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The association of puerperal sepsis with HIV infection at two tertiary hospitals in Zimbabwe

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Abstract

Objective: To evaluate the association between HIV infection and puerperal sepsis among women in Zimbabwe.

Methods: A subanalysis was performed using data from a prospective cohort study conducted between September 2, 2014, and July 1, 2015, at two tertiary hospitals in Zimbabwe. Eligible participants were consecutive women who met the WHO criteria for puerperal sepsis. Variables assessed included HIV-infection status and the use of antiretroviral therapy. Severity of immunosuppression was defined by the number of T cells that expressed cluster of differentiation 4 (CD4). Endocervical swabs and blood samples were collected for microbial culture and susceptibility testing.

Results: In all, 33 (21.9%) of the 151 women included in the present analysis had HIV. Among women with HIV, severe immunosuppression (CD4-positive T cell count <200/mm^3) was associated with a mean hospital stay of 19.0 days versus 10.2 days for mild-advanced immunosuppression (CD4-positive T cell count 200–500/mm^3) and insignificant immunosuppression (CD4-positive T cell count >500/mm^3; P=0.030). Use of antiretroviral therapy did not independently influence clinical outcomes. Furthermore, infection with HIV did not influence the microorganisms isolated from blood or endocervical samples.

Conclusion: Severe immunosuppression was associated with increased length of hospitalization among women with HIV who had puerperal sepsis.

Keywords

Bacteriology; Clinical outcomes; HIV infection; Hospitalization; Immunosuppression; Puerperal sepsis

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Author contributions

RM was involved in conception and design of the study; data collection; analysis and interpretation of the data; and drafting the manuscript. ZMC and MFG were involved in conception and design of the study; analysis and interpretation of the data; and revising the manuscript for important intellectual content. All authors approved the final version of the manuscript for publication.

Conflicts of interest

The authors have no conflicts of interest.

1 INTRODUCTION

Puerperal sepsis causes 10%–12% of all maternal deaths in low-income countries such as Zimbabwe [1, 2]. This condition is defined by WHO as, “infection of the genital tract occurring at any time between the rupture of membranes or labor, and the 42nd day postpartum in which two or more of the following are present: pelvic pain, fever (i.e. oral temperature 38.5°C or higher on any occasion), abnormal vaginal discharge (e.g. presence of pus), abnormal smell/foul odor of discharge, and delay in the rate of reduction of the size of the uterus (<2 cm/day during the first 8 days)” [3].

The major risk factors for puerperal sepsis are cesarean delivery; immunosuppression (e.g. in response to HIV infection); prolonged and premature rupture of membranes; prolonged labor (>24 hours); frequent unsanitary vaginal examinations; retained products of conception; hemorrhage; anemia; malnutrition; and obesity [4, 5].

The worldwide incidence of puerperal sepsis is 2.7%–5.7% [3]. The prevalence of HIV infection is 16.7% among women in Zimbabwe aged 15–49 years [6]. Therefore, a need exists to assess the interaction of this high burden of HIV infection with puerperal sepsis among postpartum women. In a systematic review, Calvert and Ronsmans [5] noted that women infected with HIV had a greater than three-fold increased risk of developing puerperal sepsis than did women without HIV infection. This risk rose to six-fold when the analysis was limited to studies that assessed only women who had undergone cesarean delivery [5].

Infection with HIV has been associated with severe clinical manifestations in most studies of pelvic inflammatory disease [7]. Although evidence exists that HIV infection increases the risk of postpartum infections [5], there is insufficient information on the association of HIV infection with the bacteriological and clinical outcomes of puerperal sepsis.

The aim of the present study was to evaluate the association between puerperal sepsis and HIV-infection status among women in Zimbabwe.

2 MATERIALS AND METHODS

A subanalysis was performed using data from a prospective cohort study that had been conducted between September 2, 2014, and July 1, 2015, among women with puerperal sepsis who were admitted to either Parirenyatwa Hospital or Harare Central Hospital in Harare, Zimbabwe [8]. Both of these hospitals provided tertiary care. Approval to conduct the parent study and the present analysis was granted by the Harare Hospital Ethics Committee (Harare Central Hospital, Harare, Zimbabwe); the Joint Research Ethics Committee of the University of Zimbabwe College of Health Sciences and Parirenyatwa group of Hospitals (Harare, Zimbabwe); and the Medical Research Council of Zimbabwe (Harare, Zimbabwe). Written informed consent was obtained from all participants.

A convenience sample of consecutive women who met the WHO clinical criteria for puerperal sepsis [3] was used for the present subanalysis. The inclusion criteria were assessed through face-to-face interviews and clinical examinations. To be eligible for

inclusion women needed a clinical diagnosis of puerperal sepsis based on WHO criteria. The exclusion criteria were isolated extragenital infection and the inability to provide informed consent.

Demographic data were collected using an interviewer-administered questionnaire. An endocervical swab and a blood sample were collected for microbial culture and susceptibility testing at the time of hospital admission, preferably before administration of antibiotics [9]. Information regarding any prior antibiotic administration before sample collection was captured using a case investigation form [8]. Microbial culture and susceptibility testing were performed as described previously [8].

Testing for HIV infection is routine standard of care among Zimbabwean women during the prenatal period and breastfeeding, with such testing offered using an opt-out approach [10]. Both the HIV-infection status and the number of T cells expressing cluster of differentiation 4 (CD4) were obtained from a primary source document if testing was performed within 3 months and 6 months of presentation for puerperal sepsis, respectively. If no documented results were available, provider-initiated counselling and testing was offered via the public health laboratory using rapid HIV antibody tests. Women who had positive test results for HIV were subdivided on the basis of their CD4-positive T cell count: insignificant immunosuppression (>500/mm^3); mild immunosuppression (350–500/mm^3); advanced immunosuppression (200−349/mm^3); and severe immunosuppression (<200/mm^3) [11].

Women were admitted to hospital and managed as per routine standard of care by the on-call physicians. Results of microbial culture and sensitivity testing were provided by the present study researchers to the attending physicians, who used them at their discretion. Renal function was also tested at hospital admission and monitored as per the attending physician's discretion until hospital discharge. Women were followed-up until either discharge from hospital or death from peuerperal sepsis-related complications.

Medical records from local clinics, hospitals, and laboratories were reviewed to determine the clinical course of puerperal sepsis and identify appropriate outcome variables. Researchers retrieved paper copies of medical records for extraction into electronic storage to minimize loss of data.

The first outcome measure assessed the relationship between HIV-infection status and the type of bacterial isolates, the number of isolates per specimen, and the presence of MDROs. The second outcome measure assessed the association between time to onset of puerperal sepsis and the HIV-infection status, the CD4-positive T cell count, and the use of antiretroviral therapy (ART). Variables of puerperal sepsis included the development of complications such as wound dehiscence, peritonitis, pelvic abscess, septic shock, renal failure and death; the need for surgery; the need for admission to the intensive care unit; and length of hospital stay.

The data were analyzed using Entryware version 6.4 (Techneos, Vancouver, Canada) and SPSS version 16 (SPSS, Chicago, IL, USA). Patient characteristics were expressed as number (percentages). Descriptive statistics were expressed as mean (standard deviation) for quantitative variables that were normally distributed. Proportions, categorical values, and

means were compared using the *Z* test, Fisher exact test, and *t* test. A *P* value of less than 0.05 was considered to be statistically significant.

3 RESULTS

A total of 151 women met the WHO criteria for puerperal sepsis and agreed to participate. Valid HIV-infection status results were obtained for all of the participants. The HIV-positive group comprised 33 (21.9%) women and the group who did not have HIV comprised 118 (78.1%) women. The mean age among women with HIV was 27.0 ± 5.8 years, whereas the mean age of women without HIV was 24.6 ± 5.7 years (*P*=0.030).

The characteristics of the participants are outlined in Table S1. When compared with women who did not have HIV, women with HIV were more likely to be single (*P*=0.027); attend fewer prenatal clinic visits (*P*=0.006); deliver before term (*P*=0.035); and deliver at home (*P*=0.010).

Analysis of the potential association of HIV-infection status with the clinical course of puerperal sepsis is outlined in Table 1. No statistically significant between-group differences were found.

Valid CD4-positive T cell counts were available for 29 of the 33 patients who had HIV. One woman declined to undergo testing and three women were discharged from hospital before collection of a sample. The CD4-positive T cell counts were as follows: greater than 500/mm^3 (7 [21%]); 350–499/mm^3 (10 [30%]); 200−349/mm^3 (5 [15%]); and less than 200/mm^3 (7 [21%]). Severe immunosuppression (<200/mm^3; n=7) resulted in a mean hospital stay of 19.0 days versus 10.2 days for the other classes of immunosuppression (>200/mm^3, n=22; *P*=0.030). A nonsignificant trend toward early onset of puerperal sepsis in the severe immunosuppression group (2.5 days vs 11.8 days for the other classes of immunosuppression) was also observed (*P*=0.070).

Table 2 compares the level of immunosuppression with the clinical course of puerperal sepsis among the 29 women who underwent testing for CD4-positive T cells. Although increased rates were observed for most of the variables among women with severe immunosuppression, none of these differences reached statistical significance. Likewise, no statistically significant between-group differences were found among participants with CD4-positive T cell counts of 200–500/mm^3 (n=15) versus those with results of greater than 500/mm^3 (n=7; Table S2).

In all, 23 (69.7%) of the 33 women who had HIV were receiving ART. Infection with HIV was first diagnosed at the time of hospital admission for puerperal sepsis among the remaining 10 (30.3%) women who were not receiving ART. Seven (70%) of these 10 women had not attended any prenatal care visits and three (30%) had declined HIV testing during prenatal visits before the development of puerperal sepsis.

The mean time to onset of puerperal sepsis was 10.2 days among women using ART versus 7.8 days among women who were not using ART (*P*=0.724). The mean length of hospital

stay was 9.8 days and 14.5 days in the ART and no ART groups, respectively (*P*=0.316). As shown in Table 3, use of ART was not related to the clinical course of puerperal sepsis.

Of the 23 women receiving ART, 11 (48%) had been undergoing treatment for greater than 6 months and 12 (52%) had been undergoing treatment for less than 6 months. The mean time to onset of puerperal sepsis was 12.1 days among women who had used ART for less than 6 months versus 7.9 days among women who had used ART for greater than 6 months (*P*=0.331). The mean length of hospital stay was 11.7 days and 7.7 days among women who had used ART for less than 6 months or greater than 6 months, respectively (*P*=0.398). As shown in Table 4, duration of ART use was not related to the clinical course of puerperal sepsis.

The microbial findings among both groups are presented in Table 5. Bacterial growth on culture was observed for 103 of 151 (68.2%) endocervical swabs and 14 of 150 (9.3%) blood samples. The specimens were obtained before antibiotic use among 23 of 151 (15.2%) women. Overall, the most frequently detected microorganisms from the endocervical swab were Gram-negative enterobacteriaceae; in particular, *Escherichia coli* and *Klebsiella pneumoniae*, which comprised 57 (46.0%) of 124 total isolates. No statistically significant differences were found between the groups with regard to the type of organisms isolated from the blood or endocervical samples.

In addition, there was no between-group difference in the number of isolates per endocervical swab. One isolate was recorded among 63 (53.4%) women who did not have HIV versus 20 (60.6%) women with HIV (*P*=0.543); two isolates were recorded among 15 (12.7%) women who did not have HIV versus four (12.1%) women who did (*P*=0.928); and three isolates were recorded for one (0.8%) woman in the group of patients without HIV versus none (0.0%) in the group with HIV (*P*=0.596).

Finally, the proportion of women with MDROs did not differ between the two groups: there were three (9.1%) in the group with HIV versus 11 (9.3%) women who did not have HIV (*P*=0.968). The microbial culture results for the seven severely immunosuppressed women are as follows: five women had isolates from endocervical swabs; the three MDROs among HIV-infected women were found in two of the women with severe immunosuppression; one woman had *Klebsiella pneumoniae* and *Escherichia coli* which were both MDRO; one had MDRO *Enterobacter sp.* Other endocervical swab isolates were *K. pneumonia* (n=2) and *Kluyvera sp* (n=1). No bacterial growth was detected in the blood culture specimens for these seven women.

4 DISCUSSION

In the present study, severe immunosuppression arising from HIV infection was associated with an increased length of hospital stay for puerperal sepsis among women attending two tertiary hospitals in Zimbabwe.

Although infection with HIV did not appear to appreciably alter the clinical course of puerperal sepsis in the present study, severe immunosuppression among women with HIV was found to be a risk factor for prolonged hospitalization. A clinically significant trend

toward early onset of puerperal sepsis and increased complication rates was observed among women with severe immunosuppression. This finding was consistent with previous work by Cohen et al. [12] who showed that a CD4-positive T cell count of less than 14% was associated with an increased likelihood of a tubo-ovarian abscess and increased duration of hospitalization among women with acute salpingitis.

In the present study, use of ART did not independently affect clinical manifestations of puerperal sepsis. This finding could have been confounded by the duration of ART; immunologic and virologic status of the participants at initiation of ART; or adherence to medication, which was not measured in the current study. Use of ART is known to improve immunologic function among women with virologic control of HIV infection [13]. Wiewel et al. [14] did not find an effect of HIV infection on the clinical and pathophysiological course of sepsis among HIV-infected patients admitted to the intensive care unit when compared to their uninfected counterparts. Approximately 70.7% of these patients were already receiving ART when they developed sepsis [14], which is similar to the rate reported in the present study (69.7%). In the era of ART, women with HIV co-infections should be treated just as aggressively and without hesitation as their HIV-uninfected counterparts.

The rates of positive bacterial cultures from endocervical swabs (68.2%) and blood samples (9.3%) recorded in the present study were similar to previous work [4, 15]. Failure of microbial culture among patients with sepsis is not unusual and could reflect prior antibiotic administration; inadequate number of specimens collected; the presence of organisms requiring special nutritional and growth factors; and inappropriate specimen handling [4, 16]. Puerperal sepsis is a life-threatening infection that represents a major cause of maternal mortality in Zimbabwe. Therefore, it is usual practice to administer antibiotics before collection of specimens for bacteriological culture to avoid delaying treatment. In the present study population, specimens were obtained before antibiotic use in only 23/151(15.2%) women.

In the present study, HIV infection did not influence colonization with any of the microorganisms detected; the rate of polymicrobial colonization of the genital tract or blood stream; or the proportion of MDROs detected. The microorganisms causing infection of the upper genital tract are similar among HIV-infected and uninfected women [12, 17, 18]. In a case–control study, HIV-infected women exhibited more severe clinical manifestations of pelvic inflammatory disease than did their uninfected counterparts, with no effect on microbial cause or response to antibiotic treatment [18].

The present study found that women with HIV tended to be single mothers; attend fewer prenatal visits than women who did not have HIV; deliver at preterm; and deliver at home. The opportunity to detect and alleviate these interlinked risk factors for poor obstetric outcomes is missed when women do not attend prenatal care or fail to undergo delivery in the presence of a skilled healthcare provider. Therefore, it is essential to provide health education and affordable obstetric care in low-resource settings to encourage women to seek prenatal care.

The major limitation of the present study was the small number of women included in the group with HIV. This factor might have precluded detection of important differences in bacteriology and clinical outcomes based on HIV status, level of immunosuppression, and use of ART. Data on viral load were not available for all of the women with HIV; therefore, the effect of virologic control on the course of puerperal sepsis was unknown. Despite the above limitations, the present study provided preliminary data in a low-resource setting that should now be verified in a large study.

In conclusion, severe immunosuppression was found to be an independent risk factor for prolonged hospitalization among women with HIV who had puerperal sepsis. Clinicians managing severely immunocompromised women should be meticulous and vigilant for clinical complications.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Synopsis:

Severe immunosuppression in response to HIV infection was associated with prolonged duration of hospitalization for puerperal sepsis among women in Zimbabwe.

Table 1

Association of HIV-infection status with the clinical course of puerperal sepsis.[a]

Variable	HIV-positive group (n=33)	HIV-negative group (n=118)	*P* value [b]
Pelvic abscess	4 (12.1)	19 (16.1)	0.785
Septic shock	2 (6.1)	6 (5.1)	>0.99
Wound dehiscence	11 (33.3)	48 (40.7)	0.546
Renal failure	4 (12.1)	5 (4.2)	0.106
Peritonitis	1 (3.0)	3 (2.5)	>0.99
Death	4 (12.1)	7 (5.9)	0.456
Laparotomy	5 (15.2)	20 (16.9)	>0.99
Admission to the intensive care unit	6 (18.2)	12 (10.2)	0.214

[a] Values are given as number (percentage), unless indicated otherwise.

[b] Fischer exact test; $P<0.05$ was considered statistically significant.

Table 2

Association of the level of immunosuppression with the clinical course of puerperal sepsis among women with HIV (n=29). [a, b]

Variable	CD4-positive T cell count <200/mm^3 (n=7)	CD4-positive T cell count ≥200/mm^3 (n=22)	*P* value [c]
Pelvic abscess	2 (28.6)	2 (9.1)	0.238
Septic shock	1 (14.3)	1 (4.5)	0.431
Wound dehiscence	4 (57.1)	7 (31.8)	0.071
Renal failure	0 (0.0)	4 (18.2)	0.546
Peritonitis	1 (14.3)	0 (0.0)	0.241
Death	2 (28.6)	2 (9.1)	0.136
Laparotomy	3 (42.9)	1 (4.5)	0.082
Admission to the intensive care unit	3 (42.9)	3 (13.6)	0.290

Abbreviation: CD4, cluster of differentiation 4.

[a]Values are given as number (percentage), unless indicated otherwise.

[b]Valid CD4-positive T cell counts were available for 29 of the 33 patients who had HIV. One woman declined to undergo testing and three women were discharged from hospital before collection of a sample. The level of immunosuppression was defined as follows: CD4-positive T cell counts <200/mm^3 were classified as severe immunosuppression: 200−349/mm^3 classified as advanced immunosuppression; 350–500/mm^3 classified as mild immunosuppression; and >500/mm^3 classified as insignificant immunosuppression.

[c]Fischer exact test; $P<0.05$ was considered statistically significant.

Table 3

Association of antiretroviral therapy use with the clinical course of puerperal sepsis among women with HIV (n=33). [a]

Variable	Using ART (n=23)	Not using ART (n=10)	*P* value [b]
Pelvic abscess	3 (13.0)	1 (10.0)	>0.99
Septic shock	1 (4.3)	1 (10.0)	0.534
Wound dehiscence	8 (34.8)	3 (30.0)	>0.99
Renal failure	3 (13.0)	1 (10.0)	>0.99
Peritonitis	1 (4.3)	0 (0.0)	>0.99
Death	3 (13.0)	1 (10.0)	0.534
Laparotomy	4 (17.4)	1 (10.0)	>0.99
Admission to the intensive care unit	5 (21.7)	1 (10.0)	0.634

Abbreviation: ART, antiretroviral therapy.

[a] Values are given as number (percentage), unless indicated otherwise.

[b] Fischer exact test; $P<0.05$ was considered statistically significant.

Table 4

Association of duration of antiretroviral therapy use with the clinical course of puerperal sepsis. [a]

Variable	ART use <6 mo (n=12)	ART use >6 mo (n=11)	*P* value [b]
Pelvic abscess	2 (16.7)	1 (9.1)	>0.99
Septic shock	0 (0.0)	1 (9.1)	0.478
Wound dehiscence	4 (33.3)	4 (36.4)	0.400
Renal failure	1 (8.3)	2 (18.2)	0.590
Peritonitis	1 (8.3)	0 (0.0)	>0.99
Death	1 (8.3)	2 (18.2)	0.590
Laparotomy	3 (25.0)	1 (9.1)	0.586
Admission to the intensive care unit	3 (25.0)	2 (18.2)	0.635

Abbreviation: ART, antiretroviral therapy.

[a] Values are given as number (percentage), unless indicated otherwise.

[b] Fischer exact test; $P<0.05$ was considered statistically significant.

Table 5

Association of the microbial culture findings with HIV-infection status. [a]

Variable	HIV-positive group	HIV-negative group	*P* value [b]
Testing by endocervical swab	33	118	
Escherichia coli	11 (33.3)	27 (22.9)	0.392
Klebsiella pneumoniae	6 (18.2)	13 (11.0)	0.386
Klebsiella oxytoca	0 (0.0)	1 (0.8)	>0.99
Morganella morganii	0 (0.0)	2 (1.7)	>0.99
Providencia species	1 (3.0)	1 (0.8)	0.396
Pseudomonas aeruginosa	0 (0.0)	5 (4.2)	0.585
Alcaligenes species	0 (0.0)	3 (2.5)	>0.99
Bacillus species	0 (0.0)	2 (1.7)	>0.99
Citrobacter freundii	1 (3.0)	3 (2.5)	>0.99
Staphylococcus aureus	1 (3.0)	7 (5.9)	>0.99
CoNS	1 (3.0)	8 (6.8)	0.685
Streptococcus pyogenes	1 (3.0)	3 (2.5)	>0.99
Corynebacterium species	0 (0.0)	5 (4.2)	0.585
Enterobacter species	3 (9.1)	2 (1.7)	0.081
Salmonella species	0 (0.0)	1 (0.8)	>0.99
Kluyvera species	1 (3.0)	0 (0.0)	0.224
Yersinia species	0 (0.0)	2 (1.7)	>0.99
Group D streptococcus	2 (6.1)	6 (5.1)	>0.99
Streptococcus viridans	0 (0.0)	2 (1.7)	>0.99
Streptococcus agalactiae	0 (0.0)	1 (0.8)	>0.99
Shigella species	0 (0.0)	2 (1.7)	>0.99
No bacterial growth	9 (27.3)	39 (33.1)	0.839
Testing by blood culture	32	118	
Escherichia Coli	0 (0.0)	2 (1.7)	>0.99
Bacillus species	1 (3.1)	4 (3.4)	>0.99
Staphylococcus aureus	0 (0.0)	1 (0.8)	>0.99
Moraxella species	0 (0.0)	1 (0.8)	>0.99
Alcaligenes species	0 (0.0)	1 (0.8)	>0.99
CoNS	2 (6.3)	1 (0.8)	0.124
Fungal contamination	0 (0.0)	1 (0.8)	>0.99
No bacterial growth	29 (90.6)	107 (90.7)	>0.99

Abbreviation: CoNS, coagulase-negative staphylococcus.

[a] Values are given as number (percentage), unless indicated otherwise.

[b] Fischer exact test; $P<0.05$ was considered statistically significant.