$). This is expected as patients undergoing ileostomy were in advanced peritonitis. The MPI score of >32 was significantly associated with WI ($p<0.000$). This is supported by Gedik et al. [30].

WI was the most common morbidity; this has been reported by others also [3, 20, 25, 31, 32]. We observed a higher incidence of wound infection in the ileostomy group that was statistically significant as compared to the non-ileostomy group. This was comparable to the study done by Adesunkanmi et al. [3]. WI is multifactorial and depends on the clinical condition of the patient and associated co-morbidities, virulence of the organism, host resistance, duration of peritonitis, amount of contamination, and differences in hygiene and aseptic techniques. These factors may explain variable incidence of WI reported in different studies. All aforementioned factors and other exogenous sources of infection would also have played their part in the causation of wound infection in the peri-operative. However, as the study was randomized and these factors remained same for all patients, this would probably have nullified the influence of these parameters on any selective group.

It was interesting to note that DPSC significantly delayed the onset of WI. This can be explained by the fact that DPSC prevents the formation of seroma and anaerobic environment in the wound, thus avoiding bacterial proliferation. Few authors have reported a lower incidence of WI following DPSC

Table 3 Wound outcome parameters in various subgroups

Parameters	Subgroup 1A (I-PSC, <i>n</i> = 19)	Subgroup 1B (I-DPSC, <i>n</i> = 19)	Subgroup 2A (NI = PSC, <i>n</i> = 15)	Subgroup 2B (NI-DPSC, <i>n</i> = 15)
Wound infection	14 (74 %)	14 (74 %)	8 (53.3 %)	7 (46.7 %)
Superficial wound dehiscence	4 (21.0 %)	3 (15.79 %)	1 (6.67 %)	0 (0 %)
Total wound dehiscence	9 (47.36 %)	11 (57.89 %)	7 (46.7 %)	5 (33.34 %)

Table 4 Incidence of adverse wound outcomes on different post-operative days among subgroups

Parameters	Post-operative day (POD)	Subgroup 1A (I-PSC, <i>n</i> = 19) (%)	Subgroup 1B (I-DPSC, <i>n</i> = 13+6 ^a) (%)	Subgroup 2A (NI-PSC, <i>n</i> = 15) (%)	Subgroup 2B (NI-DPSC, <i>n</i> = 12+3 ^b) (%)
WI	0–4	13 (68)	6 (31.5)	8 (53.3)	3 (20)
	>4	1 (5.2)	8 (42)	0 (0)	4 (26)
<i>P</i> value		0.0046 (s)		0.0125 (s)	
SWD	0–4	2 (10.5)	0 (0)	1 (6.6)	0 (0)
	>4	2 (10.5)	3 (23)	0 (0)	0 (0)
<i>P</i> value		>0.189 (s)			
TWD	0–4	7 (36.8)	1 (5.2) ^d	2 (13.3)	1 (6.6) ^c
	>4	2 (10.5)	10 (52.6)	5 (33.3)	4 (26.6)
<i>P</i> value		0.0018 (s)		0.7353(ns)	

s significant, *ns* non-significant

^a Six patients in subgroup 1B could not undergo suturing on or after fourth POD and subsequently developed TWD.

^b Three patients in subgroup 2B could not undergo suturing on or after fourth POD and subsequently developed TWD.

^c Out of three patients who could not undergo suturing in subgroup 2B, one developed TWD on fourth POD and two developed TWD later.

^d Out of six patients who could not undergo suturing in subgroup 1B, one developed TWD on fourth POD and five developed TWD later.

than PSC [15–17]. These authors postulated that in the patients undergoing PSC, the bacteria are trapped in the subcutaneous tissue. This space has poor vascularity, and the collection of exudates, blood clots, and other surgical debris in this space provide an excellent culture medium, allowing bacteria to grow and multiply rapidly leading to increased incidence of WI. However, there was no significant difference in the incidence of WI among the four subgroups, indicating that development of WI is multifactorial.

The incidence of SWD was 12 %. Edino et al. [25] and Cliby et al. [33] in their study found the incidence of SWD of 7.6 and 5 %, respectively. The higher incidence of SWD in this study may be due to the higher incidence of WI; however, the incidence of SWD was not significantly different among the four subgroups, suggesting that the skin closure technique has no role in altering the incidence of SWD.

The incidence of 47 % of TWD is comparable to that reported by Capoor et al. [6] (49 %). Adesunkanmi et al. [3] and Edino et al. [25] have reported the incidence of TWD as 34

and 24.2 %, respectively. In subgroup 1A, seven patients (36.8 %) experienced TWD in less than 4 days after surgery. It may be difficult to narrow down to a single factor for this high incidence of early TWD observed in one subgroup, considering the fact that the etiology of wound dehiscence is multifactorial. Common factors for early TWD are poor technique of abdominal closure and use of an unsuitable suture for closing the laparotomy wound. However, the method of abdominal wall closure was same in all patients, so this should not be the cause of high incidence of TWD in this subgroup. The development of TWD was significantly delayed by DPSC in group 1 ($p = 0.0018$). This may be due to the increased tensile strength of the wounds undergoing DPSC as compared to the wounds closed primarily that may have prevented the burst abdomen for a longer duration. Wound infection was found to be the antecedent cause for total wound dehiscence. This is also reported by various other authors [3, 13].

Four patients had multiple ileal perforations. The perforation-bearing segment of ileum was resected, and a

Table 5 Significant factors in patients with wound infection

Variables ^a		Subgroup 1A (I-PSC, <i>n</i> = 14) (%)	Subgroup 1B (I-DPSC, <i>n</i> = 14) (%)	Subgroup 2A (NI-PSC, <i>n</i> = 8) (%)	Subgroup 2B (NI-DPSC, <i>n</i> = 7) (%)
DOPC	Mild	0 (0)	1 (7)	3 (37.5)	2 (28.5)
	Moderate	6 (43)	1 (7)	3 (37.5)	1 (14.3)
	Severe	8 (58)	12 (85.7)	2 (25)	4 (57)
MPI score	27–32	2 (14.3)	0 (0)	1 (12.5)	0 (0)
	>32	12 (85.7)	14 (100)	7 (87.5)	5 (71.4)

n number of patients with WI

^a More than one variable was present in same patient

double barrel or end ileostomy was performed in these patients. Two of them developed wound infection.

There was no incidence of enterocutaneous fistula. Mean hospital stay in our study is comparable to that reported by Edino et al. [25]. There was no difference among the four subgroups with regards to the duration of hospital stay, indicating that DPSC can be done in selected patients without prolonging the hospital stay.

The mortality rate was 10.3 %. This was comparable to other series [7, 8, 34]. All deaths occurred in ileostomy group. This is expected as the patients in this group were sicker. All patients who died had an MPI score of >32.

The wound-related complications were similar in both groups; however, the onset of wound complications was significantly delayed in patients when delayed primary closure of the skin was performed. Based on this observation, we recommend that delayed primary skin closure should be done in all patients undergoing laparotomy for peritonitis. However, more studies with larger number of patients will further add to the existing knowledge and will help in clarifying this issue better.

Conclusions

Methodology of wound closure has no significant impact on incidence of WI and TWD; however, onset of WI, SWD, and TWD is significantly delayed when wounds undergo DPSC.

References

1. Afridi SP, Malik F, Ur-Rahman S, Shamim S, Samo AK (2008) Spectrum of perforation peritonitis in Pakistan: 300 cases Eastern experience. *World J Emerg Surg* 3:31
2. Jhobta RS, Attri AK, Kaushik R, Sharma R, Jhobta A (2006) Spectrum of perforation peritonitis in India—review of 504 consecutive cases. *World J Emerg Surg* 1:26
3. Adesunkanmi ARK, Ajao OG (2002) The prognostic factors in typhoid ileal perforation: a prospective study of 50 patients. *J R Coll Surg Edinb* 42:395–399
4. Wani RA, Parray FQ, Bhat NA, Wani MA, Farzana F (2006) Nontraumatic terminal ileal perforation. *World J Emerg Surg* 1:1–6
5. Sharma D, Bhansali M, Saxena A, Raina VK (2002) Treatment of typhoid ileal perforation by resection and temporary ileostomy. *Indian J Gastroenterol* 21:205–206
6. Capoor MR, Nair D, Chintamani MS, Khanna J, Aggarwal P, Bhatnagar D (2008) Role of enteric fever in ileal perforations: an overstated problem in tropics? *Indian J Med Microbiol* 26:54–57
7. Ansari GA, Naqvi SQH, Ghumro AA, Jamali AH, Talpur AA (2009) Management of typhoid ileal perforation: a surgical experience of 44 cases. *Gomal J Med Sci* 7:21–30
8. Malik AM, Laghari AA, Mallah Q, Qureshi GA, Talpur AH, Effendi S, Memon JM (2006) Different surgical options and ileostomy in typhoid perforation. *World J Med Sci* 1:112–116
9. Leaper DJ (2008) Surgical infection. In: Williams NS, Bulstrode CJK, O’Connell PR (eds) *Bailey and Love’s short textbook of Surgery*, 25th edition. Hodder Arnold, p32–48
10. Burt BM, Tavakkolizadeh A, Ferzoco SJ (2007) Incisions, closures, and management of the abdominal wound. In: Zinner MJ (ed) *Maingot’s Abdominal Operations*, 11th edition. McGraw Hill Medical Publication, p 71–98
11. Kulaylat MN, Dayton MT (2008) Surgical complications. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL (eds) *Sabiston Textbook of Surgery*, 18th edition. Saunders Publication, p328–334
12. Gottrup F, Melling A, Hollander DA. An overview of surgical site infections: aetiology, incidence and risk factors. 2005, Feb 21, available from: <http://www.worldwidewounds.com/2005/september/Gottrup/Surgical-Site-Infections-Overview.html>
13. Waqar SH, Malik ZI, Razzaq A, Abdullah MT, Shaima A, Zahid MA (2005) Frequency and risk factors for wound dehiscence/burst abdomen in midline laparotomies. *J Ayub Med Coll Abbottabad* 17: 123–126
14. Agrawal V, Sharma N, Joshi MK, Minocha VR (2009) Role of suture material and technique of closure in wound outcome following laparotomy for peritonitis. *Trop Gastroenterol* 30:237–240
15. Cohn SM, Giannotti G, Ong AW, Varela E, Shatz DV, McKenney MG, Sleeman D, Ginzburg E, Augenstein JS, Byers P, Sands LR, Hellinger MD, Namias N (2001) Prospective randomised trial of two wound management strategies for dirty abdominal wounds. *Ann Surg* 233:409–413
16. Ussiri EV, Mkony CA, Aziz MR (2004) Sutured and open clean-contaminated and contaminated laparotomy wounds at Muhimbili National Hospital: a comparison of complications. *East Cent Afr J Surg* 9:89–95
17. Brown SP (2000) Laparotomy and intraperitoneal sepsis. In: Ellis BW, Brown SP (eds) *Hamilton Bailey’s Emergency Surgery*, 13th edition. Arnold Publication, p307–26
18. Duttaroy DD, Jitendra J, Duttaroy B, Bansal U, Dhameja P, Patel G, Modi N (2009) Management strategy for dirty abdominal incisions: primary or delayed primary closure? A randomized trial. *Surg Infect (Larchmt)* 10:129–136
19. Rucinski J, Fabian T, Panagopoulos G, Schein M, Wise L (2000) Gangrenous and perforated appendicitis: a meta-analytic study of 2532 patients indicates that the incision should be closed primarily. *Surgery* 127:136–141
20. Ugochkwu AI, Amu OC, Nzegwu MA (2013) Ileal perforation due to typhoid fever—review of operative management and outcome in an urban centre in Nigeria. *Int J Surg* 11:218–222
21. Usang UE, Sowande OA, Ademoyiwa AO, Bakare TIB, Adejuyigbe O (2009) Outcome of primary closure of abdominal wounds following typhoid perforation in children in Ife Ife, Nigeria. *Afr J Paediatr Surg* 6:31–34
22. Nuhu A, Gali B, Dawha S (2009) Postoperative complications of typhoid ileal perforation in children in Azare, Nigeria. *Internet J Surg* 21: number 1
23. Ahmad T, Khan MI, Hussain N, Siddiqui E, Islam ZU (2009) Perforation operation interval as a prognostic factor in typhoid ileal perforation. *J Surg Pakistan (International)* 14:11–14
24. Caronna R, Boukari AK, Zaongo D, Hessou T, Gayito RC, Ahononga C et al (2013) Comparative analysis of primary vs resection and anastomosis, with laparoscopy, in management of typhoid intestinal perforation: results of a rural hospital in Northwestern Benin. *BMC Gastroenterol* 13:102
25. Edino ST, Yakubu AA, Mohammed AZ, Abubakar S (2007) Prognostic factors in typhoid ileal perforation: a prospective study of 53 cases. *J Natl Med Assoc* 99:1042–1045
26. Kouame J, Kouadio L, Turquin T (2004) Typhoid ileal perforation surgical experience of 64 cases. *Acta Chir Belg* 104:445–447

27. Sharma L, Gupta S, Soin AS, Sikora S, Kapoor V (1991) Generalised peritonitis in India—the tropical spectrum. *Jpn J Surg* 21:272–277
28. Kologlu M, Elker D, Altun H, Sayek I (2001) Validation of MPI and PIA II in two different groups of patients with secondary peritonitis. *Hepato-Gastroenterology* 48:147–151
29. Notash A, Salimi J, Rahimian H, Fesharaki MSH, Abbasi A (2005) Evaluation of Mannheim peritoneal index and multi organ failure score in patients with peritonitis. *Indian J Gastroenterol* 24:197–200
30. Gedik E, Girgin S, Tacyildiz IH, Akgun Y (2008) Risk factors affecting morbidity in typhoid ileal perforation. *Langenbeck's Arch Surg* 393:973–977
31. Hussain M, Khan RM, Rehmani B, Haris H (2011) Omental patch technique for the ileal perforation secondary to typhoid fever. *Saudi J Gastroenterol* 17:208–211
32. Agu K, Nzegu M, Obi E (2014) Prevalence, morbidity and mortality patterns of typhoid ileal perforation as seen at the University of Nigeria Teaching Hospital Enugu Nigeria: an 8-year review. *World J Surg* 38:2514–2518
33. Cilby WA (2002) Abdominal incision wound breakdown. *Clin Obstet Gynaecol* 45:507–517
34. Shukla VK, Sahoo SP, Chauhan VS (2004) Enteric perforation—single layer closure. *Dig Dis Sci* 49:161–164