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Research Article

The significance of HIV status in patients with a spontaneous intracerebral hematoma taken for operative intervention in South Africa – A prospective cohort study.

Dr Adrian Kelly[1*], Professor Patrick Lekgwara[2], Professor Vanessa Moodley[3]

[1*]MMed Neurosurgery cum laude (SMU); FC Neurosurgery (SA) E-mail: adriankelly1000@yahoo.co.uk
[2]FC Neurosurgery (SA) E-mail: plekgwara@gmail.com
[3]MMed Haem cum laude (SMU) E-mail: vanessa.moodley@smu.ac.za

Abstract
Introduction: Patients diagnosed with a spontaneous intracerebral hematoma commonly have specific co-morbid medical and social risk factors.
Materials and methods: A prospective cohort study enrolling 38 HIV-negative and 32 HIV-positive subjects with a spontaneous intracerebral hematoma who underwent operative intervention. SMUREC/M/32/2022: PG.
Results: Significance was demonstrated between age category and HIV result where subjects in the HIV positive cohort were 10-20 years younger than in the HIV negative cohort (p=0.01). In the HIV positive cohort (n=32), 22/32 (69%) subjects demonstrated Stage II – IV HIV infection (<500 cells/mm2), and 17/32 (53%) subjects were not on HAART. Considering primary hypertension, a clinical trend was demonstrated where in the HIV positive cohort 26/32 (81%) had primary hypertension, while in the HIV negative cohort 28/38 (74%) had primary hypertension (p=0.45). Considering the location of the intracerebral hematoma 46/70 (66%) subjects had a basal ganglia bleed and 24/70 (34%) subjects had a lobar bleed. Considering the basal ganglia bleed location, in the HIV negative group 16/23 (70%), and similarly in the HIV positive group 15/23 (65%), had putaminal bleeds which was the commonest site (p=0.39). In the lobal bleed subset no significance was demonstrated regarding the location of the lobal bleed (p=0.64). Considering the etiology of the intracerebral hematoma in the HIV negative cohort 22/27 (81.5%), and in the HIV positive cohort 23/25 (92%) subjects incurred hypertensive bleeds (p=0.16).
Conclusion: In South Africa co-morbid uncontrolled primary hypertension and stage II-IV HIV infection predominated in our HIV positive cohort. Although HIV positive subjects and HIV negative subjects incurred the same etiology and location of disease, they were generally 10-20 years younger.

Author of correspondence Email: *adriankelly1000@yahoo.co.uk*

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Introduction

The cause of 2/3rds of primary spontaneous intracerebral hematomas is largely attributed to deep micro-perforator hypertensive cerebral small vessel disease. The common sites of primary spontaneous intracerebral hematomas are the basal ganglia, internal capsule, brainstem, and cerebellum [1]. Secondary spontaneous intra-cerebral hematomas are those that occur secondary into underlying disease processes and here hemorrhage associated with arteriovenous malformations, cavernomas, venous thrombosis, and aneurysmal parenchymal bleeds are the causes classically listed [1].

In HIV positive patients additional underlying causes of secondary spontaneous intracerebral hematomas include hemorrhage associated with toxoplasmosis, tuberculosis, primary cerebral lymphomas, cryptococcoses, high grade gliomas, metastatic Karposi's sarcoma, Varicella zoster vasculitis, autoimmune vasculitis, complicated neurosyphilis, and metastases. Regarding specific metastases papillary thyroid carcinoma, renal cell carcinoma, melanoma, germ cell tumors, and non-small cell lung carcinoma, are the most common histopathological subtypes associated with secondary spontaneous intracerebral hematomas [2-17].

Increasing age, male gender, hypertension, obesity, diabetes mellitus, hypercholesterolemia, renal insufficiency, tobacco use, and anti-coagulant use are each recognized to be independent risk factors for haemorrhagic cerebrovascular disease in both HIV negative and HIV positive patients [18-22]. In HIV positive subjects the plethora of specific underlying space occupying lesions, of which the spontaneous intracerebral hematoma is simply an epiphenomenon, makes histopathological examination of clot mandatory [23].

In a HIV positive patient, once a spontaneous intracerebral hematoma has been diagnosed, investigations should be directed towards identifying treatable underlying causes. Examples of treatable causes include toxoplasmosis, tuberculosis, cryptococcus, and varicella zoster. Towards this aim a pre- and post- contrast CT brain, MRI brain, chest X-ray, and in appropriate cases a lumbar puncture, should be performed. Additional ancillary tests directed towards establishing if these is an underlying lesion include sputum and CSF analysis [24]. Comprehensive CSF analysis should include Varicella zoster IgG quantitative assessment and CSF Varicella zoster DNA PCR; CSF treponemal antibody quantitative assessment; and if vasculitis is suspected quantitative anti-neutrophil cytoplasmic antibody assessment should be performed [25-29]. While these indirect ancillary tests may provide clues as to whether there is an underlying cause, biopsy, or histopathological clot analysis, may be indicated in inconclusive cases [23].

Nosocomial infections in neurosurgery are often life-threatening. Surgical site sepsis post cranial neurosurgery is reported to occur in 0–5%-7.2% of patients [30,31]. Lower respiratory tract infections are also common with several studies conducted in Neurosurgical intensive Care units reporting rates between 17% and 30% [32,33]. Studies have shown that at least 50% of nosocomial infections are preventable. The implementation of evidence-based guidelines are regarded a fundamental measure that lowers the nosocomial infection rate and its devastating consequences [34]. Preventative measures published by the Centre for Disease Control and Prevention (CDC) include appropriately timed administration of antimicrobial prophylaxis prior to skin incision, shortening the duration of operative time, and maintaining intra-operative euglycemia and normothermia [35].

Besides these interventional measures there are several factors outside of the neurosurgeon's control that contribute to the problem. Patient age, specifically being over the age of 60 years, is independently associated with an increased incidence of nosocomial infections. [36,37]. Being HIV positive and in an Intensive Care unit has also been linked to a substantially increased risk of acquiring a nosocomial infection. One study that compared nosocomial infections in HIV negative and HIV positive subjects in the Intensive Care Unit reported an overall nosocomial infection rate of 119/486 (24%) subjects. In this study, comparing the HIV positive cohort to the HIV negative cohort, the incidence of ventilator acquired pneumonia was 38.6% and 30.6% respectively, primary bacteremia was 22.8% and 22.4% respectively, urinary tract infection was 12.8% and 6.1% respectively, and surgical site infections were 1.4% and 0% respectively [38].

Besides HIV status itself, the absolute CD4 count is another important marker regarding the risk of acquiring a nosocomial infection. One study that considered 969 nosocomial infections over a 2-year period in Southern Brazil noted an absolute CD4 count below 50 cells/mm to be an independent significant risk factor for the development of a nosocomial infection. In this study AIDS patients with a CD4 < 50 cells/mm had a 50% incidence of nosocomial infection [39].

Materials and methods

The study design was a prospective cohort study performed by enrolling 32 consecutively admitted HIV positive patients with a spontaneous intracerebral hematoma who were taken for operative intervention. The study was conducted over a 24-month period from the 03 March 2022 – 30 March 2024. Additional subject inclusion criteria were subject age greater than 18 years, informed consent to be included in the study, and informed consent to be tested for HIV unless the subject was known to be HIV positive. Study exclusion criteria included age less than 18 years, subjects who did not give informed consent to be included in the study, and subjects who after appropriate counselling refused to be tested for HIV, and subjects admitted with an intracerebral hematoma who did not undergo operative intervention. To ensure data confidentiality a unique study number was given to each enrolled subject with no identifiable variable being recorded on each subject's individual data collection sheet. In total 32 subjects were enrolled in the study.

Pre-operative data collected in this study included subject age, gender, admission absolute CD4 count, primary hypertension, use of Highly Active Anti-Retro-viral Therapy (HAART), tobacco smoking, alcohol consumption, location of basal ganglia bleed, location of lobar bleed, intra-ventricular haemorrhage, and incidence of hydrocephalus. Regarding the admission CD4 count categories chosen in our study the World Health organization CD4 count staging was utilized where in HIV positive subjects: Stage 1 = CD4 > 500cells/mm^2; Stage 2 = CD4 500-350 cells/mm^2; Stage 3= CD4= 349-200 cells/mm^2; and Stage 4 = CD4 < 200 cells/mm^2. The WHO recommends HAART be instituted in HIV subjects with a CD4 < 500 cells/mm^2 [49]. In our study we considered a CD4 count < 500cells/mm^2 to be a pivotal discussion variable due to many of our subjects being in this admission CD4 count category, which according to the WHO guidelines is when HAART should be initiated. Regarding our study imaging findings these were standardised by the CT / MRI brain radiology report measuring the size of a particular subject's intracerebral hematoma to avoid selection bias.

Operative data collected included external ventricular drain insertion, craniotomy, and intra-operative blood loss. With regards the indications for surgical intervention clinical deterioration considered by the attending neurosurgeon to be secondary to the radiology report of obstructive hydrocephalus was the indication used for placement of an external ventricular drain. With regards the indications for evacuation of an intracerebral hematoma, although the ICH Score [50] was considered and utilized during family counselling for the purpose of the family understanding prognosis and thereby giving informed consent, the indications for evacuation of an intracerebral hematoma was largely at the discretion of the attending neurosurgeon. In our unit we utilise the following clinical and radiological guidelines namely a presenting Glasgow Coma Score of 10-13 with a frontal, parietal, or occipital lobe intracerebral hematoma with volume 30-60ml; Temporal lobe intracerebral hematoma with volume above 20ml; A posterior fossa intracerebral hematoma with a maximal transverse diameter > 2.5cm, as our local indications for surgical evacuation.

Post operative data collected included the two study outcome measures namely nosocomial infection incidence and Glasgow Outcome Score. This study comprised a descriptive and inferential analysis of each pre-operative and operative variable in relation to these two outcome measures in our HIV positive cohort and aimed to identify which variables demonstrated significance.

This study was granted approval by the Sefako Makgatho University Research Ethics Committee with Ethics Number SMUREC/M/32/2022/PG.

Results

Pre-operative variables

In total 32 HIV positive subjects were enrolled in the study. Regarding subject age category by admission CD4 count category no significance was demonstrated (p=0.31). A clinical trend was demonstrated where in HIV positive subjects who present with a spontaneous intracerebral hematoma taken for operative intervention, 12/32 (38%) were in the 4th decade of life, which was the peak incidence decade, and within in this sub-set 9/12 (75%) subjects had an absolute CD4 count < 500cells/mm2 [Fig 1]. Regarding gender in our HIV positive cohort 24/32 (75%) were male and 8/32 (25%) were female. No significance was demonstrated between gender and admission CD4 count category (p=0.91) [Fig 2]. Regarding admission CD4 count in HIV positive subjects, 3/32 (9.3%) demonstrated Stage IV HIV infection (CD4 < 200 cells/mm2), 8/32 (25%) demonstrated Stage III HIV infection (CD4 200-349 cells/mm2), 11/32 (34.4%) demonstrated Stage II HIV infection (CD4 350 - 500 cells/mm2), and 10/32 (31%) demonstrated Stage I HIV infection (CD4 > 500 cells/mm). Hence in the HIV positive cohort, 22/32 (69%) subjects demonstrated Stage II – IV HIV infection (<500 cells/mm2) [Fig 3].

Regarding primary hypertension 26/32 (81%) subjects had primary hypertension and 6/32 (19%) did not have primary hypertension. Regarding primary hypertension by admission CD4 count category, no significance was demonstrated (p=0.16). A clinical trend was demonstrated where in subjects with a CD4 < 500 cells/ml2, 17/26 (65%) had concomitant primary hypertension [Fig 4]. Considering HAART in HIV positive subjects, 15/32 (47%) subjects were on HAART and 17/32 (53%) subjects were not on HAART [Fig 5].

Regarding tobacco smoking and admission CD4 count category no significance was demonstrated (p=0.88). A clinical trend was demonstrated where in HIV positive subjects with CD4 count <500 cells/ml2, 9/22 (41%) admitted to smoking tobacco [Fig 6]. Considering alcohol consumption by admission CD4 count category, no significance was demonstrated (p=0.33). A similar clinical trend was demonstrated where in HIV positive subjects with <500 cells/ml2, where 9/22 (41%) admitted consuming alcohol [Fig 7].

In terms of the location of the intracerebral hematoma, 23/32 (72%) had basal ganglia bleeds and 9/32 (28%) had lobar bleeds. When considering basal ganglia hematoma location by admission CD4 count category, no significance was demonstrated (p=0.59). A clinical trend was demonstrated where in HIV positive subjects who incurred a putaminal basal ganglia bleed, 9/15 (60%) had an admission CD4 count <500 cells/ml2 [Fig 8]. Regarding the location of the lobar bleed no significance was demonstrated with admission CD4 count category (p=0.67). A clinical trend was demonstrated where in HIV positive subjects who incurred a parietal lobar bleed, 4/5 (80%) had an admission CD4 count <500 cells/ml2 [Fig 9].

On radiological review, considering the volume of the spontaneous intra-cerebral hematoma category by admission CD4 count category, no significance was demonstrated (p=0.62). A clinical trend was demonstrated where in HIV positive subjects who incurred a spontaneous intracerebral hematoma > 30ml, 11/19 (58%) had an admission CD4 count <500 cells/ml2 [Fig 10].

Regarding intraventricular haemorrhage 27/32 (84%) subjects had intraventricular haemorrhage and 5/32 (16%) did not have intraventricular haemorrhage. No significance was demonstrated between intraventricular hemorrhage and admission CD4 count category (p=0.70). A clinical trend was demonstrated where in HIV positive subjects who had intraventricular hemorrhage, 18/27 (67%) had an admission CD4 count <500 cells/ml2 [Fig 11].

When considering hydrocephalus by CD4 count category, 26/32 (81%) subjects developed hydrocephalus, and 6/32 (19%) subjects did not develop hydrocephalus. No significance was demonstrated between hydrocephalus and admission CD4 count category (p=0.69). A clinical trend was demonstrated where in HIV positive subjects who developed hydrocephalus, 18/26 (69%) had an admission CD4 count <500 cells/ml2 [Fig 12].

Operative variables

Considering external ventricular drain insertion, 26/32 (81%) subjects had an external ventricular drain inserted and 6/32 (19%) subjects did not have an external ventricular drain inserted. No significance was demonstrated between external ventricular drain insertion and admission CD4 count category (p=0.30). A clinical trend was demonstrated where in HIV positive subjects who had an external ventricular drain inserted, 17/26 (65%) had an admission CD4 count <500 cells/ml2 [Fig 13].

Regarding craniotomy, 12/32 (38%) subjects underwent a craniotomy, and 20/32 (63%) subjects did not undergo a craniotomy. No significance was demonstrated between craniotomy and admission CD4 count category (p=0.21). A clinical trend was demonstrated where in HIV positive subjects who underwent a craniotomy 6/12 (50%) had an admission CD4 count <500 cells/ml2 [Fig 14].

In terms of intra-operative blood loss, no significance was demonstrated between intra-operative blood loss category and admission CD4 count category (p=0.30). A clinical trend was demonstrated in that 4/6 (67%) of HIV positive subjects, who demonstrated > 500ml intra-operative blood loss, had an admission CD4 count of <500 cells/mm2 [Fig 15].

Post-operative variables

When evaluating the incidence of 30-day nosocomial infection, 11/32 (34%) subjects developed this complication, and 21/32 (66%) subjects did not develop this complication. No significance was demonstrated between nosocomial infection within 30-days of admission and admission CD4 count category (p=0.42) [Fig 16].

Regarding Glasgow Outcome Score, 3/32 (9%) subjects had a favourable outcome (GOS 4/5), and 29/32 (91%) subjects had an unfavourable outcome (GOS 1-3) and, overall, 16/32 (50%) of our HIV positive subjects demised in hospital. No significance was demonstrated between Glasgow Outcome Score and Admission CD4 count category (p=0.23) [Fig 17].

Discussion

Pre-operative variables

In our study a clinical trend was demonstrated where in HIV positive subjects who present with a spontaneous intracerebral hematoma taken for operative intervention, 12/32 (38%) were in the 4th decade of life, which was the peak incidence decade. Regarding gender in our HIV positive cohort 24/32 (75%) were male and 8/32 (25%) were female.

The epidemiological relevance of our study findings when considering age and gender is comparable to the results of one large observational study that utilized the North American Partners Health Care System Research Patient Data Registry. This study, comprising a U.S clinical care cohort, compared the incidence of hemorrhagic cerebrovascular events in 4251 HIV positive subjects to that is 35268 HIV negative subjects. The results of this study, on multi-variate analysis when considering subject age and gender, found that the highest significance was also demonstrated in HIV positive men in their 4th decade of life, with the validity of this result being ensured by adjustment being made for age and gender to make the two groups directly comparable. This study finding persisted after further adjustments were made for hypertension, diabetes mellitus, smoking, and anti-coagulation. Regarding gender this same study noted that while being female was protective in the HIV negative cohort, this did not afford protection in the HIV positive cohort whether being of either gender afforded a similarly increased risk when compared to HIV negative individuals [40].

In our study while no significance was demonstrated between primary hypertension and admission CD4 count category (p=0.16), a clinical trend was demonstrated where in subjects with a CD4 < 500 cells/ml^2, 17/26 (65%) had concomitant primary hypertension. Regarding the clinical significance of medical co-morbidities in HIV positive patients, several studies have demonstrated that there is an increased prevalence of essential hypertension in HIV positive patients when compared to HIV negative patients of comparable age. These same studies note that essential hypertension directly correlates with a HIV positive subjects overall increased cerebrovascular risk. This specific co-morbidity, in HIV positive patients admitted after incurring a haemorrhagic cerebrovascular event, is noted in several papers to be an independent co-morbid risk factor [18,19, 40].

In our study, regarding the location of the intracerebral hematoma, 23/32 (72%) had basal ganglia bleeds and 9/32 (28%) had lobar bleeds, neither of which demonstrated significance with admission CD4 count category. Our study finding is consistent with the findings of several studies that note that the intracranial deep basal ganglia micro-perforators in HIV positive subjects demonstrated leucoclastic vasculitis of the tunica media and vasa vasorum [41,42]. Chronic leucocyte mediate vasculitis of the deep micro-perforators is in turn recognized to mediate lipohyalinosis which is defined as a necrosing and subsequently fibrosing process of the small intra-cranial vasculature characterized by fibrinoid vessel wall necrosis and the histopathological presence of foam cells [43]. Both endothelial dysfunction and lipohyalinosis are postulated to explain the association between HIV infection and spontaneous intracerebral hematoma [23], the risk of which is significantly propagated by co-morbid hypertension [18,19].

Considering HAART in our HIV positive cohort, 15/32 (47%) subjects were on HAART and 17/32 (53%) subjects were not on HAART. Considering the clinical relevance of HAART on overall cerebrovascular risk, one retrospective study reported that in the short-term

HAART reduces the incidence of ischemic stroke [51], and therefore by the author's inference hemorrhagic conversion of the infarcted tissue. Other studies offer an opposing long-term view and report that long term HAART is toxic to endothelial cells with mounting evidence to suggest a causative link to vascular dysfunction [51,52]. In terms of pathophysiology direct endothelial toxicity and lipid modification are both proposed, however their direct link to clinical cerebrovascular disease has not yet been conclusively established in the literature [53]. Another study reports a similar view by reporting that HAART does not prevent HIV-associated endothelial dysfunction and on the contrary may be associated with both increased endothelial activation and increased endothelial inflammation. The authors of this study conclude that HAART has little to no anti-inflammatory effect on the endothelium, and therefore while these patients may live longer, they incur progressive endothelial changes increasing their cerebrovascular risk [44].

In our study, regarding tobacco smoking and admission CD4 count category, no significance was demonstrated (p=0.88). A clinical trend was demonstrated where in HIV positive subjects with CD4 count <500 cells/ml2, 9/22 (41%) admitted to smoking tobacco. The clinical relevance of our study finding is supported by several studies that have reported tobacco smoking to be a significant risk factors for the development of an ischemic stroke [54-56], which again by the author's inference hemorrhagic conversion of the infarcted tissue.

In our study, when considering intraventricular haemorrhage, 27/32 (84%) subjects had intraventricular haemorrhage and 5/32 (16%) did not have intraventricular haemorrhage. Regarding hydrocephalus 26/32 (81%) of our subjects developed hydrocephalus and 6/32 (19%) subjects did not develop hydrocephalus. In our study no significance was demonstrated between either the incidence of intraventricular hemorrhage and admission CD4 count category (p=0.70) nor hydrocephalus and admission CD4 count category (p=0.69). A clinical trend was however demonstrated where in HIV positive subjects who developed hydrocephalus, 18/26 (69%) had an admission CD4 count <500 cells/ml2. According to a PubMed review this clinical trend, namely that HIV positive subjects who incur a spontaneous intracerebral hematoma with intraventricular hemorrhage are more likely to develop hydrocephalus, than a similar group of HIV negative subjects, has never been described before.

Operative variables

Regarding craniotomy in our study 12/32 (38%) subjects underwent a craniotomy, and 20/32 (63%) subjects did not undergo a craniotomy. No significance was demonstrated between craniotomy and admission CD4 count category (p=0.21). The clinical relevance of only 1/3rd of our subjects undergoing a craniotomy is supported by several studies that have evaluated the role of craniotomy for supratentorial hematomas. These same studies recognize that although the role of a craniotomy to treat spontaneous ICH remains controversial, the use of this procedure to treat patients with supratentorial hematoma hematomas is still a relatively common strategy in most centers [59,60].

Post-operative variables

In our study, regarding the incidence of nosocomial infection, 11/32 (34%) subjects developed this complication, and 21/32 (66%) subjects did not develop this complication. No significance was demonstrated between nosocomial infection within 30-days of admission and admission CD4 count category (p=0.42). The clinical relevance of our study finding, namely that over 1/3rd of our subjects developed a nosocomial infection, is supported by the result of one study that reported that being HIV positive and in an Intensive Care unit is linked to an increased risk of acquiring a nosocomial infection when compared to subjects who are HIV negative [57]. Our study finding is however inconsistent with the results of another study, that considered 969 nosocomial infections over a 2-year period in Southern Brazil, which noted that only a much lower absolute CD4 count below 50 cells/mm to be an independent significant risk factor for the development of a nosocomial infection, which was not supported by the result of our study where we found a much higher CD4 count to carry an increased risk [45].

Regarding outcome, in our study 3/32 (9%) subjects had a favourable outcome (GOS 4/5), and 29/32 (91% subjects had an unfavourable outcome (GOS 1-3) and, overall, 16/32 (50%) of subjects demised in hospital. In our study no significance was demonstrated between Glasgow Outcome Score and Admission CD4 count category (p=0.23). Although not directly comparable as in our study because we only considered HIV positive subjects, our finding is in keeping with the findings of a meta-analysis, that considered 17 randomized clinical controlled trials, and found no benefit in primary outcomes to support routine surgical evacuation [46]. Several additional papers, again not directly comparable to our study for the same reason, but are nevertheless relevant, including the landmark STITCH II trial, support best medical care of these patients as the most effective management strategy to improve outcome, and reserve surgical evacuation for patients with a Glasgow Coma Score of 10-13 at presentation and hematomas that are large and superficial [47-48].

Conclusion

Our study highlights HIV positive subjects who present with a spontaneous intracerebral hematoma to peak in the 4th decade of life, and within this specific age decade subset, 65% had primary hypertension, and 75% had an absolute CD4 count < 500cells/mm2. Regarding the location of the intracerebral hematoma we note the similar clinical trend, already well known from studies considering HIV negative subjects, that HIV positive subject's trend towards incurring a putaminal bleed, with no significance being demonstrated this and the admission absolute CD4 count category. Regarding outcome, our positive subjects trend towards an unfavourable outcome, with no significance was demonstrated between this and the admission CD4 count category.

Study limitations

Limitations of our study are the relatively small sample size and that our study was a single-center experience. A further study limitation was that only uni-variate analysis was utilized. We emphasize the need for larger studies that employ multi-variate analysis to validate our study findings.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Figures

The significance of HIV status in patients with a spontaneous intracerebral hematoma taken for operative intervention in South Africa

Age categories

1=20-30 years
2=31-40 years
3=41-50 years
4=51-60 years
5=61-70 years
6=71-80years
7=81-90 years

Table of AGE_CAT by HIV

AGE_CAT	HIV		
Frequency, Col Pct	N	P	Total
1	7, 18.42	3, 9.38	10
2	1, 2.63	6, 18.75	7
3	6, 15.79	12, 37.50	18
4	7, 18.42	7, 21.88	14
5	13, 34.21	3, 9.38	16
6	4, 10.53	1, 3.13	5
Total	38	32	70

Statistics for Table of AGE_CAT by HIV

Statistic	DF	Value	Prob
Chi-Square	5	14.8160	0.0112
Likelihood Ratio Chi-Square	5	15.7976	0.0074
Mantel-Haenszel Chi-Square	1	3.4480	0.0633

Figure 1: Age category and HIV result (n=70)

Table of GENDER by HIV

GENDER / HIV Frequency, Col Pct	N	P	Total
F	14 36.84	8 25.00	22
M	24 63.16	24 75.00	48
Total	38	32	70

Statistics for Table of GENDER by HIV

Statistic	DF	Value	Prob
Chi-Square	1	1.1304	0.2877
Likelihood Ratio Chi-Square	1	1.1424	0.2852
Continuity Adj. Chi-Square	1	0.6477	0.4209
Mantel-Haenszel Chi-Square	1	1.1142	0.2912
Phi Coefficient		0.1271	
Contingency Coefficient		0.1261	
Cramer's V		0.1271	

Figure 2: Gender and HIV result (n=70)

Table of CD4_CAT by HIV

CD4_CAT / HIV Frequency, Col Pct	N	P	Total
1	0 .	3 9.38	3
2	0 .	8 25.00	8
3	0 .	11 34.38	11
4	0 .	10 31.25	10
Total	0	32	32

Absolute CD4 category

1= <200 cells/mm

2=201-350 cells/mm

3= 351-500 cells/mm

4= >500 cells/mm

Figure 3: Admission CD4 count in HIV positive subjects (n=32)

Table of HAART by HIV

HAART (Frequency, Col Pct)	HIV N	HIV P	Total
N	38, 100.00	17, 53.13	55
Y	0, 0.00	15, 46.88	15
Total	38	32	70

Figure 4: HAART in HIV positive subjects (n=32)

GCS (Frequency, Col Pct)	HIV N	HIV P	Total
2	0, 0.00	1, 3.13	1
3	0, 0.00	1, 3.13	1
4	3, 7.89	0, 0.00	3
5	4, 10.53	1, 3.13	5
6	2, 5.26	1, 3.13	3
7	1, 2.63	3, 9.38	4
8	4, 10.53	6, 18.75	10
9	3, 7.89	3, 9.38	6
10	3, 7.89	5, 15.63	8
11	2, 5.26	0, 0.00	2
12	3, 7.89	2, 6.25	5
13	2, 5.26	5, 15.63	7
14	7, 18.42	3, 9.38	10
15	4, 10.53	1, 3.13	5
Total	38	32	70

Statistics for Table of GCS by HIV

Statistic	DF	Value	Prob
Chi-Square	13	15.5188	0.2761

Figure 5: Admission GCS and HIV result (n=70)

Table of HPT by HIV

HPT HIV

Frequency, Col Pct	N	P	Total
N	10	6	16
	26.32	18.75	
Y	28	26	54
	73.68	81.25	
Total	38	32	70

Statistics for Table of HPT by HIV

Statistic	DF	Value	Prob
Chi-Square	1	0.5639	0.4527
Likelihood Ratio Chi-Square	1	0.5699	0.4503
Continuity Adj. Chi-Square	1	0.2165	0.6417

Figure 6: Primary hypertension and HIV result (n=70)

Frequency, Col Pct	N	P	Total
N	26	18	44
	68.42	56.25	
Y	12	14	26
	31.58	43.75	
Total	38	32	70

Statistics for Table of SMOKER by HIV

Statistic	DF	Value	Prob
Chi-Square	1	1.1022	0.2938
Likelihood Ratio Chi-Square	1	1.1017	0.2939
Continuity Adj. Chi-Square	1	0.6425	0.4228

Figure 7: Tobacco smoking and HIV result (n=70)

Table of ROH by HIV

ROH HIV

Frequency, Col Pct	N	P	Total
N	28, 73.68	17, 53.13	45
Y	10, 26.32	15, 46.88	25
Total	38	32	70

Statistics for Table of ROH by HIV

Statistic	DF	Value	Prob
Chi-Square	1	3.1981	0.0737
Likelihood Ratio Chi-Square	1	3.2082	0.0733
Continuity Adj. Chi-Square	1	2.3653	0.1241

Figure 8: Alcohol consumption and HIV result (n=70)

<u>Basal ganglia location</u>

C= caudate

P= putamen

T= thalamic

ICH_BG_LOC HIV

Frequency, Col Pct	N	P	Total
C	3, 13.04	1, 4.35	4
P	16, 69.57	15, 65.22	31
T	4, 17.39	7, 30.43	11
Total	23	23	46

Frequency Missing = 24

Statistics for Table of ICH_BG_LOC by HIV

Statistic	DF	Value	Prob
Chi-Square	2	1.8504	0.3964
Likelihood Ratio Chi-Square	2	1.9074	0.3853
Mantel-Haenszel Chi-Square	1	1.7551	0.1852

Figure 9: Location of basal ganglia bleed and HIV result (n=46)

Lobar bleed location

F= frontal

P= parietal

T= temporal

Table of LOBAR by HIV

LOBAR HIV

Frequency, Col Pct	N	P	Total
F	6, 40.00	2, 22.22	8
P	7, 46.67	5, 55.56	12
T	2, 13.33	2, 22.22	4
Total	15	9	24

Frequency Missing = 46

Statistic	DF	Value	Prob
Chi-Square	2	0.8889	0.6412
Likelihood Ratio Chi-Square	2	0.9119	0.6339
Mantel-Haenszel Chi-Square	1	0.8118	0.3676

Figure 10: Location of lobar bleed and HIV result (n=24)

Etiology.

Acom = aneurysmal

AVM = arteriovenous malformation

HT= hypertension

Table of AETIOLOGY by HIV

AETIOLOGY HIV

Frequency, Col Pct	N	P	Total
ACOM	0, 0.00	1, 4.00	1
AVM	5, 18.52	1, 4.00	6
HT	22, 81.48	23, 92.00	45
Total	27	25	52

Frequency Missing = 18

Statistics for Table of AETIOLOGY by HIV

Statistic	DF	Value	Prob
Chi-Square	2	3.6173	0.1639
Likelihood Ratio Chi-Square	2	4.2426	0.1199
Mantel-Haenszel Chi-Square	1	0.3208	0.5711

Figure 11: Etiology of intracerebral hematoma and HIV result (n=52)

Table of IVH by HIV

IVH / Frequency, Col Pct	HIV N	HIV P	Total
N	10 26.32	5 15.63	15
Y	28 73.68	27 84.38	55
Total	38	32	70

Statistics for Table of IVH by HIV

Statistic	DF	Value	Prob
Chi-Square	1	1.1792	0.2775
Likelihood Ratio Chi-Square	1	1.2023	0.2729
Continuity Adj. Chi-Square	1	0.6297	0.4275

Figure 12: Intraventricular hemorrhage and HIV result (n=70)

Table of HYDROCEPH by HIV

HYDROCEPH / Frequency, Col Pct	HIV N	HIV P	Total
N	14 36.84	6 18.75	20
Y	24 63.16	26 81.25	50
Total	38	32	70

Statistics for Table of HYDROCEPH by HIV

Statistic	DF	Value	Prob
Chi-Square	1	2.7862	0.0951
Likelihood Ratio Chi-Square	1	2.8564	0.0910
Continuity Adj. Chi-Square	1	1.9702	0.1604

Figure 13: Hydrocephalus and HIV result (n=70)

Table of EVD by HIV

EVD HIV

Frequency, Col Pct	N	P	Total
N	19 50.00	6 18.75	25
Y	19 50.00	26 81.25	45
Total	38	32	70

Statistics for Table of EVD by HIV

Statistic	DF	Value	Prob
Chi-Square	1	7.3889	0.0066
Likelihood Ratio Chi-Square	1	7.6818	0.0056
Continuity Adj. Chi-Square	1	6.0905	0.0136

Figure 14: External ventricular drain insertion and HIV result (n=70)

Table of CRANI by HIV

CRANI HIV

Frequency, Col Pct	N	P	Total
N	17 44.74	20 62.50	37
Y	21 55.26	12 37.50	33
Total	38	32	70

Statistics for Table of CRANI by HIV

Statistic	DF	Value	Prob
Chi-Square	1	2.1997	0.1380
Likelihood Ratio Chi-Square	1	2.2145	0.1367
Continuity Adj. Chi-Square	1	1.5446	0.2139

Figure 15: Craniotomy and HIV result (n=70)

Type of craniotomy performed.

DC = decompressive craniectomy

F= frontal craniotomy

O = occipital craniotomy

P = parietal craniotomy

PT = Parietal temporal craniotomy

T= temporal craniotomy

Table of DESCRIP_CRANI by HIV

DESCRIP_CRANI (Frequency, Col Pct)	HIV N	HIV P	Total
DC	2 9.52	5 41.67	7
F	8 38.10	2 16.67	10
O	3 14.29	0 0.00	3
P	2 9.52	2 16.67	4
PT	5 23.81	3 25.00	8
T	1 4.76	0 0.00	1
Total	21	12	33

Statistics for Table of DESCRIP_CRANI by HIV

Statistic	DF	Value	Prob
Chi-Square	5	7.4881	0.1868
Likelihood Ratio Chi-Square	5	8.7478	0.119[illegible]
Mantel-Haenszel Chi-Square	1	0.6678	0.4138

Figure 16: Type of craniotomy performed and HIV result (n=33)

Blood loss categories

1=0-500ml

2=501-1000ml

3=1001-1500ml

4=1501-2000ml

5=>2000ml

Table of BLD_LOSS_CAT by HIV

BLD_LOSS_CAT (Frequency, Col Pct)	HIV N	HIV P	Total
1	10 47.62	4 33.33	14
2	7 33.33	5 41.67	12
3	2 9.52	2 16.67	4
4	2 9.52	0 0.00	2
5	0 0.00	1 8.33	1
Total	21	12	33

Frequency Missing = 37

Statistics for Table of BLD_LOSS_CAT by HIV

Statistic	DF	Value	Prob
Chi-Square	4	3.7275	0.4441
Likelihood Ratio Chi-Square	4	4.6644	0.32[illegible]5
Mantel-Haenszel Chi-Square	1	0.5275	0.4676

Figure 17: Intra-operative blood loss category and HIV result (n=33)

Table of NOSOC_INFEC by HIV

NOSOC_INFEC HIV

Frequency, Col Pct	N	P	Total
N	20, 52.63	21, 65.63	41
Y	18, 47.37	11, 34.38	29
Total	38	32	70

Statistics for Table of NOSOC_INFEC by HIV

Statistic	DF	Value	Prob
Chi-Square	1	1.2086	0.2716
Likelihood Ratio Chi-Square	1	1.2159	0.2702
Continuity Adj. Chi-Square	1	0.7325	0.3921

Figure 18: Nosocomial infection and HIV result (n=70)

<u>**Site of nosocomial infection**</u>

BC = blood culture

TR= tracheal aspirate

W=wound

U=unknown

Table of SITE by HIV

SITE HIV

Frequency, Col Pct	N	P	Total
BC	6, 33.33	4, 40.00	10
TR	7, 38.89	6, 60.00	13
U	2, 11.11	0, 0.00	2
W	3, 16.67	0, 0.00	3
Total	18	10	28

Frequency Missing = 42

Statistics for Table of SITE by HIV

Statistic	DF	Value	Prob
Chi-Square	3	3.4749	0.3240
Likelihood Ratio Chi-Square	3	5.0933	0.1651
Mantel-Haenszel Chi-Square	1	1.9006	0.1680

Figure 19: Site of nosocomial infection positive culture and HIV result (n=28)

Table of NOSO_TIME by HIV

NOSO_TIME / HIV Frequency, Col Pct	N	P	Total
2	1	0	1
	5.56	0.00	
3	2	1	3
	11.11	10.00	
4	2	1	3
	11.11	10.00	
5	3	4	7
	16.67	40.00	
6	0	1	1
	0.00	10.00	
7	0	3	3
	0.00	30.00	
8	3	0	3
	16.67	0.00	
11	1	0	1
	5.56	0.00	
12	2	0	2
	11.11	0.00	
13	2	0	2
	11.11	0.00	
17	1	0	1
	5.56	0.00	
25	1	0	1
	5.56	0.00	
Total	18	10	28

Frequency Missing = 42

Statistics for Table of NOSO_TIME by HIV

Statistic	DF	Value	Prob
Chi-Square	11	14.7259	0.1954
Likelihood Ratio Chi-Square	11	19.2995	0.0559
Mantel-Haenszel Chi-Square	1	2.8916	0.0890

Figure 20: Days from admission until diagnosis of nosocomial infection and HIV result (n=28)

Table of GOS by HIV

GOS / Frequency, Col Pct	HIV N	HIV P	Total
1	22	16	38
	57.89	50.00	
2	0	2	2
	0.00	6.25	
3	11	11	22
	28.95	34.38	
4	3	3	6
	7.89	9.38	
5	2	0	2
	5.26	0.00	
Total	38	32	70

Statistics for Table of GOS by HIV

Statistic	DF	Value	Prob
Chi-Square	4	4.4659	0.3466
Likelihood Ratio Chi-Square	4	5.9816	0.2005
Mantel-Haenszel Chi-Square	1	0.0003	0.9865

Figure 21: Glasgow Outcome Score and HIV result (n=70)