Kiersten Anderson
Dietetic Intern- UCSD Medical Center
Clinical Nutrition Case Study
April 19, 2011

Gastric Kaposi Sarcoma in an AIDS Patient
A Case Study

Abstract

Human immunodeficiency virus (HIV) affects an estimated 33 million people worldwide. Progression of the disease to acquired immunodeficiency syndrome (AIDS) is associated with compromised nutrition status and increased caloric needs. As the infection attacks and weakens the immune system, secondary opportunistic infections and malignancies often occur, which further compromise nutritional status and nutrient provision. One such condition is Kaposi sarcoma, an opportunistic malignancy examined in this case study.

The patient, a young transgender male-to-female diagnosed with AIDS, presented with nausea, vomiting, and abdominal pain in November 2010. A gastric mass was discovered after an esophagogastro-duodenoscopy (EGD) was preformed and a subsequent biopsy diagnosed the patient with gastric Kaposi sarcoma. The position of the mass, coupled with the nutritional issues inherent with an AIDS diagnosis, complicated the provision of adequate nutrition with the patient. The medical nutrition therapy (MNT) included distal enteral nutrition, total parenteral nutrition, and oral intake as tolerated.

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) and its precursor human immunodeficiency virus (HIV) profoundly affect and alter the nutritional status of those infected. This results in an increased need for nutrition monitoring and intervention with disease progression, as wasting and complications associated with secondary opportunistic infections and malignancies occur (1). This case study will examine the effects on nutrition in a patient who presents with advanced AIDS complicated by gastric Kaposi Sarcoma. The nutritional interventions and rationale will be discussed throughout the progression of the patient's clinical course.

BACKGROUND OF DISEASE CONDITION

HIV and AIDS

Pathophysiology

HIV infection is marked by a decrease in the body's immune defense system as the virus attacks and destroys memory CD4/CD5 T-lymphocytes which play an integral role in immune function. As the number of CD4 cells decrease, the body becomes increasingly susceptible to opportunistic infections and malignancies. These infections can further compromise immunity and accelerate HIV progression (2). Progression to an AIDS diagnosis occurs when the CD4 count drops below 200 cells/mm^3 or the individual is diagnosed with an AIDS-defining illness (3).

Kaposi Sarcoma

Pathophysiology

A severe drop in CD4 cell count (<200 cells/mm^3) is associated with an increased risk of several cancers (1, 4). Of these, Kaposi Sarcoma (KS) is the most common malignancy to occur. Cancers and

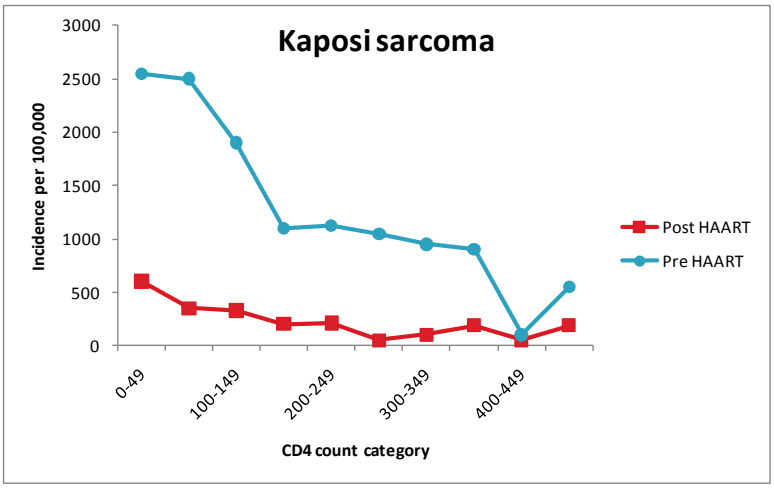
other opportunistic infections which occur at a CD4 count below 200 cells/mm^3 are considered AIDS-defining illnesses and signify a progression from HIV to AIDS (5, 6, 7, 8).

KS is caused by the Kaposi sarcoma herpesvirus (KSHV) and is either manifest as mucocutaneous or visceral malignancies. Skin lesions are the most frequently occurring type of KS while visceral lesions and masses are present in less than 40% of diagnoses. Visceral malignancies can include any portion of the gastrointestinal tract (GI) or the lungs (5, 9).

Incidence and Prognosis

Before the advent of combination or highly active antiretroviral therapy (cART or HAART), the risk for an individual diagnosed with HIV of acquiring KS was as high as fifty percent (7). The development of HAART in 1996 brought with it a rapid decline in the incidence of opportunistic infections and malignancies, including KS. This decline is reflective of improving CD4 counts in the HIV population, attributed to the widespread use of HAART which acts directly in preventing CD4 destruction (7, 8, 10, 11). However, the risk for KS remains high for those individuals with low or dropping CD4 counts. This may include those not receiving HAART treatment or those who are noncompliant (10).

Figure 1



Source: Biggar RJ, et al.

In the United States, KS occurs most frequently in men and is most common in the African American population (7, 8) (see Figure 2). The prognosis of KS without any treatment is very poor, with less than 10% of patients surviving at least five years after diagnosis. With treatment, mortality is drastically reduced with a 90% survival rate past five years of diagnosis with mucocutaneous KS and 50% in visceral KS (7).

Diagnosis & Treatment

Diagnosis of KS is made through biopsies of the tumor or lesions. The treatment for KS is determinant on the type of malignancy and location. Mucocutaneous KS is frequently treated with HAART alone, which has been shown to lead to a regression of the tumor(s) (10, 12). KS of visceral sites, however, is treated with a combination of HAART and chemotherapy, as HAART alone has not proven to have a significant effect (12).

INTRODUCTION OF CASE STUDY PATIENT

Background and Medical History

KB is a twenty-three year old, African American transgender male-to-female who presented to the hospital November 4, 2010 with chest and abdominal pain, fevers, weight loss, diarrhea, nausea and vomiting. The patient was homeless, working as a prostitute and estranged from his family at the time of admission. KB had an extensive medical history which included HIV/AIDS, methamphetamine abuse, and multiple opportunistic infections (see Table 1). The patient had been admitted to the hospital four additional times in the six months leading up to the current admission and during these was treated for various infections relating to his ongoing noncompliance with his HIV medications. The initial diagnoses at the current admission included AIDS and probable pneumonia.

INITIAL NUTRITION ASSESSMENT

KB was initially seen for evaluation of a weight loss identified by the nursing staff in the admission summary. During the assessment interview, KB reported a weight loss of 16 lbs over the previous three months. KB was visibly thin and ill in appearance. When asked questions, he answered softly, and was obviously in pain. An examination of previous admission charts confirmed a weight loss of 13 lbs since an admission two months prior to this interview. The patient attributed the weight loss to nausea, vomiting, and a loss of appetite. KB at this time had been placed on a regular diet and reported a fair appetite.

Table 2

KB at admission (11/4)				
Height	Weight	IBW	%IBW	BMI
72in (188cm)	158lb (72kg)	190lb (86kg)	83%	20

Laboratory Results

The laboratory results at admission are outlined in Table 3. Although most of the labs were within normal limits, they were significant for sodium, albumin and CD4. Prealbumin and C-reactive protein (CRP) were checked the following week and were also abnormal. The low albumin level is indicative of possible nutrient deficit over the previous few months. Although the prealbumin was low as well, it was not a reliable marker of nutritional status at that time given the CRP was elevated, indicating that the patient was in a current inflammatory or stressed state (13). The CD4 was low at 4 cells/mm3, reflective of KB's suppressed immune system.

Sodium remained consistently low throughout KB's hospitalization. This hyponatremia will not be extensively discussed, though it is noted that the medical team determined the hyponatremia to be secondary to either increased anti-diuretic hormone (ADH) or to decreased oncotic pressure. The hyponatremia was treated with replacement of hypotonic IV solutions with isotonic solutions and through minimizing free water given. Throughout admission, KS remained euvolemic.

Estimated Needs

Upon the initial nutrition assessment, KB's needs were estimated using the Harris Benedict equation for men, given that KB's frame size remained that of a man, regardless of his gender orientation.

Energy: 2520-2880kcal (35-40kcal/kg) Protein: 108-144gPro (1.5-2g/kg) BEE: 1840kcal

The estimated caloric needs were calculated in accordance with department policy (see Table 4) and correspond to his BEE x1.3-1.5 which reflects the anticipation that KB's needs would be increased secondary to his advanced AIDS and history of multiple infections.

Rationale

Caloric requirements are elevated in individuals with HIV/AIDS above that of the general population. This increase has been found to be linked not only to immunodeficiency; it occurs even in those whose CD4 counts are high and who are on HAART (14). However, the degree of increase in energy needs corresponds to the condition of the patient and stage of their disease. In all HIV patients there is an average of 8-15% increase in BEE. As the disease progresses to AIDS this changes to a 20-30% increase in caloric needs, and at least a 30% increase if opportunistic infections are present (3). Protein needs are also elevated when HIV is uncontrolled, with most studies showing at least a 33% increase which corresponds to about 1.5-1.8 g/kg. The protein requirements may be further exacerbated by infections (3, 15).

Given the patient's history and the possibility of current opportunistic infections (KB was admitted with fever), along with the weight loss, the dietitian formulated the following initial PES statement and plan:

D: Involuntary wt loss R/T loss of appetite and nausea/vomiting AEB 13lb weight loss x2mo

Goal: Pt to tolerate >75% of oral intake + supplements by next f/u

Plan:
1. Change diet to High kcal/High protein
2. Provide Ensure and snacks TID
3. Consider MVI
4. Check baseline prealbumin and then weekly to trend
5. Weigh pt 3x/week (MWF) to trend

TREATMENT AND MEDICAL COURSE

Overview

KB's hospitalization was characterized by a long, complicated course. After being admitted November 4, he was discharged briefly December 27, only to return the next day with worsening symptoms. He was again discharged March 17, returning March 22 with the same worsening of symptoms. KB currently remains hospitalized, now in his sixth month of hospitalization since November. Over this time period, KB developed several different infections (see Table 5), likely related to the fact that his CD4 count remained very low, <100 cells/mm^3, throughout this time.

Table 5

Hospital Problem List	
Main Diagnosis	
HIV/AIDs	
Opportunistic Infections/Malignancies	
Kaposi sarcoma	Sepsis
Esophageal Candidiasis	Hepatitis
Acute Pancreatitis	Perianal ulceration
Severe Luekocytosis	Intra-abdominal infection
Gram-positive bacteremia	Cytomegalovirus (CMV)
Mycobacterium avium complex	
Other	
Bascilar Atelectasis	Eosinophilia
Tachycardia	Hyponatremia
Nausea and Vomiting	Diarrhea

For the purpose of this case study, only one of these opportunistic infections/malignancies will be examined, as it dramatically affected his nutritional status.

Diagnosis of Kaposi Sarcoma

KS presented with nausea and vomiting and complaints of decreased appetite, along with abdominal pain. These symptoms were treated primarily with anti-emetics and an appetite stimulant,

with no apparent improvement during the first three weeks of hospitalization. On November 25th, a computed tomography (CT) scan of the abdomen revealed a gastric mass which had been missed on a previous CT scan. The differential diagnosis at the time was malignancy vs Candida vs Mycobacterium avium complex (MAC). An esophagogastroduodenoscopy (EGD) was performed and a biopsy taken Nov 26 which confirmed the mass to be Kaposi Sarcoma (9).

Reviewing the patient's history, it is evident that KB had the risk factors for developing KS. He is an HIV-positive African American male, non-compliant with his HAART medications, and had a CD4 count of 4 cells/mm^3 upon admission. This is well below 200 cells/mm^3, under which a person's risk for developing KS increases exponentially (1, 4, 7, 8, 10).

With the diagnosis of visceral Kaposi Sarcoma, plans were made with Hematology/Oncology to begin KB on a chemotherapy regimen along with his HAART medications. KB received six doses of chemotherapy over the course of his stay, a process often delayed by repeated infections, during which chemotherapy was contraindicated. Repeat EGD and CT-scans done in January and March demonstrated minimal change or reduction in the size of the mass.

NUTRITIONAL PROBLEMS AND INTERVENTIONS

Kaposi Sarcoma and Gastric mass

Impact on nutrition

Throughout the first weeks of KB's hospitalization prior to the diagnosis of Kaposi Sarcoma, oral food intake was minimal to none, due to persistent nausea, vomiting, and abdominal pain upon eating. In an effort to increase intake, his diet was changed from High Kcal/High Protein to mechanical soft, and then to full liquids as he was increasingly unable to tolerate most foods. Once Kaposi Sarcoma was diagnosed, the etiology of his symptoms became clear, as the mass was found to be almost completely blocking his gastric antrum.

Medical Nutrition Therapy

Once the mass was identified, the focus of nutrition therapy shifted to finding a way to bypass the mass in order to deliver nutrition, while optimizing oral intake. It was recommended to the medical team to place a naso-jejunal (NJ) tube in order to deliver nutrition past the gastric mass into the jejunum.

Rationale

Feeding distal to the stomach has been indicated in cases where a patient is intolerant of gastric feeds such as with high residuals or aspiration of gastric contents. By employing the use of distal feeds, the use of parenteral nutrition can often be avoided. KB was clearly unable to receive adequate nutrition via feeding into the stomach given the contents were frequently vomited. KB was determined to be an appropriate candidate for distal feedings (16).

Peptamen AF, a semi-elemental formula, was recommended due to the patient having chronic diarrhea. Although evidence is conflicting as to the superiority of semi-elemental formulas to polymeric formulas in cases of malabsorption (17), it was decided to act cautiously and recommend the semi-elemental formula. The rationale for this decision is based on the fact that individuals with HIV/AIDS are often afflicted with diarrhea, and, when coupled with poor nutrition this may contribute to the development of further systemic infections (15). The patient at this point had already had several weeks of very poor nutrition. Thus, the use of a semi-elemental formula as an effort to optimize nutrition was determined to be appropriate (see Table 6).

Complications

<u>Enteral and Parenteral Nutrition</u>

After the diagnosis of KS, the medical team agreed that placing an NJ tube would be the focus. However, both the Interventional Radiology and Gastroenterology teams were hesitant to place a tube

due to the difficulty of bypassing the mass. These delays resulted in the team deciding to start the patient on total parenteral nutrition (TPN) until the tube could be placed (see Table 6). Given the risks of infection with long-term TPN, especially in an immune-compromised patient (18), it was continually recommended to the medical team to initiate distal enteral nutrition as the gastrointestinal tract was fully functional past the gastric mass (16). An NJ tube was finally placed in mid-December and tube-feeds were initiated.

Further complications arose when the tube was determined to be non-functional after only two weeks of use. The tube was not replaced, and the patient was sent home days later on an oral diet. When the patient presented the following day with worsened nausea, vomiting, and abdominal pain it was apparent that an oral diet was not practical for the patient at this time. Again, it was recommended to re-initiate distal tube feeds. However, the team decided to place a percutaneous endoscopic gastrostomy (PEG) tube, although the dietitian again recommended to feed past the stomach. This tube was placed early January, delivering very little nutrition, until a gastro-jejunal (G-J) bridge was placed in February. This G-J bridge extended the PEG tube into the jejunum. KB was started again on TPN during this period (from January through February), though the PICC line had to be pulled twice due to line infections.

Oral Nutrition

KB remained for the most part on some form of oral diet, most frequently full liquids. His nausea and vomiting persisted throughout his hospitalization, however, and as a result his tolerance of these diets was very poor. Therefore, very little nutrition was achieved via oral intake. KB often refused to take any food by mouth, stating the fear that if he consumed anything, he would subsequently vomit and have increased pain. KB was on a very full anti-emetic regimen throughout his clinical course (included reglan, zofran, scopolamine, and phenergan), though the medications did not seem to

decrease his nausea and vomiting. The failure of the anti-emetics to alleviate his symptoms indicated that the presence of the gastric mass was the underlying cause.

HAART compliance

In addition to refusing oral intake of food, KB often also refused any oral medications. As a result, he received minimal amounts of his HAART medications during the periods when a functional feeding tube was not in place. The inability to consistently administer the HAART medications intravenously during these times (HAART was only available in oral form) likely was a large contributing factor in the failure of the gastric mass to respond to chemotherapy (12).

KB's consistently low CD4 count reflects the poor adherence to an HAART treatment plan as is evidenced by the long list of infections which KB acquired during his hospital course. Although the CD4 count showed indications of trending up, it remained well below the 200 cells/mm^3 threshold, as is shown in Figure 3.

The nutritional implications of KB's low CD4 count were evident given the continued presence of the gastric mass prevented significant oral intake. In addition to this very visible complication, a low CD4 count also places the body is in a persistently stressed state with altered metabolism. The increased caloric needs combined with a decreased intake leads to accelerated weight loss and a weakened body further susceptible to infections. The resulting cycle of increased malnutrition and poor immune function is illustrated in Appendix 3. Adherence to an HAART therapy would be a crucial component to any attempt to improve nutrition (15) (see Figure 4).

Wasting and Underweight

KB was not diagnosed with AIDS wasting during this hospitalization, nor did he meet all of the criteria outlined in Table 7, as his BMI never fell below 18.8. However, it is evident that KB remained at risk for under-nutrition and wasting, given his weight loss of 8% over 2 months at the time of admit and the presence of Kaposi Sarcoma and other opportunistic infections (15).

Table 7

Criteria for AIDS Wasting
BMI <18.5
Weight loss > 5% over 3 months
Lack of weight increase
Active opportunistic infections

Source: Szetela B, Gąsiorowski J

Compounding these factors, the prealbumin tested soon after admit was low at 9, indicating the possibility that visceral protein stores were low and that KB was at increased risk for under-nutrition.

Medical Nutrition Therapy

Every effort was made to provide nutrition via enteral feedings as it became evident that oral feedings alone were not warranted. When enteral nutrition was not possible, the TPN administered to KB was carefully monitored along with corresponding lab values to ensure appropriate nutritional provision. KB was on TPN for a total of two months and during this time received on average 40-80% of the recommended volume. The medical team was repeatedly notified of the inadequacy of TPN provided, though provision remained below goal.

When KB was consuming oral diets, the dietitian regularly assessed KB's po intake and provided oral nutritional supplements per his preferences. These included Resource Breeze, Ensure, and Juven.

KB reported attempting to drink some of these every day, though intake was inconsistent due to intermittent nausea and vomiting. KB was also recommended to be placed on a multivitamin early in his hospitalization, given poor oral intake.

Once the GJ tube was placed in February, access for nutrient provision improved greatly and TPN was discontinued. Actual provision of tube feedings was sub-optimal, rarely reaching full goal volume, though tube feeds were consistently well-tolerated (see Figure 5).

MNT Monitoring and Evaluation

Although nutritional access and provision was carefully monitored by the dietitian, actual intake remained inadequate through the hospital stay with the combination of poor oral intake and sub-optimal provision of TPN and EN (see Figure 5). This is further evidenced in the failure of prealbumin to reach normal limits and for weight to increase. The patient remained at severe nutrition risk throughout his stay as a result of the difficulty of providing adequate nutrition for his needs.

Prealbumin

Although prealbumin remained low throughout KB's hospitalization (with the exception of one value), the overall trend was positive, though widely fluctuating (see Figure 6). These fluctuations are reflective of the continuous series of secondary opportunistic infections that KB succumbed to. Prealbumin is decreased during times of serious infection, and each sharp dip in KB's prealbumin may correspond to these periods.

KB received several steroid and corticosteroid medications over his clinical course, including epinephrine. The use of these medications can result in elevated levels of prealbumin and may therefore mask low values. With the combination of a chronic inflammatory state due to systemic

infections and steroid therapy the use of prealbumin as an indicator of nutritional status in this patient is likely not valid (13).

Weight

In contrast to the results for prealbumin as shown in Figure 6, KB's weight followed a definite downward trend (see Figure 7). Although weight is often not regarded as an accurate indicator of nutrition status (2), this trend is likely a more accurate representation in this instance. KB did not have any fluid-related issues during his hospitalization, which frequently can render weight useless, nor was KB in a state where he would be dependent on the use of a bed scale for weighing (frequently prone to errors). However, it is important to note that the weighing methods (bed-scale vs standing scale) were often not specified.

PROGNOSIS AND DISCHARGE PLAN

Medical

Kaposi Sarcoma

The last dose of chemotherapy to this point was given in early April. The gastric mass still does not seem to have shrunk significantly and there are plans for further chemotherapy as an outpatient should KB be discharged, which is the pending plan. KB is now receiving all of his HAART medications through his GJ-tube and he has been receiving training on how to do this on his own once discharged.

All of the other infections that KB acquired while hospitalized have resolved and, medically, KB appears to be stable.

Nutrition

Nutrition continues to be inadequate as appetite has remained poor with nausea and vomiting continuing to present difficulties to oral feeding. The medical team hopes to discharge KB with nocturnal tube feeds and an oral diet. In an effort to ascertain the appropriateness of this plan, the

dietitian placed KB on a calorie count. This results of the calorie count showed that KB consumed 30-60% of his caloric needs orally. However, a sharp decline in caloric intake was noted at the end of the three-day calorie count as nausea and vomiting grew in severity. The wide fluctuations in oral intake, which seem to be representative of the past six months, do not at this time support the decision to provide only 50% of needs via nocturnal tube feeds.

If KB is to discharge, it is likely that he will go home on continuous or bolus tube feeds, in order to provide a consistent amount of nutrition as he continues to attempt to increase his oral intake. Once the HAART and chemotherapy are able to diminish the size of the gastric mass, it would seem appropriate to start KB on an appetite stimulant to try to optimize oral nutrition and, if possible, work towards discontinuing the tube feeds. At this time, the likelihood of this happening is slim, given the severity of the mass and KB's advanced AIDS.

Socioeconomic:

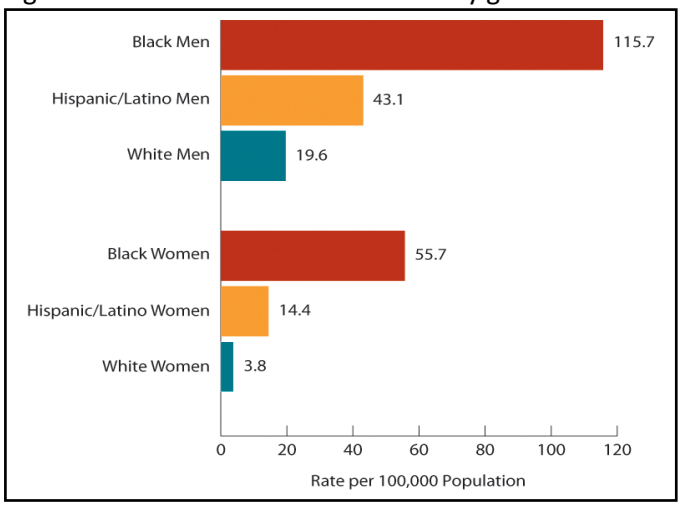
KB's lifestyle prior to admission necessitates a plan for living and food acquisition arrangements to be made prior to discharge. Since admit, contact has been made with KB's family and godmother. They have provided support and plan to assist KB upon discharge. Given the level of care needed at this point, KB will discharge to a facility which specializes in providing care to individuals with HIV/AIDS until they are able to return to independent living or until they die. If KB eventually returns to independent living, information on government and community programs for food provision would need to be provided.

SUMMARY AND CONCLUSION

The diagnosis of HIV and AIDS brings with it immediate and long-lasting effects on nutritional status. Caloric and energy needs are increased, especially with progression to AIDS, and when secondary opportunistic infections are present (3, 14, 15). This increase in needs, coupled with the frequent inability of these individuals to consume adequate amounts, often results in wasting and compromised nutrition. As was evidenced with this patient, opportunistic infections and malignancies can result in further complications to providing adequate nutrition. The challenge of increasing nutrient provision through oral intake, EN, or TPN requires close monitoring and intervention. However, when adequate nutrition is achieved, along with an adherence to HAART medications, rates of mortality are decreased and quality of life can be maintained (15).

Appendix

Figure 2 - Incidence of HIV in the U.S. by gender and ethnicity



Source: CDC HIV surveillance report

Table 1

Past Medical History
HIV/AIDS
Thrush
Pneumonia
Cytomegalovirus (CMV)
Candida esophagitis
Meth use
Mycobacterium avium complex

Table 3
KB- Labs at and soon after admission

Na	133↓
K+	4.1
Cl	100
Ca	9.1
Phos	3.2
BUN	8
Creat	0.7
GFR	>60
Glucose	87
Albumin	2.9↓
Prealbumin	9↓ (11/9)
CRP	25.2↑ (11/12)
CD4 count	4↓

Table 4

UCSD Policy - HIV/AIDS
Calories: ≥100% IBW: 25-30kcal/kg ≥90% IBW: 30-35kcal/kg ≤90% IBW: 35-40kcal/kg Protein: No less than 1.5g/kg (unless renal issues)

Source: UCSD Department of Nutrition- Policies and Procedure Manuel

Table 6

Nutrition Support:
Peptamen AF at at 85mL/hr Provides 2448kcal and 155g protein 97% minimum kcal needs 100% minimum protein needs **TPN Rx: D15 A5% at 100mL/hr + IL 20mL/hr x12 hrs** Provides 2184kcal and 120g protein 86% minimum kcal needs 100% minimum protein needs

Figure 3

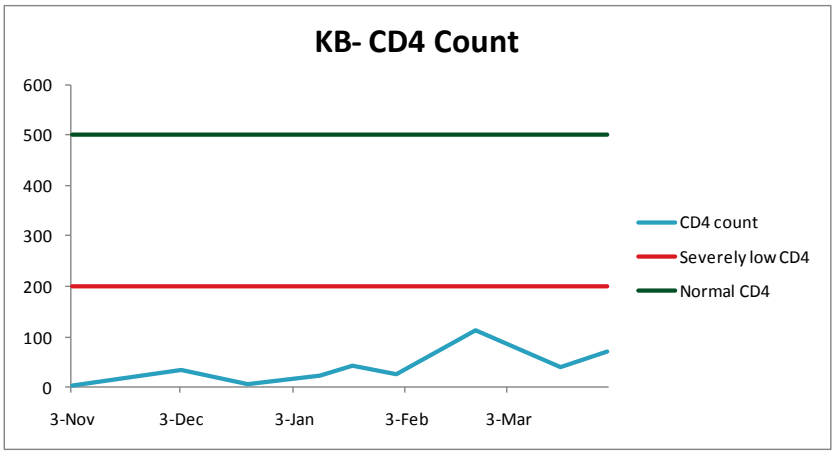
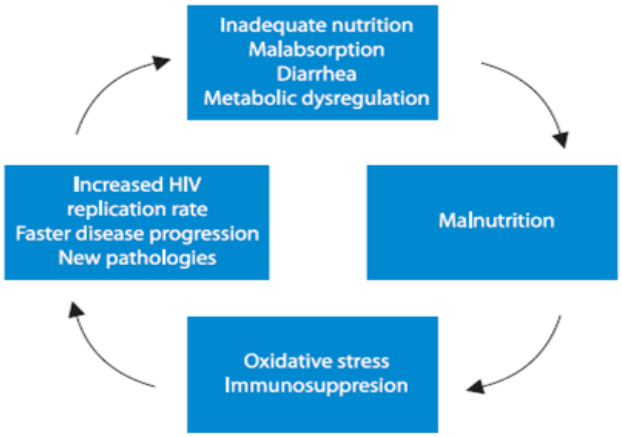


Figure 4



Source: Szetela B, Gąsiorowski J. Nutritional support for patients living with HIV or AIDS. HIV&AIDS REVIEW. 2010;9:79-82

Figure 5

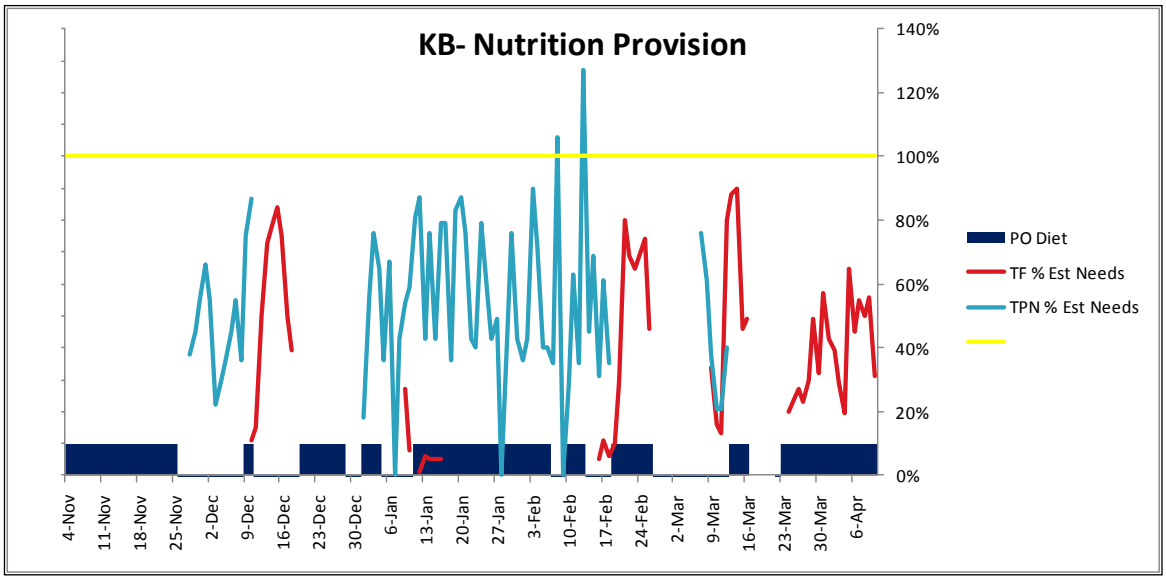


Figure 6

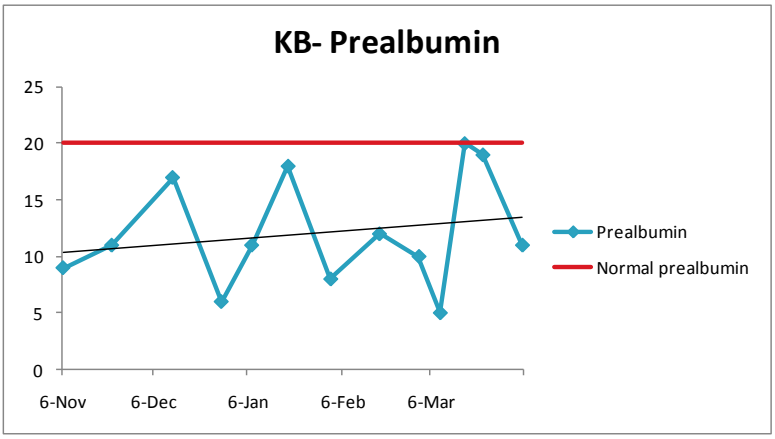
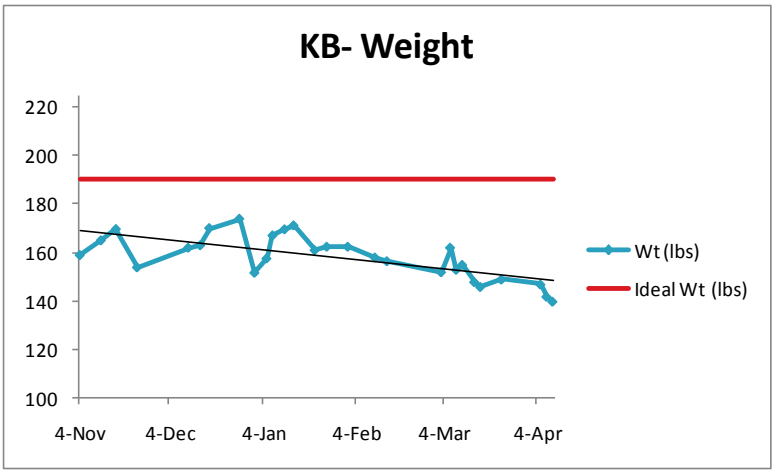


Figure 7



References

1. Fields-Gardner C, Campa A. Position of the American Dietetic Association: Nutrition intervention and human immunodeficiency virus infection. *J Am Diet Assoc.* 2010;110:1105-1119.
2. CDC HIV surveillance report: Diagnoses of HIV Infection and AIDS in the United States and Dependent Areas, 2009. http://www.cdc.gov/hiv/surveillance/resources/reports/2009report/ Accessed April 9, 2011.
3. Gottschlich MM, ed. *The A.S.P.E.N. Nutrition Support Core Curriculum*. Silver Spring, MD: A.S.P.E.N.;2007
4. Guiguet M, Boué F, Cadranel J, Lang J, Rosenthal E, Costagliola, D. Effect of immunodeficiency, HIV viral load, and antiretroviral therapy on the risk of individual malignancies: a prospective cohort study. *Lancet Oncol*. 2009;10:1152-1160
5. Attia S, Dezube BJ, Torrealba JR, Sosman JM, McHaffie DR, Pfau PR, Bailey HH, Kozak KR. AIDS-related Kaposi's sarcoma of the gastrointestinal tract. *J Clin Onc.* 2010;28:250-251
6. Chu KM, Mahlangeni G, Swannet S, Ford NP, Boulle A, Van Cutsem G. AIDS-associated Kaposi's sarcoma is linked to advanced disease and high mortality in a primary care HIV programme in South Africa. *J Int AIDS Soc*. 2010;13:1-5
7. Kaposi Sarcoma. American Cancer Society Website. http://www.cancer.org/cancer/kaposi sarcoma /detailedguide/kaposi-sarcoma-what-is-key-statistics. Accessed April 9, 2011.
8. Bower M, Weir J, Francis N, Newsom-Davis T, Powles S, Crook T, Boffito M, Gazzard B, Nelson M. The effect of HAART in 254 consecutive patients with AIDS-related Kaposi's sarcoma. *AIDS*. 2009;23:1701–1706
9. Di Lorenzo G, Konstantinopoulos PA, Pantanowitz L, Di Trolio R, De Placido S, Dezube BJ. Management of AIDS-related Kaposi's sarcoma. *Lancet Oncol.* 2007;8:167-176.
10. Biggar RJ, Chaturvedi AK, Goedert JJ, Engels EA. AIDS-related cancer and severity of immunosuppression in persons with AIDS. *J Natl Cancer Inst.* 2007;99:962–972.
11. Engels EA, Biggar RJ, Hall HI, Cross H, Crutchfield A, Finch JL. Cancer risk in people infected with human immunodeficiency virus in the United States. *Int J Cancer.* 2008;123:187–194.
12. Bihl F, Mosam A, Henry LN, Chisholm JV 3rd, Dollard S, Gumbi P. Kaposi's sarcoma-associated herpesvirus-specific immune reconstitution and antiviral effect of combined HAART/ chemotherapy in HIV clade C-infected individuals with Kaposi's sarcoma. *AIDS.* 2007;21:1245–1252.
13. Shenkin A. Serum Prealbumin: Is it a marker of nutritional status or of risk of malnutrition? *Clin Chem*. 2006;52.12: 2177-2179
14. Makola D. Elemental and semi-elemental formulas: Are they superior to polymeric formulas? *Pract Gastroenterol.* 2005;14-12:59-64
15. Szetela B, Gąsiorowski J. Nutritional support for patients living with HIV or AIDS. *HIV&AIDS REVIEW.* 2010;9:79-82
16. Krenitsky J. Gastric versus Jejunal Feeding: Evidence or emotion? *Pract Gastroenterol.* 2006;15.9:46-65

17. Madsen H, Frankel EH. Guide to parenteral nutrition management for adult patients. *Pract Gastroenterol.* 2006;15.7:46-68