Analysis of six major opportunistic infections to understand the high deaths among HIV/AIDS patients: A Case of Homa Bay District Hospital

by

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Abstract

Two-way analysis of variance tests the equality of population means when classification of treatments is by two factors or variables. Two-way ANOVA is possible if there exist one measurable and two nominal variables. Principles of experimental design were used in the layout, considering that disease distribution is addressed in terms of time, place and gender. Sampling techniques was used in the selection of the period of attack and cases studied. Duration between initiation of anti-retroviral drugs and an attack of an infection was the measurable variable of the two factors analyzed where cases considered had been on the therapy for more than six months. Gender at two levels and major opportunistic infections associated with HIV/AIDS at six levels were the factors in the design. Opportunistic infections associated with the HIV/AIDS epidemic based on gender were analyzed considering that funding for the fight against the disease is directed to incident rate reduction while deaths due to HIV/AIDS complications has continued to be high. Homabay district has a HIV prevalence rate of 17%. Reasons for the high deaths were unsatisfactory besides being less quantitatively statistical. We examine already infected society in order to guide and give clear situation analysis.

Chapter 1

Introduction

1.1 Background Information

Pneumocystis pneumonia, Cryptococcosis Extapulmonary, Encephalopathy by HIV, Extra pulmonary or disseminated TB, Herpes simplex and Kaposi sarcoma are among twenty one W.H.O stage four (major) opportunistic infections. These opportunistic infections if not detected and managed at an early stage lead to loss of life. Reported deaths of HIV/AIDS patients has been due to an attack of an opportunistic infection and failure to detect and manage the infection at an earlier stage. [6]. ART therapy has been a success in managing the HIV menace though for elimination of HIV/AIDS related deaths, focus should shift to investment on the detection and treatment/managemnt of opportunistic infections as a

result of HIV/AIDS. Currently, Extra pulmonary or disseminated TB as an aspect of TB umbrella is specially managed with almost sufficient resource allocation as opposed to other major opportunistic infections. We use ANOVA to understand if there do exist significant differences in the six opportunistic infections and gender.

Analysis of variance models (ANOVA) have been applied and used especially as a tool for experimental design, in a large variety of disciplines ranging from biostatistics to economics since Sir Ronald A. Fisher first developed it in the 1920's as a method for analyzing agricultural and biological data. The models have several advantages; they are generally robust and produce powerful tests [8]. ANOVA is a tool for estimating the effects of factors on a continuous response variable with the goal of detecting differences in means for different factor categories, called levels [20].

Focus in this study was entirely on fixed effects ANOVA model which is part of a larger set of general linear models including random effect models and mixed models. Thus, since factors are assumed to be fixed, levels of factors are not considered to be random samples from a larger

populations of levels. Hence, inference from this analysis is only valid within the specific population and factors included in the model [20]. Many situations involve a two way balanced ANOVA model taking the form

$$X_{ijk} = \mu + \alpha_i + \beta_j + r_{ij} + \epsilon_{ijk}, \tag{1.1}$$

where $\epsilon_{ijk} \sim N(0, \sigma^2)$ is the random error term taking care of uncontrollable factors, $i = 1, 2, \cdots, a$ while $j = 1, 2, \cdots, b$ and $k = 1, 2, \cdots, n$. where μ is the overall mean, α_i is factor A at ith level, β_j is the second factor$B$ at jth level,r_{ij} is the interaction effect between the ith level of factor A and jth level of factor B. From the model above, the factors investigated in our study were two; opportunistic infection at six levels and gender at two levels. The duration between the initiation of antiretroviral drugs and the attack of the disease was investigated for the two factors. Design of experiment was effective in its uniqueness in identifying treatment structure eventually facilitating the application of replication process.

In this study we analyzed using ANOVA six major opportunistic infections associated with HIV/AIDS patients' in Homa Bay District Hospital's Comprehensive Care Clinic. Homa Bay sub county has a high

HIV/AIDS prevalence rate of 17% [3] compared to national prevalence rate of about 6%. Statistic from the national Aids control council indicate that Nyanza province has one of the highest HIV prevalence rate's at 14%. HIV scourge has left 1.2 million orphaned from HIV related deaths [10]. Seven out of ten HIV infections are rural resident though the prevalence in rural areas is lower compared to urban, but the greatest burden of the disease is in rural areas since most Kenyans' live in rural areas [11]. Poverty level is high as a result of high dependency level caused by deaths of the productive generation. Through numerous interventions, the antiretroviral therapy is freely available but the HIV/AIDS related deaths has continued to be relatively high. This is against the expected norm as 60%-70% of the infected population is on antiretroviral therapy facilitating the shift in focus towards accelerated smart investment to achieve rapid success in AIDS response [21].

AIDS deaths in Kenya have a profound and increasing societal and economic impact; life expectancy in Kenya has dropped from 60 years in 1993 to 47 years in 2010 due to the impact of HIV/AIDS [2]. It was against this background that we were motivated to analyze the major opportunistic infections among the HIV/AIDS infected population. This analysis was

aimed at facilitating among other things the UNAIDS and WHO five-year strategies (2011-2015) aimed at building on progress in HIV/AIDS by: setting of ambitious plan; setting new targets; zero AIDS-related deaths through focusing on four strategic directions (Optimizing HIV prevention, leveraging broader health outcomes through HIV responses, building strong, sustainable health and community system, reducing vulnerability, removing structural barriers to access services and diagnostic treatment and care) [6]. Therefore, this study will be of use in resource allocation to combat HIV/AIDS deaths through smart dealing with the six major opportunistic infections analyzed.

1.2 Statement of the Problem

The awareness, prevention and treatment of the other major (W.H.O stage 4) opportunistic infections lag behind the awareness, prevention and treatment of tuberculosis despite continued deaths of HIV/AIDS clients already on antiretroviral therapy with good drug adherence.

1.3 Objectives of the Study

The following are the objectives of the study:

1. To understand if there is a significant difference in mean duration between attack of opportunistic infections and antiretroviral drug initiation among the HIV infected population based on gender.

2. To understand if there is a significant difference in mean duration between attack of opportunistic infections and antiretroviral drug initiation among six major opportunistic infections.

3. To understand if there is significant difference in mean duration between attack of opportunistic infections and antiretroviral drug initiation among the HIV infected population based on gender and opportunistic infection.

4. To understand the cause of high deaths among HIV positive patients despite free antiretroviral therapy.

The hypothesis tested include

1. H_0 - There is no significant difference in mean duration between attack of opportunistic infections and antiretroviral drug initiation

among the major six opportunistic infections.

i.e $H_0 : a_i = a_j = 0$ where $i \neq j \quad i = 1, 2, \cdots, 6$.

H_1 - There is a significant difference in mean duration between attack of opportunistic infections and antiretroviral drug initiation among the major six opportunistic infections.

i.e. $H_1 : a_i \neq a_j$ where $i \neq j \quad i = 1, 2, \cdots, 6$.

2. H_0 - There is no significant difference in mean duration between attack of opportunistic infections and antiretroviral drug initiation between males and females.

   i.e $H_0 : b_i = b_j = 0$ where $i \neq j \quad i = 1 \quad$ or $\quad 2$.

   H_1 - There is a significant difference in mean duration between attack of opportunistic infections and antiretroviral drug initiation between males and females.

   i.e. $H_1 : b_i \neq b_j$ where $i \neq j \quad i = 1 \quad$ or $\quad 2$.

3. H_0: Differences of mean duration between attack of opportunistic infections and antiretroviral drug initiation due to gender and one level of opportunistic infection equal the differences of mean duration of opportunistic infections and antiretroviral drug initiation due to gender at another level of opportunistic infections.

1.4 Research Methodology

This project was undertaken in homa Bay district hospital in Homa Bay County. Homa Bay County has one of the highest HIV prevalence at about 17% according to the ministry of Health survey report of 2012.

We used design of experiment which eventually facilitated use of two-way (6×2) ANOVA to understand six major opportunistic infections having HIV/AIDS patients as the cases of interest.

Data used was basically secondary data collected from patient files at the facility's comprehensive care clinic. Only patients who had been on antiretroviral therapy for at least six months and had good adherence before a new episode of a major opportunistic infection were considered in the study. In the study of diseases, time, place and gender are key towards disease distribution and this guided the design.

Sampling was used at every point to eliminate bias in the design. Six opportunistic infections were selected after ranking the morbidity in terms of frequency in a randomly selected year. The six opportunistic infec-

tions were selected from a list of twenty two world health organization (W.H.O) stage 4 opportunistic infections. Number six was selected randomly from the number 22. Stratified random sampling with equal allocation was used with the population divided into twelve strata; each cell was treated as a stratum for selection of units of interest. Stratification allowed the heterogeneous existence in terms of gender and opportunistic infection to be eliminated. Once in the strata, because of uniformity in the groupings of the cells in our layout, simple random sampling (random excel) was used to get unique identifiers for our layout. The written data capturing tool was structured to capture information relating to patients and the opportunistic infection. Health records officers helped us to extract the data from patients' files at the facility. The design was balanced in that the number of respondents/cases were equal for each cell i.e. $n_1 = n_2 = \cdots = n_{12}$. This implies $\sum n_i = n$ where i is the cell ranging from 1 to 12. The experimental units were the HIV/AIDS positive patients who had been on antiretroviral therapy for more than six months. The variable of interest was the duration between date of ART initiation and date of attack of major opportunistic infections grouped in terms of gender. To create non overlapping subpopulation we eliminated experimental units with double or multiple infections within the selected period, thus uniquely identifiable cases in the sample. We selected cases

that occurred in 2009 (through simple random sampling).

1.4.1 Overall Sample Size

Most of the available literature recommends that if the target population is less than ten thousand then the required sample size would be smaller. Since our population was about 8000 we adopted the formula where the estimate of the sample size is obtained as follows:

$$S = \frac{C}{1 + \frac{C}{\text{Population}}},$$

where

$$C = \frac{z^2[p(1-p)]}{D^2},$$

p is a true proportion of factor in the population or the expected frequency value of active clients with the opportunistic infections in 2009, D is maximum difference between the sample mean and the population mean or the expected frequency value minus worst accepted value, Z is the Z-score under normal curve corresponding to the confidence level of 95%.

With

$$Z = 1.96, \quad p = 5\%, \quad D = 0.05,$$

we have

$$C = \frac{(1.96)2[0.05(0.95)]}{0.05^2} = 72.9904.$$

Hence

$$S = \frac{72.9904}{1+\frac{72.9904}{6840}} = 72.2206 \cong 72.$$

But we note that when the sample size cell is equal, then each cell will have $n_i = 6$ i.e $(\frac{72}{12})$. This was a fairly good sample considering our target population of 400. We saw this necessary so that the discrepancy (sampling error) between the sample characteristics and population characteristics could be reduced. We distributed out 80 forms for data extraction so that we could meet our target of 72 should the forms be incomplete. Out of 80 forms given, only 75 were returned filled properly. From these we randomly eliminated 3 from the categories/cells which had excess.

1.5 Significance of the Study

Research can provide decision makers with a clear picture of relations and occurrences. In this, it will help to understand the distribution of

major opportunistic infections in the HIV/AIDS patients thus helping in the resource allocation in terms of gender and opportunistic infections. Finally, the study was expected to help in building sufficient skills to provide leadership in statistical methods in area of medical research.

Chapter 2

Literature Review

HIV/AIDS has continued to be a menace despite free anti-retrovirus therapy provided by the government and its partners. This has been noted to be as a result of opportunistic infections particularly the world health organization stage four opportunistic infections. Incidence trend for most frequent AIDS-defining opportunistic infections have been examined in many studies. In [9, 5],Trend occurred for 11 opportunistic infections. Among them were the major infections that we are looking at which included Kaposi Sarcoma, Extapulmonary Cryptococcosis, chronic Herpes simplex and tuberculosis. It was noted that the difference in trend was due to the difference in medical care offered to the patients considered and patients' adherence to preventive medication. In addition, analysis of HIV/AIDS on maternal and child health services which included

among other things fertility regulation, pregnancy, delivery and postpartum period was conducted. Consequently, analysis of the associations and trend in the frequencies of particular opportunistic infections revealed that the percentage of AIDS patients reported to have particular diseases varied with the patients behavioral and demographic characteristics [18]. According to studies [16, 15] ,understanding delineating factors associated with opportunistic infections can guide preventive interventions in HIV/AIDS patients through understanding the strength of association between the opportunistic infections and HIV/AIDS.

The proportion of patients presenting HIV - related care ranged from 20.2% to 24.5% according to Francis Kinyanjui. Mburu studied the impact of HIV/AIDS on mortality among the inpatients at Moteband Hospital, Lesotho. The average cost of treating HIV positive patients were significantly higher than those for non HIV patients. Despite widespread availability of HAART, Opportunistic illnesses (OI) still occurs and results in an increased risk of mortality among persons with AIDS. Estimation among new adult AIDS cases was associated with AIDS defining OI verses AIDS diagnosis based on low CD4 values only [14, 4]. Trend in infection between countries and regions highlight the complexity of

the HIV epidemic and the enormous prevention potential that exists in most countries though the HIV/AIDS burden is more in the sub saharan Africa compared to other regions [17]. Most Physicians and other care-givers have no speciality in the management of individual opportunistic infection. [7].

Design of experiments has facilitated use of powerful tests like analysis of variance in many fields. The (statistical) design of experiments (DOE) is an efficient procedure for planning experiments which facilitate analysis of data collected to yield valid and objective conclusions. The design of experiment begins with the determination of the experiment's objectives and selection of the process factors for the study. In our study the objective was based on understanding if their was significant difference in mean duration of; the six opportunistic infection, gender and any interaction effect. The factors we selected were opportunistic infections at six levels and gender at two levels. Such a process has been used in many studies in different fields like engineering, agriculture and industrial domain. Design of experiment method is key in any successful research for the determination of analysis method and allocation of experimental units. Most studies reveal that proper design of experiment is the goal to prob-

lem solving in the scientific research as it determines among other things the analysis process to be undertaken. [19].

Analysis of variance models (ANOVA) have been applied and used in a large variety of disciplines ranging from biostatistics to economics since Sir Ronald A. Fisher first developed it in the 1920's as a method for analyzing agricultural and biological data. ANOVA models are generally robust and produce powerful tests [8]. It is a tool for estimating the effects of factors on a continuous response variable with the goal of detecting differences in means for different factor categories (levels). Two-way ANOVA has been used in many particular studies over time. ANOVA was used in examining influences on teachers selection in the united states where descriptive factors were utilized to determine the impact/challenge index for minority and special population percentages of students, mobility indexes, and free and reduced price lunch percentages. Furthermore, data distinctive to each school's accreditation status in addition to adequate yearly progress were determined. ANOVA was used to determine if there was a significance difference between the said variables in the study [12]. In china, Two way ANOVA was used examination of the working conditions, challenges and tensions experienced by mathematical teachers.

Combination of psychological theories and organizational thinking was analyzed to understand the teachers' dilemma [13].

In conclusion, the project employs use of principle of experimental design to model six major opportunistic infections and gender in a balanced design enabling us to employ two-way analysis of variance to understand if there is significant difference between mean duration of attack (difference between opportunistic infection attack and anti-retroviral initiation) based on the two factors of interest.

Chapter 3

Basic Concepts

3.0.1 Modeling Process

Data was imported into SPSS (statistical package for social sciences version 17) for analysis from Excel spreadsheet used for data entry. Descriptive statistics was done for the measurable variable to get the basic statistics of mean, variance, range, maximum and maximum values. This was done for the measurable factor independently and also group/cell statistics.

Factor A (opportunistic infection) and Factor B (gender) were the categorical variables, while the duration (period) between ART initiation and disease attack as the quantitative aspect. Factor A was at six levels while

factor B at two levels. The ANOVA process was initiated based on the following assumptions.

3.0.2 Assumptions

- The variance of the populations was equal.
- The populations from which the samples were obtained were normally or approximately normally distributed.
- The samples were independent.
- The groups/cells have the same sample size.

3.0.3 Test for Homogeneity or Equal Variance

Levene's test of Homogeneity was used in accessing the equal variance in the population.

$$L = \frac{(N-k)\sum_{i=1}^{k} N_i(Z_{i.}-Z_{..})^2}{(k-1)\sum_{i=1}^{k}\sum_{j=1}^{N_i}(Z_{ij}-Z_{i.})^2},$$

where

- L is the calculated Levene test result,
- k is the number of different groups/cells to which the sampled cases belong. In our case this is 12,

- N is the total number of cases i.e, $\sum n_i = n$,
- N_i is the number of cases in the ith group i.e n_i,
- Z_{ij} is the value of the measured variable(duration between attack of opportunistic infection and ART initiation) for the jth case from the ith cell,

$\overline{Z}_{..} = \frac{1}{N}\sum_{i=1}^{k}\sum_{j=1}^{N_i} Z_{ij}$, and

$\overline{Z}_{i.} = \frac{1}{N_i}\sum_{j=1}^{N_i} Z_{ij}$.

are the grand mean and group mean respectively.

The significance of L is tested against $F(\alpha, k-1, N-k)$, where $k-1$, $N-k$ are the degrees of freedom as $k = 12$ and $N = 72$ and α is the level of significance which is 0.05.

Reject null hypothesis of equal variance if the resulting p-value of Levene's test is less than significance level α, where $\alpha = 0.05$. Thus the obtained differences in sample variances are unlikely to have occurred based on random sampling from a population with equal variances.

We proceeded with the decomposition of the total response variation into components that measure how much variation in the response is due to

opportunistic infection, gender, interaction between factors and random error.

Total sum of squares is given by $SS_{\text{Total}} = SS_A + SS_B + SS_{A\times B} + SS_{\text{Error}}$. The degrees of freedom due to total are $N - 1 = (a-1) + (b-1) + (a-1)(b-1) + ab(n-1)$, where SS_{Total} is Total sum of squares, SS_A is sum of squares related to opportunistic infection/factor A, SS_B is sum of squares related to gender/factor B, $SS_{A\times B}$ is sum of squares due to the interactions of factor A and B, and SS_{Error} is sum of squares due to random factors/error term.

The sum of squares formulae are as follows:

$$\begin{aligned} SS_{\text{Total}} = \sum_{i=1}^{a}\sum_{j=1}^{b}\sum_{k=1}^{n}(X_{ijk} - \overline{X}_{...})^2 &= nb\sum_{i=1}^{a}(\overline{X}_{i..} - \overline{X}_{...})^2 + an\sum_{j=1}^{b}(\overline{X}_{.j.} - \overline{X}_{...})^2 \\ &+ n\sum_{i=1}^{a}\sum_{j=1}^{b}(\overline{X}_{ij.} - \overline{X}_{i..} - \overline{X}_{.j.} + X_{...})^2 \\ &+ \sum_{i=1}^{a}\sum_{j=1}^{b}\sum_{k=1}^{n}(\overline{X}_{ijk} - \overline{X}_{ij.})^2. \end{aligned}$$

We proceeded to get the mean square which is the measure of variation as follows:

MEAN SQUARES (measures of variation)

The mean square for an effect is the effect sum of squares divided by the degrees of freedom.

$$MS_{\text{effect}} = \frac{SS_{\text{effect}}}{df_{\text{effext}}},$$

where the effects are A (opportunistic infection), B (gender), $A \times B$ (interaction effect.)

We divide all the MS_{effect} with the MS_{error} to get the ratios which are F-distributed. We calculate corresponding F-statistics and compare to a one tailed critical value from the F-distribution for our hypothesis tests.

$F_A = \frac{MS_A}{MS_E}$ for Ho: no effect of opportunistic infection on response variable (duration),

$F_B = \frac{MS_B}{MS_E}$ for Ho: no effect of gender on response variable, and

$F_E = \frac{MS_{A\times B}}{MS_E}$ for Ho: no interaction between opportunistic infection and gender.

We reject any H_0 if $F_{computed} \geq F_{Critical}$; otherwise, we do not reject H_0 i.e when the null hypothesis of "no effect" is true the mean squares are all estimates of σ^2, the common response variance for all treatment combinations.

3.0.4 Testing Effect Significance

For testing the main effects (A and B) and the interaction effect ($A \times B$) we simply compare the size of the MS_{effect} to the MS_{error}. If the $MS_{\text{effect}} \geq MS_{\text{error}}$ we have evidence that the effect is significant. If $MS_{\text{effect}} \approx MS_{\text{error}}$ then we have little evidence that the effect is significant.

To compare the mean squares we use the ratio, which has an F-distribution.

$$F_0 = \frac{MS_{\text{effect}}}{MS_{\text{error}}} \sim F - \text{distribution.}$$

$F_0 \gg 1$ will lead to the conclusion that the effect in question significantly impacts the response. Large F_0 values lead to small p-values which support effect significance.

Thus the ANOVA for the model in Equation (1.1) is summarized in the table below:

Table 3.1: Two-way ANOVA table

Source	Sum of Squares	df	MS	F
A	$\sum_{i,j,k}(\bar{x}_{i..} - \bar{x}_{...})^2$	a-1	$\frac{SS_A}{a-1}$	$F = \frac{MS_A}{MS_E}$
B	$\sum_{i,j,k}(\bar{x}_{.j.} - \bar{x}_{...})^2$	b-1	$\frac{SS_B}{b-1}$	$F = \frac{MS_B}{MS_E}$
AB	$\sum_{i,j,k}(\bar{x}_{ij.} - \bar{x}_{i..} - \bar{x}_{.j.} + \bar{x}_{...})^2$	$(a-1)(b-1)$	$\frac{SS_{AB}}{(a-1)(b-1)}$	$F = \frac{SS_{AB}}{MS_E}$
Error	$\sum_{i,j,k}(x_{ijk} - \bar{x}_{ij.})^2$	$ab(n-1)$	$\frac{SS_E}{ab(n-1)}$	
Total	$\sum_{i,j,k}(x_{ijk} - \bar{x}_{...})^2$	$abn-1$		

3.0.5 Groups/cells have the same sample size

In the balanced design all cell frequencies are equal, i.e. the number of observations in each combination of treatments is the same as illustrated in the table below:

Table 3.2: Balanced Design layout

	Factor B (Gender)	
Factor A (Opportunistic infection)	Male	Female
Cryptococcosis Extrapulmonary	$n = 6$	$n = 6$
Encephalopathy by HIV	$n = 6$	$n = 6$
Extrapulmonary and disseminated TB	$n = 6$	$n = 6$
Herpes simplex infection	$n = 6$	$n = 6$
Kaposi Sarcoma	$n = 6$	$n = 6$
Pneumocystis pneumonia	$n = 6$	$n = 6$

Chapter 4

Results and Discussion

4.1 Descriptive Statistics

The first step in the analysis was to explore the data. This was done by calculating the median, mean, range and standard deviation for all data looking at the duration on treatment for all cases as the point of interest irrespective of the factors. This was summarized in the table below.

	Range	Minimum	Maximum	Sum	Mean	SE	SD
Duration	73.59	6.70	80.30	1175.16	16.3217	1.34209	11.38802

Table 4.1: Descriptive Statistics

Table 4.1 above shows the statistics of the measurable variable. The duration illustrated is in months and the range is given as 73.59 months.

Consequently the mean is 16.3217 while standard deviation and standard error are 11.38802 and 1.34209 respectively. The range given by 73.59 is the difference between the maximum value for the duration given by 80.30 and a minimum value given by 6.70 months.

Figure 4.1 below shows a graphical representation of mean duration on treatment attack of the disease/opportunistic infections. Generally, pneumocystis pneumonia had a relatively high mean rating both for males and females while encephalopathy by HIV had the minimum rating in terms of mean duration. From the the figure 4.1, it was also evident that range in terms of mean duration between males and females was high in pneumocystis pneumonia and lowest in encephalopathy by HIV. Looking at the means individually based on gender, both males and females with an episode of encephalopathy by HIV had the minimal mean duration of attack while for the maximum mean values for the mean duration, pneumocystis pneumonia and herpes simplex infection was noted for males and females respectively. For males, the maximum mean rating was 38.56 and the minimum mean rating was 13.2 while for females the maximum was 28 while the minimum mean duration was 14.6 months.

Table 4.2 below shows a split of the means for the duration taken before

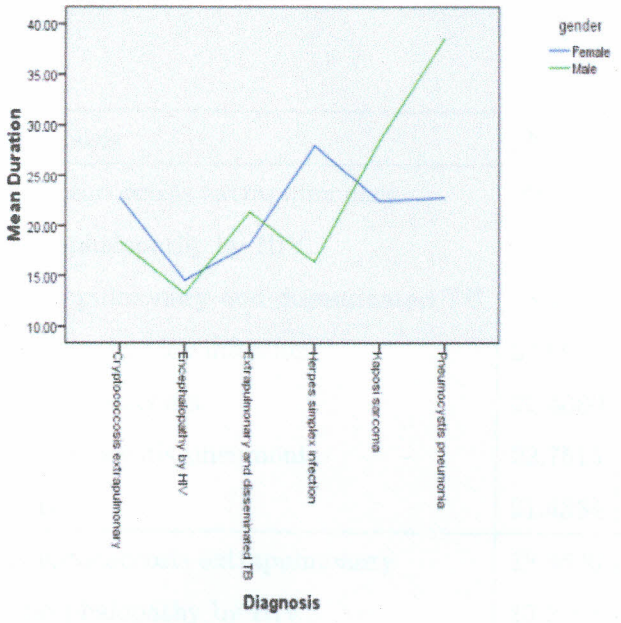

Figure 4.1: Mean duration on Treatment attack.

attack of an opportunistic infection and sex i.e. male and female. In addition to the means the standard deviations are also shown. The table also illustrates that for the totals ignoring the gender, pneumocystis pneumonia has the maximum mean rating of 30.7 months while encephalopathy by HIV had the mean minimum rating of 13.9 months.

gender	diagnosis	Mean	sd	N
Female	Cryptococcosis extrapulmonary	22.7936	25.74214	6
	Encephalopathy by HIV	14.5544	5.27725	6
	Extrapulmonary and disseminated TB	18.1027	14.95630	6
	Herpes simplex infection	27.9901	22.15424	6
	Kaposi sarcoma	22.4080	15.97572	6
	Pneumocystis pneumonia	22.7515	18.92385	6
	Total	21.4334	17.50704	36
Male	Cryptococcosis extrapulmonary	18.4600	12.94682	6
	Encephalopathy by HIV	13.2108	5.67983	6
	Extrapulmonary and disseminated TB	21.3285	15.79448	6
	Herpes simplex infection	16.3749	7.09450	6
	Kaposi sarcoma	28.4863	20.73143	6
	Pneumocystis pneumonia	38.5517	36.51794	6
	Total	22.7354	19.94500	36
Total	Cryptococcosis extrapulmonary	20.6268	19.55815	12
	Encephalopathy by HIV	13.8826	5.27399	12
	Extrapulmonary and disseminated TB	19.7156	14.76175	12
	Herpes simplex infection	22.1825	16.81570	12
	Kaposi sarcoma	25.4472	17.92894	12
	Pneumocystis pneumonia	30.6516	28.93142	12
	Total	22.0844	18.64456	72

Table 4.2: Table for group mean descriptive statistics

4.1.1 Homogeneity of Variance

We checked for homogeneity of variance using the Levene's test. This is illustrated below:

F	df_1	df_2	p-value
1.903	11	60	0.057

Table 4.3: Levene's Test of Equality of Error Variances.

From the table 4.3 above, the p-value is given as 0.057. The criterion being 0.05 which is less than the significance value of 0.057 which implies that the variances in the different experimental groups are not significantly different i.e. the variance in the groups are roughly equal.

4.1.2 Test of between-subjects effect

Table 4.4 below shows the main ANOVA summary table. It shows an effect of each independent variable which are referred to as main effects. The first row gives the heading of each column. For the main and the interaction effects, we are interested with the significance of the computed

Source	Type III Sum of Squares	df	Mean Square	F	p-value
Corrected Model	3274.120	11	297.647	0.834	0.607
Intercept	35115.844	1	35115.844	98.424	0.000
gender	30.513	1	30.513	0.086	0.711
diagnosis	1916.640	5	383.328	1.074	0.384
gender × diagnosis	1326.967	5	265.393	0.744	0.594
Error	21406.873	60	356.781		
Total	59796.836	72			
Corrected Total	24680.992	71			

Table 4.4: Tests of Between-Subjects Effects

F-value.

For gender as a factor, The experimental effect $SS_B = 30.513$ and this compares with the unsystematic variation in the data, or SS_E which 21406.873. This is converted to mean squares or average effect by dividing by the degrees of freedom of the respective source of variation which is 1 for gender and 60 for unexplained variation. Thus the effect in gender is simply the mean for gender divided by mean square error. This gives us a value of 0.086 as illustrated in the table. This is interpreted as

being the probability of getting an F-ratio of this magnitude by chance alone. In this case the probability is 0.711 which is higher than the cut off point 0.05 hence we can say that there was no significant effect of gender on the mean duration of attack. The above can also be illustrated by plotting a graph of the overall means for duration ignoring the type of opportunistic infection as illustrated below:

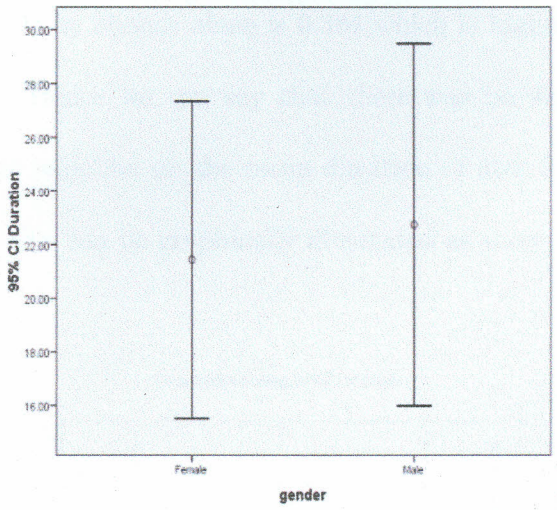

Figure 4.2: A Graph of Means duration based on Gender.

Consequently for the opportunistic infections noted as diagnosis in table 4.4, the experimental effect $SS_A = 1916.640$ and this compares with the unsystematic variation in the data, or SS_E which 21406.873. This is converted to mean squares or average effect by dividing by the degrees of

freedom which is $a-1=5$ for diagnosis and 60 for unexplained variation.

Thus the effect in diagnosis is simply the mean for diagnosis divided by mean square error. This gives us a value of 1.074 as illustrated in the table.

The output illustrated tells us that the probability of getting an F-ratio of this magnitude by chance alone is 0.384 which is higher than the cut off point 0.05. Hence we can say that there was no significant effect of opportunistic infection on the mean duration of attack. Ignoring the gender aspect, this can be graphically illustrated as shown below:

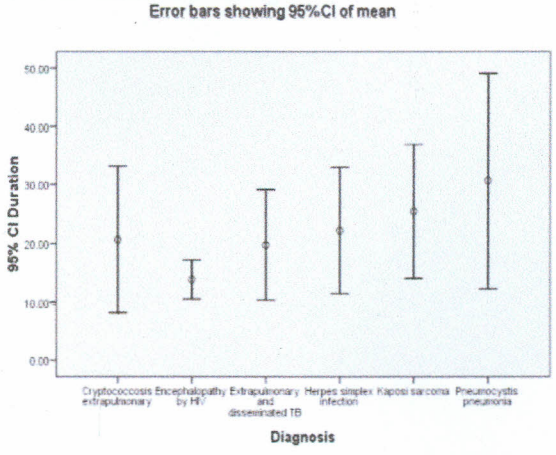

Figure 4.3: A Graph of mean duration based on opportunistic infections.

For the interaction effect between gender and opportunistic infection, The experimental effect $SS_{AB} = 1326.967$ compares with the unsystematic variation in the data, or SS_E which 21406.873. This is converted to mean squares or average effect by dividing by the degrees of freedom which is $(a-1)(b-1) = 5$ for interaction and 60 for unexplained variation. Thus the interaction effect (*gender* $\times$ *diagnosis*) is simply the mean for *gender* $\times$ *diagnosis* divided by mean square error. This gives us a value of 0.744 as illustrated in the table. The output illustrated tells us that the probability of getting an F-ratio of this magnitude by chance alone is 0.594 which is higher than the cut off point 0.05 hence we can say that there was no significant effect on the mean duration of attack between specific opportunistic infection and gender.

Chapter 5

Summary, Conclusions and Recommendations

From the analysis we note that there was no significant difference in the mean duration between the six major opportunistic infections and antiretroviral drug initiation among infected population based on gender. This implies that both males and females have no significant difference in mean duration of attack thus for decision making, no gender specific resources including clinics for the detection and treatment of any of the major opportunistic infections.

Consequently from the analysis, there was no significant difference in mean duration between the six major opportunistic infections and antiretroviral drug initiation among major six opportunistic infections. This

implies that HIV/AIDS patients after six months on ART therapy and good adherence can have an attach of any of the six opportunistic infection thus resources should be focused on capacity building, specialization and set up of different clinics for early detection and management of the six opportunistic infections.

We also take note from the analysis that there was no significant difference in mean interaction effect and thus mean duration of attack was relatively same for all opportunistic infections across all gender.

Lastly, from the above discussion, We conclude that the high deaths among the HIV/AIDS patients was as a result of non smart investment; concentration has been on the detection and management of extapulmonary TB with special clinics and experts specifically dedicated to this opportunistic infection and lack of the same resources in prevention, detection and management of other opportunistic infections. This biased investment facilitate the high deaths due to opportunistic infections. Thus we recommend that all HIV/AIDS care facilities through the government should have capacity to detect and handle all the six opportunistic infections effectively at the point of attack to avert mortality.

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