ABSTRACT

Title of Document: PREVALENCE OF DYSPHAGIA AND COGNITIVE IMPAIRMENTS IN ADULTS WITH HIV/AIDS IN THE

ACUTE CARE SETTING: AN EPIDEMIOLOGICAL STUDY

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Human Immunodeficiency Virus (HIV) and Acquired Immunodeficiency Syndrome (AIDS) are life-threatening conditions. This virus causes the suppression of the immune system, allowing opportunistic infections and diseases to attack the body. Due to these opportunistic infections, individuals with HIV/AIDS are susceptible to conditions such as dysphagia (difficulty swallowing), odynophagia (painful swallowing), and cognitive impairment (e.g., HIV dementia). The comorbidity of these conditions is not clearly documented in the literature. Therefore, in this study, the prevalence of dysphagia (with and without cognitive impairment) in the HIV/AIDS population was determined by a review of 300 medical charts from patients admitted to a metropolitan hospital. Possible associations between the conditions and several demographic variables (e.g., age, CD4 cell count, recommended diet) were also explored. Twenty-one percent of the charts of patients with HIV/AIDS reported confirmed dysphagia and/or complaints of odynophagia. Of that 21%, only about five percent also had a documented cognitive impairment. This study supports previous prevalence estimates of dysphagia and odynophagia and reports prevalence of concurrent dysphagia and cognitive impairment, a potentially challenging complex. Lower CD4 counts were associated with the presence of dysphagia in this population. Individuals with dysphagia and cognitive impairments

were more likely to be older and were more likely to be recommended a restrictive diet consistency. Implications and recommendations for future study of this population are discussed.

PREVALENCE OF DYSPHAGIA AND COGNITIVE IMPAIRMENTS IN ADULTS WITH HIV/AIDS IN THE ACUTE CARE SETTING: AN EPIDEMIOLOGICAL STUDY

By

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Prevalence of Dysphagia and Cognitive Impairments in Adults with HIV/AIDS in the Acute Care Setting: An Epidemiological Study

Nutritional intake is fundamental to life. If a person is unable to transfer food from the mouth to the stomach, they most likely have a disorder known as dysphagia. Individuals with dysphagia may be unable to meet their dietary needs without clinical intervention. They may also be at risk of developing life-threatening conditions such as aspiration pneumonia and malnutrition.

Human Immunodeficiency Virus (HIV) is a virus that infects an individual's lymphoid system and causes deficiencies in their immune system. Therefore, the individual with HIV often has difficulty fighting off infections and life-threatening diseases. Some of the diseases and infections that this population is susceptible to can cause dysphagia and thus affect general nutrition and overall health.

Although many people can develop dysphagia, there are many unanswered questions regarding how HIV and Acquired Immunodeficiency Syndrome (AIDS, which is the end stage of HIV), affect the ability to swallow. For example, are HIV/AIDS patients especially at risk of developing dysphagia because of their compromised immune system? How often do individuals with HIV/AIDS develop dysphagia? What are the underlying causes of their dysphagia? How do specialists such as speech-language pathologists, who are closely involved in dysphagia care, treat this population?

With the exception of a few studies, the literature in this topic area is not clear in regards to the incidence/prevalence of dysphagia, the severity of dysphagia, or the role of the speech pathologist in the care of these patients. In order to better explore

and understand this topic, this study examines these factors as they relate to individuals with HIV/AIDS. It examines these questions by reviewing the medical charts of a population of patients at a large urban hospital with a significant HIV/AIDS patient population. This large population pool is unique because previous studies have only had small population sizes.

HIV/AIDS: Background

As of 2009, there have been an estimated 1.7 million people in the United States infected and living with the Human Immunodeficiency Virus (HIV) and over 468,000 living with Acquired Immunodeficiency Syndrome (AIDS) (Kaiser Family Foundation, 2009). HIV is a chronic, progressive viral infection that is transmitted through blood and blood products, seminal/vaginal fluid, and breast milk (Fitzgerald et al., 2010). HIV is a ribonucleic acid (RNA) virus that uses reverse transcriptase to convert viral DNA and infiltrate the host cell genome (Bell, 2004). HIV targets lymphocytes primarily by means of host cell surface CD4+ (cluster differentiation 4) and chemokine receptors (Bell, 2004). Acquired immunodeficiency syndrome (AIDS) is the terminal phase of HIV infection. The virus systemically weakens the body due to the destruction of the immune system.

This infection occurs in several phases. The first phase is the initial HIV infection, typically through blood or vaginal/seminal fluid transfer during sexual activity or intravenous drug use. The virus infects the CD4+ T cells and rapidly replicates, spreading to various organs such as the spleen and lymph nodes. This is evidenced through a high viral load and a low CD4+ cell count. Clinically, newly infected individuals may experience a brief period of flu-like symptoms or have no

symptoms at all. After this acute infection, the virus can lie dormant in the body for a period of time. Then, after two to four weeks, the immune system uses killer T cells, known as CD8+ T cells, and antibodies to fight against the virus, which results in reduced viral loads, and higher CD4+ cell counts. Next, the virus may go into a latency period in which HIV-related complications are absent, yet the virus continues to replicate in the lymphoid organs. Also during this time, depending on the individual, the virus may be active and the CD4 count can fall and/or the viral load level can increase. This leaves the individual susceptible to opportunistic infections or diseases. Therefore, medical intervention through HIV medication may be recommended. Finally, after an indeterminate amount of time, the infection can progress to AIDS. This occurs when the individual is immunocompromised to the point in which they can no longer fight off infection independently. The viral load once again increases and the CD4+ cell decreases (National Institutes of Health, 2009). The body is again susceptible to opportunistic infections that can cause more health complications.

Clinically, AIDS is defined as having at least 1 opportunistic infection and a critically low CD4+ cell count (Castro et al., 1992). The time between the initial HIV infection to the progression to AIDS varies per individual, ranging from five to twenty years (Uyl, van der Horst-Bruinsma & van Agtmael, 2004).

Medical professionals who work with HIV/AIDS patients use several tests to diagnose an HIV infection. One of the ways to diagnose and monitor the progression of the infection is through the cluster differentiation count (CD4), which is collected by a blood draw and analysis. This count indicates the health of the immune system

because CD4+ (which is found on the surface of helper T-lymphocytes) is a major receptor of the HIV virus (Fitzgerald et al., 2010). Physicians and researchers use this value to stage the progression of the virus and to determine when medicinal intervention is warranted. A healthy adult's CD4 count is between 500-1500 cells per micro liter. Individuals with HIV, especially in the asymptomatic stage or those who are under constant medical supervision with a medicinal regimen, can maintain a CD4 count in this range. It has been found that even if the CD4 count is greater than 500, individuals with HIV can have complications such as Acute Retroviral syndrome, candidial vaginitis, Guillian-Barre disease, or aseptic meningitis (Bartlett, Gallant, & Pham, 2010). Individuals with a CD4 count of 200-500 are still classified as having HIV, but they are more at risk of contracting opportunistic infections. In this range, it is normally recommended that the individual begin a medicinal regimen to help raise the CD4 count and decrease the viral load. Once the virus reduces the CD4+ T lymphocyte count below 200 cells/µ1³, the disease may now be classified as AIDS, and the individual is at the most risk of developing complications due to opportunistic infection (Castro et al., 1992). In summary, the Centers for Disease Control (Castro, et al., 1992) define the three CD4+ T-lymphocyte categories as follows:

- Category 1: greater than or equal to 500 cells/μl³
- Category 2: 200-499 cells/μ³
- Category 3: less than 200 cells/μl³

The HIV/AIDS population is continually surviving longer due to improved management of associated opportunistic diseases and due to the development of

highly active antiretroviral therapy (HAART), which incorporates drugs to block aspects of viral replication (transcriptase and protease) (Bell, 2004). HAART has the capability to improve immune function, decrease symptoms that manifest in the central nervous system (CNS) and prevent opportunistic diseases and infections (Bell, 2004).

Nonetheless, HIV/AIDS has the potential to cause devastating and debilitating diseases because the body's immune system is decimated. Common diseases and opportunistic infections include various cancers (e.g. Kaposi's sarcoma, lymphoma), pulmonary diseases (e.g. tuberculosis, pneumonia), gastrointestinal diseases (e.g. esophagitis, candidiasis), and neurological diseases and events (e.g. toxoplasmosis, progressive multifocal leukoencephalopathy (PML), cerebrovascular accident (CVA), etc.) (Castro et al., 1992). These diseases affect the individual's ability to carry out activities of daily living (ALDs) and each opportunistic disease has the potential to cause death if left untreated.

These diseases and infections often create a 'ripple effect' of disorders and symptoms for an individual with HIV/AIDS, meaning that one opportunistic disease can cause complications that include other disorders. For example, an individual who suffers a stroke or CVA because of the HIV infection may now have nerve damage, which could result in dysphagia, cognitive deficits, or several other neurological disorders. The effects of the initial opportunistic condition thus 'ripple' out to create other disorders. This study explores how frequently the 'rippled' disorders of dysphagia (impaired swallowing), odynophagia (painful swallowing) and cognitive

deficits (e.g., dementia, cognitive-motor disorders) affect individuals with HIV and AIDS.

HIV/AIDS and Dysphagia

Dysphagia is a disorder in passing food from the mouth to the stomach in which an individual may have difficulty swallowing and chewing. Dysphagia is not only a motor impairment but can include sensory, cognitive, and emotional difficulties that affect chewing and swallowing. In general, dysphagia can be caused by changes in the musculature, structure, or neurological (nerve) functions of the oral, pharyngeal, laryngeal, and esophageal structures and cavities. Inflammation of those oropharyngeal or esophageal structures occurs frequently in individuals with HIV, thereby causing dysphagia. Associated feeding and swallowing symptoms such as xerostomia (extreme dry mouth), aspiration (solid or liquid material entering the airway), and odynophagia also arise in individuals with HIV/AIDS. Dysphagia and odynophagia are often major complaints reported by individuals with HIV/AIDS (Bahl & Hickson, 1995).

Swallowing difficulties in this population are most often associated with the opportunistic infections of HIV/AIDS (Goldberg, 1993). HIV-related lymphomas or malignancies (e.g., Non-Hodgkin's lymphoma and Kaposi's sarcoma) may cause structural changes in the oral, pharyngeal, and esophageal cavities. The following inflammations and infections can also be the cause of structural changes: oral or esophageal candidiasis, oral thrush, herpes simplex virus (HSV), and cytomegalovirus (CMV). Candidiasis is the most frequent finding (Ranganathan & Hemalatha, 2006). Notably, candidiasis can be observed at the beginning of HIV infection, as a sign of

immunosuppression (Barr, 1992), and is seen in 90% of AIDS patients (Sanjar, Queiroz, Miziara, 2011; Vazquez, 2000). A meta-analysis of HIV studies found that the prevalence of head and neck symptoms in this population is about 80% and oral manifestations are the most frequent (as opposed to symptoms in the neck, sinuses, and ears) (Sanjar, Queiroz, & Miziara, 2011). While 80% of patients may not carry a formal diagnosis of dysphagia, several oral complications such as candidiasis or oral herpes zoster can cause dysphagia or odynophagia.

Most often, these infections and malignancies are treated through medication (antivirals, antifungals, antibiotics, and antituberculosis), chemotherapy, radiation, or surgery. There are also non-infectious causes of dysphagia in the HIV/AIDS population, such as pill impaction or ulcerations due to medication that does not dissolve and remains at the level of the esophagus. This finding appears common in patients who use the numerous HAART medications or even medications needed to resolve oral lesions (Bobba, El-Dika, & Arsura, 2007).

Changes or damage in the central nervous system (CNS) stemming from cerebrovascular accident (CVA) or stroke can cause neurogenic dysphagia, which is characterized by motor and sensory impairments of the oral or pharyngeal phases of swallowing (Buchholz, 1994). Generally, CVAs can cause dysphagia in about 51-55% of patients (Martino, et al., 2005). The prevalence of stroke in HIV patients ranges between 6-34% according to various studies (Rabinstein, 2003; Ortiz, et al, 2007). Stroke in this population does not only affect older individuals, as it has been found that young adults with advanced HIV appear to be at increased risk for either cerebral infarction or intercranial hemorrhage (Modi, Modi, & Mochan, 2006). This

could indicate that dysphagia in this population may occur regardless of age, especially when the etiology is from stroke. Individuals with CNS involvement are also at risk for increased fatigue, which in turn may affect swallowing function and nutritional intake (Logemann, 1998). In addition, neuromuscular impairments seen at times in individuals with HIV, such as Bell's palsy and facial nerve paralysis, may make chewing and swallowing difficult (Bladon & Ross, 2007), causing a diagnosis of dysphagia.

Previous studies of patients with HIV/AIDS have not been able to determine the incidence/prevalence specifically associated with dysphagia. Studies have discussed the presence of oral lesions because they greatly affect quality of life and can be indicators of the progression of immunosuppression (Barr, 1992; Sanjar, Queiroz, & Miziara, 2011). Some studies have reported dysphagia or odynophagia as a complaint of patients with HIV (Chelbowski et. al, 1989; Anteyi, et al., 2003; Bladon & Ross, 2007; Tirwomwe et al., 2007). One study noted dysphagia in 21% of a cohort of 71 adult males (Chelbowski et. al, 1989). Another study reported odynophagia (usually due to esophageal involvement or oral lesions) in 22% of cases (Anteyi, et al., 2003). Bladon and Ross (2007) found other complaints of swallowing difficulty in 79.2% of their sample of 120 patients with HIV. These included symptoms such as heartburn, dry mouth, nasal regurgitation, and food falling out of the mouth. Percentages for classic symptoms of dysphagia in their study were as follows: difficulty chewing (17.5%, n=21), difficulty swallowing (20.8%, n=25), pain on swallowing (24.2%, n=29), coughing while eating or drinking (15.5%, n=21), and choking while eating or drinking (8.3%, n=10) (Bladon & Ross, 2007). These data

were gathered through patient report and were not confirmed by a speech language pathologist or instrumental examination. Researchers have focused more on the infections that cause dysphagia rather than the swallowing disorder itself. Thus, there is little information on exact incidence of HIV and dysphagia in a large sample.

Because difficulty in swallowing limits caloric intake, dysphagia is a symptom or contributing factor to severe weight loss in individuals with HIV/AIDS (Jacobson, et. al, 2003). Acute weight loss in this population was associated with oral symptom complexes (mouth sores, thrush) and trouble swallowing (Jacobson et al., 2003). In this study, the researchers conducted semiannual telephone interviews with 415 patients about weight loss and physical symptoms of HIV (e.g. genital tract, upper and lower respiratory tract, and gastrointestinal symptoms). Almost 6% of participants reported trouble swallowing as a complaint and oral system complex complaints were reported by 6.7%. The authors found that acute weight loss was associated with oral symptom complexes (mouth sores, thrush) and trouble swallowing (Jacobson et al., 2003). They concluded that 15% of the increased risk of acute weight loss was explained by the occurrence of oral symptom complex and trouble swallowing, among other symptoms in HAART recipients.

In addition to the connection between dysphagia and weight loss, researchers have begun to investigate whether or not dysphagia is under-diagnosed in patients with HIV. Patients may only be treated for the infectious etiology and not examined further in regards to the effects that the dysphagia may cause. In a study by Halvorsen and colleagues in 2003, swallowing problems were present in HIV patients whose videofloroscopic exam did not display esophagitis, which in this study

encompassed infections such as Candida albicans, herpes simplex or CMV. They observed swallowing dysfunction in all of the phases of the swallow and documented both frank aspiration and silent aspiration. These findings suggest that even patients who do not present with indicators of esophagitis upon physical examination but do complain about difficulty swallowing should have further instrumental testing; the speech-language pathologist should be involved in their patient care. It may also be surmised from this study that many patients may have dysphagia and silently aspiration, but they are not being referred to the speech pathologist for further evaluation. Halvorsen and colleagues also found that several patients who had recent CT or MRI examinations of the brain had various CNS infections such as CNS toxoplasmosis, multiple infarcts, or CNS lymphoma. They postulated that these etiologies might be the underlying cause of the subjects' dysphagia. This finding and Bladon and Ross' (2007) finding that some individuals with a CD4 count greater than 500 also complained of swallowing difficulties suggests that dysphagia can occur early in the course of HIV infection. This may support early SLP involvement with individuals with HIV/AIDS to further screen or evaluate swallowing to avoid silent aspiration.

Bladon and Ross (2007) reported complaints about swallowing difficulties in individuals with HIV. Their subjects often experienced swallowing difficulties in multiple phases of the swallow. They also explored the relationship between reported swallowing difficulties and CD4 count ranges. Individuals with CD4 counts of 500 and under seemed to report the most complaints. This observation was not significant for every swallowing complaint and a clear association between CD4 and swallowing

difficulties could not be made. The same study found that some individuals with a CD4 count greater than 500 also complained of dysphagia symptoms, which suggests that swallowing difficulties can occur early in the course of HIV infection, especially in the period when individuals are first prone to infections.

Speech-language pathologists' role in the care of HIV/AIDS patients with dysphagia

Speech language pathologists use both objective testing, such as the modified barium swallow study, or more subjective clinical observations at bedside to evaluate swallowing and document the existence and severity of a patient's dysphagia. These clinical tests assess oral motor functioning and attempt to predict the patient's ability to safely swallow various food consistencies. SLPs also make recommendations for food consistency in the diet and can assign therapeutic or rehabilitative exercises to help improve swallowing function and prevent choking or aspiration. In the typical case of an individual with HIV/AIDS, a SLP is only included upon referral from a physician or other healthcare professional and is usually not involved in treatment of swallowing until the later stages of the disease, even though mild dysphagia can occur early in the infection. Therefore, it has been suggested that swallowing function should be evaluated earlier in the course of the infection to prevent or remediate further complications (Bladon & Ross, 2007; (Halvorsen, Moelleken, & Kearney, 2003)).

Speech language pathologists can recommend dietary consistencies for individuals with dysphagia. SLPs may follow the National Dysphagia Diet (NDD) guidelines when recommending diet consistencies for individuals with dysphagia. These diets include consistencies such as pureed, mechanically soft, and advanced

soft food consistency (National Dysphagia Diet Task Force, 2002). Bahl and Hickson (1995) described some of the dietary considerations of individuals with HIV and dysphagia and suggested that modifications in food consistency and texture to a more liquid or semi-liquid consistency were beneficial. These consistencies could be especially beneficial for HIV/AIDS patients who experience dysphagia or odynophagia as a result of oral/esophageal candidiasis or thrush, which are some of the most commonly reported complaints. They also suggested certain feeding strategies (e.g., changing food temperatures, body positioning during eating, etc.) to alleviate pain, reduce swallowing difficulty, and promote better nutrition.

Cognitive changes and dysphagia in individuals with HIV/AIDS

Individuals with disorders of a cerebral etiology have the potential to develop dysphagia if the dysfunction occurs in cortical areas that control motor activity of the oral-pharyngeal-esophageal structures. Also, people with cognitive disorders such as dementia, memory loss, or cognitive decline may also experience difficulty chewing and swallowing (National Institute on Deafness and Other Communication Disorders, National Institutes of Health, 2010). It is important to explore how swallowing difficulty affects this population because when one has dysphagia, they must adhere to strict dietary guidelines and therapeutic recommendations in order to compensate for their swallowing deficits. It would also be important to know if and/or how cognition or cognitive deficits affects a person's ability to adhere to treatment strategies for dysphagia.

Before the development of HAART medication, one study noted that 30 % of AIDS patients developed dementia with some associated motor impairment (Bell,

2004). After the widespread use of HAART, the number of infections that attacked other systems decreased, but pathologies in the central nervous system (CNS), particularly cognitive impairment, seemed to persist. This may be due to the fact that the medications do not effectively cross the blood brain barrier in sufficient quantities to block and suppress HIV replication in the brain, therefore allowing the brain to be a viral reservoir for the HIV virus (Bell, 2004). Other studies have found a frequency of cognitive impairments as high as 15% in patients without HAART medication (McArthur et al., 1993) and as low as 1%, after the widespread usage of HAART medications (McArthur, 2004).

HIV/AIDS can cause several neurological disorders, which can result in cognitive difficulties or dementia. These can include be focal brain diseases such as cerebral toxoplasmosis, primary central nervous system (CNS) lymphoma, progressive multifocal leukoencephalopathy and stroke (Spudich, 2008). Cognitive impairment can also be caused by diffuse brain pathologies such as cytomegalovirus encephalitis, neurosyphilis, and CNS cryptococcal infection (Spudich, 2008). HIV-Dementia or AIDS dementia complex itself, which will be discussed below, is a diffuse brain disease caused by the HIV and no other opportunistic disease in the central nervous system (Spudich, 2008).

Specific neurological conditions affecting cognition have been described in the literature and they are known as HIV-associated neurocognitive disorders (HAND) (Antinori, Arendt, Becker, et al., 2007). HIV-1 associated minor cognitive-motor disorder (MCMD) and HIV-associated dementia (HAD), HIV-Dementia (HIV-D) or

AIDS dementia complex are just some of the classifications under the umbrella of HAND.

To further clarify the disorders of HAND, three classifications are described in Navia et al., (1986), including the most severe, global cognitive impairment, moderate dementia, and the least severe, mild dementia.

To diagnose HIV-associated dementia (HAD), there must be an acquired abnormality in at least two of six neuropsychological domains: attention, information processing speed, abstraction, visuospatial skills, memory, and language. There must also be dysfunction in daily activities. Individuals with clouding of consciousness (i.e. delirium) and evidence of another etiology for the impairments are excluded from this diagnosis (Hinkin et al., 2001).

Some of the early signs and symptoms of AIDS dementia complex are forgetfulness, loss of concentration, increased time to complete mental tasks, loss of balance, deterioration of handwriting, apathy, organic psychosis, and behavioral regression. Late manifestations include moderate-severe dementia, motor weakness, ataxia, tremor, and myoclonus (involuntary twitching of muscles) in addition to the worsening of the earlier symptoms (Navia et al., 1986). To define minor cognitive-motor disorder (MCMD), at least two of these six symptoms must be present: impaired memory, slowed movement, impaired attention or concentration, mental slowing, incoordination, and/or personality change/irritability/lability (Hinkin et al., 2001). There must also be documentable minor functional impairment of activities of daily living (ADLs), and no other known etiology for the symptoms. Also, motor impairment may or may not be present (Hinkin et al., 2001).

There recently has been a shift in the description and classification of HAND. In 2007, researchers reclassified these disorders into three groups rather than two (MCMD and HAD). The current classifications are asymptomatic neurocognitive impairment (ANI), mild neurocognitive disorder (MCD), and HIV- associated dementia (HAD) (Antinori, Arendt, Becker, et al., 2007). These new terms are mostly used in research while the older classification is still used clinically.

Risk factors for developing a HIV-associated neurocognitive disorder are older age, advanced HIV, increased viral load, comorbidities such as anemia, cytomegalovirus, herpes virus, or a history of intravenous drug abuse (IVDA) or delirium, and female gender (McArthur, Brew, & Nath, 2005; Gallego, Barreiro, & Lopez-Ibor, 2011). Women may be more vulnerable to cognitive impairments such as HIV-D, based on performance on procedural learning tasks (Martin, et. al, 2011). From these results, it may be speculated that women could be at risk of poor nutritional outcomes if they suffer from both dysphagia and cognitive impairments.

Although the terms dementia and HAND are often used to describe the etiology of cognitive impairment in this population, there can be several causes of cognitive impairment. For example, substance abuse, neurosyphillis, brain injury, brain tumors, central nervous system infections and adverse effects to medication can all cause cognitive changes (American Academy of Neurology, 1991). Chronic HIV exposure, associated with significant neuronal loss, may induce HIV-associated dementia. This specific progressive cognitive/motor syndrome is observed in approximately 20% of patients with AIDS (Scaravilli, Bazille, & Gray, 2007). An abundance of microphages appears to be a greater factor in HIV dementia than the

presence or extent of brain infection. Late stage HIV infection and an increase in trafficking of precursors of macrophages (monocytes) to the brain, may be associated with development of HIV neurological disease (Gartner, 2000). HIV-D rarely develops before profound immunosuppression (McArthur, Brew, & Nath, 2005), therefore, HIV-D or HAD may be the initial AIDS-defining condition in up to 10% of patients (Navia, 1990). Today, since the advent of HAART, these cognitive conditions are not only seen as an initial AIDS-defining condition and no longer are diseases that signal imminent death (Dougherty et al., 2002).

If an individual is apt to develop dysphagia due to infections of the oral cavity, they may also be apt to developing HIV-associated cognitive disorders in addition to the oral symptoms. Cognitive impairment might affect dysphagia care because patients may experience the symptoms that are described above that have a direct impact on therapeutic swallowing recommendations and techniques. Impaired attention and concentration will interfere with learning and implementing swallowing strategies. Impaired memory will not only impact the learning and understanding of the changes needed in the diet, they hinder the person from remembering that the changes were even implemented at all. This could put them at risk for aspiration or choking. Individuals with dementia (including HIV Dementia) may not fully understand their swallowing difficulty and the cognitive deterioration contributes to eventual disability in independent eating (Stipinovich, 2010).

A study found that a person with dysphagia is at 31% greater risk for aspiration when they are not oriented to person, place, or time (Leder, Suiter, & Warner, 2009). This same study showed that when the patient is unable to follow

single-step verbal commands, they are more at risk for aspiration and being deemed unsafe for oral intake. Compensatory swallowing strategies (e.g., Mendelsohn maneuver, chin tuck/head turn) often involve one-to-two step commands that are at times abstract or complex. A person who is unable to follow or recall these types of sequential commands is less likely to use them and is at greater risk for aspirating. Another study found that individuals with severe cognitive impairment and dysphagia were more successful with oral intake, with significant assistance, while those with mild-moderate cognitive impairments fared worse with oral intake (Steele et al., 1997). This is likely due to the fact that they had less assistance. This study focused on the elderly population, none of which had an HIV-related cognitive impairment. No previous studies were found exploring strictly HIV/AIDS-related cognitive disorders and dysphagia.

Dysphagia and odynophagia in the HIV/AIDS population would seem to be a common occurrence based on the frequency of oral infections; however, there does not seem to be a sufficient evidence of the prevalence of dysphagia/odynophagia and HIV/AIDS in the literature. Also, there is not much information on how many individuals with dysphagia in this population are also affected by cognitive impairment, which will impact their care and swallowing safety.

Purpose and Rationale

After a review of the current literature, there appears to be a need to ascertain the prevalence of swallowing difficulties in individuals with HIV/AIDS. There is also no literature that combines both dysphagia and cognitive impairments in a way that documents the frequency of co-occurrence of these disorders in the HIV/AIDS

population. The current research is not robust regarding relationships between swallowing difficulties and several variables (e.g. age, CD4 count). Also, more data might be useful in describing dysphagia in this population, in addition to current descriptions of complaints in Halvorsen (2003), Bladon and Ross (2007), and Jacobson et al. (2003).

This is a descriptive research study, which explored the prevalence of comorbid HIV/AIDS and dysphagia and co-morbidity of these two conditions and various cognitive impairments. This study, we will also explore the associations (if any) between these disorders and other factors in HIV such as CD4+ count, age, and gender. This study also examines the swallowing outcomes (dietary recommendations) of these groups and the frequency of occurrence of swallowing deficits at various stages of infection (classified by CD4 cell count range).

Research Questions

The following questions will be explored:

- 1 What is the prevalence of dysphagia in a large sample of patients with HIV/AIDS?
 - 1.1 Do referred versus non-referred groups with dysphagia differ in demographics and diagnostic profiles?
- 2 What is the prevalence of dysphagia and cognitive impairments in this sample?
- 3 Is there any association between age and CD4 count and the presence of dysphagia?

- 4 For those patients with dysphagia, is there any relationship between age, CD4 count and the recommended diet consistency?
- Do patients with both dysphagia and a cognitive impairment differ from patients without cognitive deficits in the severity of dysphagia (evidenced by oral motor abilities and penetration/aspiration status) or in recommended diet consistencies at discharge? If so, in what ways?

Methods

Participants

Three hundred (300) randomly selected medical charts at Washington Hospital Center in Washington, D.C. were examined for this study. Information from each medical chart was obtained from the online data storage database Axxyzi (a hospital-wide database that houses the medical records of all patients seen at the Washington Hospital Center complex) and/or through review of physical medicine charts accessed directly through the Health Information Management Department. The first 300 medical entries found in a search query of patients with a diagnosis of HIV/AIDS were included in this study. The patients included were at least 21 years of age to avoid involvement of pediatric cases. All patients included had acquired HIV, rather than having HIV congenitally. These patients were admitted to the acute care or inpatient units at Washington Hospital Center between the months of April 2008 to April 2011. Washington Hospital Center is the largest private hospital serving the Washington, D.C. metropolitan area. Permission to gain access to these charts was granted through the Institutional Review Board (IRB) at the Medstar Research Institute (2011-076) and the University of Maryland (11-0437).

Charts from 159 males and 141 females were included in this review, with ages ranging from 23-81 (mean= 45.92, median= 47). These patients were predominately from the Washington, D.C. metropolitan area (including parts of Maryland and Virginia).

Documentation of HIV Positivity and Immunosuppression Using CD4 Counts

Individuals were included in the study based upon a diagnosis of HIV or AIDS
and staged by CD4 cell count laboratory results obtained from a chart review.

For the purposes of comparison in this study, the patients' actual CD4 counts were used but the CDC categories were also noted. However, due to limitations in full medical history, the data collected may not have been the patient's lowest CD4 count, but the most recent CD4 for that particular hospital admittance. This was collected to help classify the subjects by stage/severity of their infection in preparation for comparison.

Documentation of Age

As stated in the participants section, the ages collected in this sample ranged from 23-81 with a mean of 45.92 and a median of 47. The subjects' ages were recorded from the medical chart. Ages were also grouped using a median split. Using this variable code, the age groupings were ages 23-47 and 48-81.

Documentation of Swallowing Deficits

Patients were identified as having dysphagia through a series of inclusionary factors obtained from chart review which included: medical history, physician and nursing notations, objective instrumental test results, bedside clinical swallowing evaluations, and diet recommendations upon discharge. If referred for assessment of swallowing, swallowing impairment was diagnosed by a certified speech-language pathologist with at least two years of clinical experience post-graduation. Swallowing impairment was documented based on a clinical bedside swallowing evaluation and/or instrumental evaluation of swallowing (i.e., modified barium swallow study,

fiberoptic endoscopic evaluation of swallowing). The presence of penetration or aspiration was documented after reviewing objective videofluoroscopic swallow studies using the Penetration-Aspiration Scale (Rosenbeck et al., 1996). This scale is used by speech pathologists during instrumental evaluations of swallowing (e.g., modified barium swallow studies). It is a scale that ranks the presence of penetration or aspiration ranging from a score 1-8, with 1 indicating that no material from the swallowed bolus enters the airway and 8 indicating that material from the bolus enters the airway and no attempt is made to eject the bolus. The 8 level scale has a numerical rating that differentiates between penetration of a bolus (material rests above the vocal folds) and aspiration of a bolus (material passes through the glottis and enters the airway), and also includes the degree of effort needed to clear the airway in the rating scale.

In the case of individuals with dysphagia or odynophagia caused by infection or inflammation (e.g., candidiasis, herpes simplex/zoster) in this sample, dysphagia was based on physician report of patient complaint found in the charts. Patients were grouped together based upon the presence of dysphagia or odynophagia with an infectious/inflammatory etiology and if they were only seen by their primary physician. If they were referred to the speech pathologist for documentation of dysphagia with other, most likely neurological, etiologies, then they were also grouped together in a separate group..

The presence of swallowing disorders was determined from therapy notes and reports in the medical chart which described signs and symptoms of dysphagia associated with each phase of the swallow. Noting all signs and symptoms of

dysphagia was done to further document and describe the type of dysphagia the patient had and to provide physiological information needed for dietary recommendations.

The most common phase deficits and symptoms in swallowing are defined relative to swallow physiology and /or bolus transport (Groher & Crary, 2010; Washington Hospital Center Hearing and Speech Center swallowing assessment Form 2349). These deficits are based on clinical observations and findings from modified barium swallow studies and other instrumental testing (e.g. fiberoptic endoscopic evaluation of swallowing).

- Oral/Oral preparatory phase- decreased saliva production,
 (xerostomia), decreased taste, decreased tongue or jaw range of motion, difficulty chewing, drooling, decreased labial seal,
 holding, pocketing, piecemeal deglutition, passive flow to valleculae or pyriform sinuses, aspiration prior to the swallow
- Pharyngeal phase- delayed initiation of the swallow, decreased hyoid movement, pooling in valleculae or pyriform sinuses, decreased epiglottal movement, supraglottic penetration, aspiration (during/after a swallow), immediate throat clear or cough after a swallow, delayed or absent throat clear or swallow (as evidenced on videoflouroscopic examination)
- Esophageal phase- esophageal reflux, delayed opening of UES,
 delayed esophageal transport (delayed esophageal peristalsis)

The oral preparatory and oral phase deficits were grouped together, due to the fact that the stages closely overlap and are noted together in the speech language pathologist's evaluation of swallowing found in the charts.

Documentation of Dietary Consistency Recommendations

Levels of dietary outcomes prescribed at time of discharge were rated based on the National Dysphagia Diet guidelines (American Dietetic Association) (National Dysphagia Diet Task Force, 2002). Washington Hospital Center has added 3 ratings (NPO, Level 0, and Regular) that are not on the National Dysphagia Diet (NDD) but were used in their discharge recommendations. These levels were used in the analysis of dietary recommendations:

- **NPO** (none by mouth; includes PEG or NG tube)
- Level 0: Full Liquid (Liquids only; soups, juice, water, carbonated beverages)
- NDD Level 1: Dysphagia-Pureed (homogenous, very cohesive, pudding-like, requiring very little chewing ability).
- NDD Level 2: Dysphagia-Mechanical Altered (cohesive, moist, semisolid foods, requiring some chewing).
- NDD Level 3: Dysphagia-Advanced (soft foods that require more chewing ability).
- **Regular** (all food textures allowed).

Documentation of Cognitive Deficits

Patients with cognitive deficits were identified by a chart diagnosis of altered mental status, HIV- Dementia (HIV-D), AIDS dementia complex, HIV Associated Dementia (HAD), or dementia. Patients were also identified for further review if they

presented with an infection or disease (e.g., neurosyphilis) that could cause cognitive impairment and were included if there was a documented cognitive impairment found upon further review of the medical records. These patients had their diagnosis confirmed by a Mini Mental State Examination (Folstein, Folstein & McHugh, 1975) or by neurologist and/or psychiatrist notes or report of a previous diagnosis of cognitive impairment found in the medical record.

Statistical Methods

The research questions were addressed using frequency counts, descriptive statistics and inferential statistics. IBM SPSS (Version 19) statistical software was used to complete all statistical analysis in this study.

Frequency counts were used to document the frequency of dysphagia and dysphagia with cognitive impairment. It was also used to document the frequency of CD4 cell count ranges and other categorical data. Measures of central tendency (e.g., mean, median, mode) were used to describe characteristics of the continuous variables of age and actual CD4 count.

For continuous variables collected in the study, an independent *t*-test and the nonparametric Mann-Whitney U test were conducted. This statistic helped to determine if there is a relationship between the presence of dysphagia and age and CD4 count.

For analysis using categorical data, a chi square (X^2) was employed. These measures helped determine if there are differences between the dysphagia groups in regards to CD4 count range, age group, and diet consistency recommendation. While categories were expanded to document and describe symptom frequency, severity of

immunosuppression, and diet consistency recommendations, as explained in the previous sections, to meet the requirements of a chi square analysis, the categories were simplified. CD4 counts ranges were simplified to normal CD4 (500 cells/µl³ and above) and abnormal CD4 (499 cells/µl³ and below). Age was categorized as 23-47 and 48-81 using a median split. Diet consistency groupings were simplified to regular consistency and altered consistency.

Subject Groups

Charts were examined and categorized into three overall groups and with subgroups:

- Group 1: HIV/AIDS with Dysphagia and no cognitive impairment- This group included subjects with confirmed dysphagia or complaints of dysphagia and/or odynophagia from oral lesions evidenced in physician notes.
 - Group 1.1 Referred Dysphagia- This group included only individuals seen by the speech language pathologist.
 - Group 1.2 Fungal/Infectious Dysphagia These patients reported complaints of dysphagia and/or odynophagia but were not referred to the SLP for swallowing evaluation.
- Group 2: Dysphagia and Cognitive Impairments group- This group of patients
 presented with dysphagia and/or odynophagia and a reported or confirmed
 cognitive impairment.
- Group 3: No Dysphagia group This group included the remainder of the subjects who did not have reported dysphagia or odynophagia.

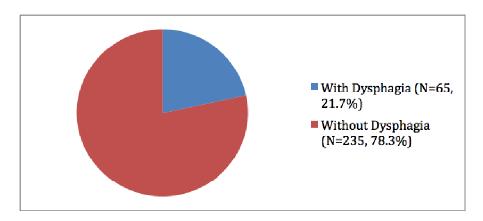
 Cognitive Impairment and No Dysphagia group- Subjects who had no documented dysphagia and odynophagia, but had a documented cognitive impairment were placed into this subgroup to document the frequency of cognitive impairment in the full sample (when combined with the frequency count in group 2).

Results

What is the prevalence of dysphagia in a large sample of patients with HIV/AIDS?

After reviewing 300 medical charts at the Washington Hospital Center, dysphagia or odynophagia was found in 65 charts (21.7%) due to an infectious, inflammatory, structural, or neurological etiology (Figure 1).

FIGURE 1 Prevalence of dysphagia in adults with HIV/AIDS

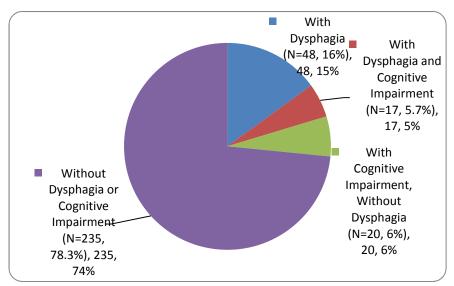


What is the prevalence of dysphagia and cognitive impairments in this sample?

Figure 2 provides a profile of subgroupings. In Group 1, (individuals with dysphagia and/or odynophagia and no cognitive deficits), there were 48 patients (16%), with 9 being referred to an SLP (Group 1.1) and 39 who were not referred (Group 1.2). Group 2 (5.7%, N=17) included patients with dysphagia and/or odynophagia and a co-morbid reported or documented cognitive deficit. The remainder of the sample in Group 3 included individuals without dysphagia (n=235, 78.3%). This subset was also divided into individuals with and without a reported cognitive impairment. Twenty subjects in Group 3 (6%) had a cognitive impairment (including diagnoses of altered mental status to dementia) and no documented dysphagia. When combined with the individuals in Group 2 who also had a

documented cognitive impairment, there was a total frequency of cognitive impairment (regardless of dysphagia status) of 12.3%.

FIGURE 2 Prevalence of Disorders by Group



Total prevalence of cognitive impairment in this sample was 12.3% (N=37).

Gender

The distribution of males and females in this sample was relatively equal, with 53% male subjects and 47% females. In Groups 1 and 3, the male/female ratio was almost equal. In Group 2, males were more frequent.

Is there any association between age and CD4 count and the presence of dysphagia?

The charts reviewed for this study included adults ages 23 to 81 with HIV/AIDS, Age was normally distributed across the sample. The average age of individuals in the sample was 45 years (Table 1). Dysphagia coupled with cognitive deficits (Group 2) affects more older patients (m=49.53) (Table 1). However, this Group 2 had an age range from 24-75 years (Table 1). This observation suggests that younger patients may also be at risk for these co-morbid conditions. The presence of

cognitive impairment could be due to a combination of normal age effects being magnified by the susceptibility to opportunistic diseases and infections that comes with HIV/AIDS infection. A t-test for independent samples was used to compare age across groups. The mean age of the combined dysphagia groups (Groups 1 and 2) was m = 44.17, sd = 10.153. The mean age of the group without dysphagia (Group 3) was m = 46.40, sd = 11.227. This difference was not statistically significant (p = 1.449) at the .05 level (t = -1.449, df = 298).

The CD4 counts of the individual patients were not evenly distributed across the sample and were negatively skewed. The group with the lowest average CD4 count was Group 1.2 (m=50.24), the individuals with oral lesions of an infectious etiology (Table 1).

Of the three CD4 categories as defined by the CDC, the charts reviewed here fell mostly in category 3 (<200) across all groups. The majority of the patients included in this study had advanced HIV infection and/or AIDS as evidenced by a median CD4 count of 104 (Table 1) and the fact that 189 out of the 284 reported CD4 counts were in Category 3. Several of the patients in groups 1, 2 and 3 had a record of an AIDS-defining illness such as progressive multifocal leukoencephalopathy, disseminated *Mycobacterium avium* complex, and cytomegalovirus disease. A t-test showed that the mean of the combined dysphagia group was m = 73.31, sd = 111.233 and that the mean from the group without dysphagia (Group 3) was m = 232.25, sd = 286.137. Because the data was not normally distributed, a nonparametric Mann-Whitney U test was used to examine the data. The difference between the two means was significant (p = .000) at the .05 level.

For those patients with dysphagia, is there any association between age, CD4 count and the recommended diet consistency?

One of the research questions of this study asked if age, CD4 count range or diet consistency recommendation could be associated with dysphagia alone or dysphagia with cognitive impairments. A chi square (X^2) was used and the variables were simplified. The dysphagia groups (Group 1 and 2) were compared using age as the variable. Age was split into two groups using a median split, creating ranges from 23-47 and 48-81. More individuals in the dysphagia only group (Group 1) were 23-47 years old, while there were more individuals in the dysphagia/cognitive impairments group (Group 2) in the 48-81 age range. The relationship between dysphagia group and age was significant (p = .02) at the .05 level ($x^2 = 5.008$).

CD4 counts, simplified to normal CD4 count (500 cells/ μ l³ and above) and abnormal CD4 count (499 cells/ μ l³ and below) were also examined. All individuals in Group 2 and all but one individual in Group 1 had an abnormal CD4 count. Nevertheless, the relationship between the groups and CD4 count was not statistically significant (p = .56) at the .05 level ($x^2 = 339$).

Regular consistency (normal) diets were most frequently recommended upon discharge (88%, n=264) (Table 1). Only in Group 2 did more restrictive diet consistencies (58%, n=10) occur more frequently than the regular diet consistency (29%, n=5), which could be due to the compounding factors of the severity of the cognitive impairment, motivation to maintain nutrition, and the ability of the patient to swallow safely and independently. Diet consistency recommendations were compared across groups 1 and 2. For this analysis, the diet consistency groupings

were simplified to two categories, regular consistency and altered consistency. Group 2 showed more likelihood to be recommended an altered diet consistency than Group 1. The relationship is statistically significant (p = .001) at the .05 level ($x^2 = 11.702$).

TABLE 1 Demographic Data, CD4 Counts and Dietary Consistency Recommendations in Adults with HIV/AIDS.

			Patients with HIV/AIDS (n= 300)		
				Dysphagia/	
	All	Referred	Infectious	Cognitive	No
	Patients	Dysphagia	Dysphagia	Impairment	Dysphagia
Groups	(n=300)	(n=9)	(n=39)	(n=17)	(n=235)
Demographic Data					
	45.92 (23-	43.33 (28-	42.03 (27-	49.53 (24-	46.40 (23-
Age, mean (range)	81)	54)	59)	75)	81)
Median y	47	47	41	50	47
Female gender % (n)	47 (141)	22 (2)	53 (21)	29 (5)	48 (113)
Male gender % (n)	53 (159)	77 (7)	46 (18)	70 (12)	51 (122)
CD4 Ct*,	199.12	194.25 (5-	50.24 (0-	65.14 (0-	232.25
mean(range)	(0-2104)	450)	347)	223)	(0-2104)
Median	104	115	33.5	33.5	161.5
Diet Consistencies %					
(n)					
NPO	2.3 (7)	22 (2)	0 (0)	23 (4)	0.4(1)
Puree	3.3 (10)	11 (1)	5(2)	29 (5)	0.8(2)
Mechanically Altered	1.3 (4)	22 (2)	0(0)	5(1)	0(0)
Advanced	0.7(2)	11 (1)	0(0)	0(0)	0.4(1)
Regular	88 (264)	33 (3)	94 (35)	29 (5)	94 (222)
Unknown#	4.3 (13)	0 (0)	5(2)	11 (2)	3 (9)

Note: *Some patients' CD4 Counts were unknown/undetectable and therefore not counted. #Some patients' diet consistencies were unknown and presented here as "Unknown".

TABLE 2 Summary of Findings for Age, CD4 Count, and Recommended Diet Consistency Across Groups 1, 2, & 3

Variable	Groups	Test (p)
CD4 Count	Combined Dysphagia	
	(Group 1 &2)	U (.000)*
	(m=44.17)	
	Vs.	
	Without Dysphagia	
	(Group 3) (m=46.40)	
Age	Combined Dysphagia	
	(Group 1 &2)	t=1.449 (1.449)
	(m=73.31)	
	Vs.	
	Without Dysphagia	
CD4 C	(Group 3) (m=232.25)	
CD4 Count	Dysphagia Only	-2 220 (56)
	(Group 1.1 and 1.2) Vs.	X = 339 (.30)
	Dysphagia/Cognitive (Group 2)	
	(Group 2)	
Age	Dysphagia Only	
1190	(Group 1.1 and 1.2)	$x^2=5.008 (.02)*$
	Vs.	11 21000 (102)
	Dysphagia/Cognitive	
	(Group 2)	
	` '	
Diet Consistency	Dysphagia Only	
	(Group 1.1 and 1.2)	$x^2=11.702 (.001)*$
	Vs.	
	Dysphagia/Cognitive	
	(Group 2)	

^{*}Finding is significant at the .05 level (p < .05)

Do patients with both dysphagia and a cognitive impairment differ from patients without cognitive deficits in the severity of dysphagia (evidenced by oral motor abilities and penetration/aspiration status) or in recommended diet consistencies at discharge?

Group 1: Dysphagia (Referred and Infectious Etiologies)

All individuals with dysphagia and odynophagia and no cognitive impairment were included in Group 1.

Group 1.1:

For those who were categorized as having dysphagia and were seen by an SLP (Group 1.1, Table 2), most complained that they "had not been able to eat or swallow liquids" and that "food is getting stuck in the back of the throat".

Diets in Group 1.1 varied from the most restricted (NPO) to the least (Regular). Three of the patients in Group 1.1 were first assigned a modified diet, but with time and before discharge, were recommended for a regular diet. Three patients who required a tracheostomy tube for breathing were placed on the restricted diets (e.g., NPO and liquid diets). Two out of three patients were advanced to less restrictive diets once the tube was removed and they were de-cannulated, or if their overall medical status improved.

Chart reviews of bedside clinical examinations and reports of instrumental testing of individuals in Group 1.1 revealed that oral deficits included labial weakness and deviation, decreases in oral sensation and function (often asymmetrically), and secretion management issues (drooling). Passive bolus flow to the valleculae and pyriform sinuses and piecemeal deglutition were also observed during swallow

studies using various bolus consistencies. Pharyngeal observations from the Modified Barium Swallow studies included impaired coordination of pharyngeal structures, delayed swallow, residuals and pooling in the pyriform sinuses and valleculae, decreased hyoid movement, decreased laryngeal elevation, supraglottic penetration, silent aspiration, and decreased/weak abduction of the true vocal folds. Three patients in Group 1.1 had esophageal swallowing issues (e.g. strictures, esophagitis) (Table 2).

According to subjective bedside swallowing evaluations, two patients in Group 1.1 showed subtle signs and symptoms of aspiration, such as an intermittent cough or throat clearing immediately after swallowing. When using objective instrumental measures such as the FEES or MBS, three patients displayed frank aspiration with and without protective reflexes. Only two patients in Group 1.1 had neurological changes (e.g., CVA) that may have contributed to their dysphagia but did not reportedly cause cognitive changes. Another patient with Guillian-Barre syndrome, which causes nerve inflammation resulting in muscular fatigue, had variable dysphagic symptoms. Dysphagia was a result of prolonged intubation or tracheostomy in three patients in Group 1.1. One patient had epiglotitis and swelling of the neck structures, which caused odynophagia and difficulty swallowing.

TABLE 3 Group 1.1 (Referred Dysphagia) Symptoms of Dysphagia by CD4 Count Range

		Category 1	Category 2	Category 3	Unknown
	All	$(\geq 500 \text{ cells}/$	(200-499	(< 200 cells/	CD4
	(n=8)	μl^3)	cells/µl ³)	μl^3)	Count
Referred Dysphagia	` /	(n=0)	(n=2)	(n=4)	(n=2)
Aspiration/penetration	3	0	1	1	1
Neurological Changes	2	0	1	1	0
Oral Impairment	3	0	0	2	1
Pharyngeal Impairment	5	0	2	1	2
Esophageal Impairment	3	0	0	2	1

Group 1.2:

Many of the subjects in this group (Group 1.2) complained of painful swallowing (odynophagia) as well as difficulty swallowing, but were not referred to an SLP. Thirty-five patients with infectious etiologies and dysphagia/odynophagia (Group 1.2) were placed on a regular consistency diet, despite pain on swallowing. One patient was recommended to be placed on a diet of thickened liquids in addition to regular consistency meals. Because none of the patients in Group 1.2 were seen by an SLP, it cannot be confirmed that a specific swallowing phase was affected. For example, if a patient had esophagitis, it may be assumed that the esophageal phase of the swallow would be impaired and that they would have discomfort during ingestion. If the person had oral thrush, then it may also be assumed that the oral phase of the swallow would be affected. Sixteen subjects in Group 1.2 had esophageal lesions or diseases and 29 had oral lesions or complaints. Neither penetration nor aspiration was reported in individuals in Group 1.2. The causes of dysphagia/odynophagia in Group 1.2 can be found in Table 3, which displays the etiologies of fungal diseases.

TABLE 4 Infectious Etiologies of Dysphagia for Group 1.2 (Fungal/Infectious Dysphagia) grouped by CD4 Count Range

	Category 1	Category 2	Category 3	
	$(\geq 500 \text{ cells}/$	(200-499	(< 200 cells/	Unknown
Dysphagia (Fungal/	μl^3)	cells/µl³)	μl^3)	CD4 Count
Infectious etiology)	(n=0)	(n=2)	(n=35)	(n= 2)
Neurological Changes	0	0	5	0
Oral Thrush	0	1	16	1
Candida Esophagitis	0	1	8	1
Oral Candidiasis	0	0	8	0
CMV Esophagitis	0	0	1	0
Oral/Gum/Palatal Ulcer	0	0	3	0
Esophageal Inflammation	0	0	1	0
Herpes Simplex Esophagitis	0	0	1	0
Esophagitis	0	0	1	0
Esophagitis/thrush	0	0	2	0

Group 2: Dysphagia and Cognitive Impairment

With only 17 patients, this group was the smallest group. This group had the most detailed reports, as they were most often referred to a speech-language pathologist. Because of cognitive involvement, the patients in this group often could not or did not make first-hand complaints about their swallowing difficulties. Swallowing difficulties often caused poor oral intake, decreased weight and malnutrition. Four of these patients progressed from a very restricted diet to a less-restrictive diet, but only five were recommended to be placed on a regular diet upon discharge.

This group, as a whole, experienced swallowing impairment at all phases. Eight out of 17 patients (47%) with documented swallowing impairments experienced deficits at multiple swallowing phases. One subject had deficits at all three phases, while seven had two phases affected (Table 4). Four patients had impaired oral motor

abilities, which could be associated with the frequent occurrence of cerebral infarct. Five patients in this group had facial paresis characterized by decreased labial seal, decreased bolus containment and poor management of secretions (drooling). Other oral phase deficits included delayed swallow, pooling in the mouth, impaired bolus formation, delayed bolus manipulation, delayed oral transfer and clearing, passive flow to valleculae and pyriform, and piecemeal swallow across various consistencies. In the pharyngeal phase, impairments observed included overall incoordination of the swallow, decreased laryngeal elevation, pharyngeal residuals, and aspiration and penetration. As far as esophageal involvement, two patients had infections in the mucosal membranes covering the tissue (e.g., esophageal candidiasis) causing odynophagia. Nine of the patients had oral or esophageal infections that contributed to their dysphagia/odynophagia, including oral and esophageal candidiasis, oral thrush, and cytomegalovirus (CMV) esophagitis.

Patients in this group displayed varying frequencies of penetration and aspiration on a specific rating scale, the Penetration-Aspiration Scale (Rosenbeck et al., 1996). One individual in this group displayed silent aspiration, evidenced by a score of 8 out of 8 (material enters the airway, passes below the vocal folds, and no effort is made to eject) on the Penetration-Aspiration Scale (Rosenbeck et al., 1996). Scores of 2 out of 8 (material enters the airway, remains above the level of the vocal folds, and is ejected from the airway) and 4 out of 8 (material enters the airway, contacts the vocal folds, and is ejected from the airway) were also assigned to patients based on the observations after modified barium swallow studies. Three patients had no visible aspiration, but they had factors that put them at risk for aspiration, such as a

CVA, cognitive impairment, or impaired oral mobility. Other signs and symptoms of risk of aspiration that were noted were immediate cough/ throat clear after thin liquid and puree trials, cough/throat clear delayed after thin liquid trial and ice chips, weak cough or throat clear, and watery eyes.

Aspiration was documented in 7 out of 17 patients in this group. Eight of the patients in Group 2 had acute stroke in their medical history. Five patients also had accompanying aphasia. The diagnoses of dementia came from neurology or other physician notes/observations or a past medical history of HIV related dementia. Many of those with mild cognitive problems such as altered mental status also had accompanying diagnoses of fever, Progressive multifocal leukoencephalopathy (PML) and HIV encephalopathy, among other diagnoses. Neurological events that occurred in these patients included seizures, head injury and stroke (CVA) resulting in aphasia, right-side neglect, and facial and bodily paresis. Based on observations made from the data, it appears that individuals in Group 2 had the highest frequency of aspiration (7/17) and were put on a more restrictive diet (10/17).

TABLE 5 Dysphagia Profile of Group 2 (Dysphagia and Cognitive Impairment)

		Category 1	Category 2	Category 3	Unknown
	All	$(\geq 500 \text{ cells}/$	(200-499	(< 200 cells/	CD4
	n= 17	μl^3)	cells/µl³)	μl^3)	Count
Dysphagia/Cognitive		(n=0)	(n=1)	(n=14)	(n=2)
Aspiration/penetration	7	0	0	6	1
Neurological Changes	9	0	0	7	2
Oral Impairment	12*	0	1	9	2
Pharyngeal Impairment	9*	0	0	9	0
Esophageal Impairment	3*	0	0	3	0

^{* =} Patients have multiple phases affected

Discussion

After a review of 300 medical charts, dysphagia was noted in 21% of patients. This prevalence rate is consistent with the numbers reported by other studies that noted or examined dysphagia directly (Chelbowski, et al., 1989; Bladon and Ross, 2007; Tirwomwe et al, 2007). However, the frequency rate of 21% was slightly higher than what was reported in some other studies (Jacobson et al., 2003). It appears that dysphagia is a common symptom of HIV/AIDS and that it can occur relatively frequently.

In our sample, we found that only 5% of patients were coded for dysphagia and a cognitive condition. No other studies could be found that explored the frequency of both dysphagia and cognitive impairment together. The cognitive conditions in this sample set included altered mental status and mild to severe HIV/AIDS-related dementia and could certainly complicate treatment.

Dysphagia can occur with any CD4 count range from low to high (Bladon and Ross, 2007; Barr, 1992; Sanjar, Queiroz, & Miziara, 2011; Vazquez, 2000). Our study revealed that dysphagia occurred primarily in the most severe CD4 count class (>200 cells/ cubic microliter) (63%, n=189 in CD4 category 3). This may be attributed to a number of factors. Because the patients with a high CD4 count are the most immunocompromised, they are at greatest risk of succumbing to opportunistic infections and may develop dysphagia from these diseases and infections. Also, it is possible that this finding could be a result of sampling error. Because this study was done with severely involved acute care patients who have to be hospitalized because they are sick or at their sickest, it is to be expected that their CD4 count would be in

the severe range. However patients with very low or very high CD4 counts were found in each group.

We found that there was an association between age and the presence of dysphagia in individuals with HIV/AIDS, but there was a relationship between CD4 count and the presence of dysphagia. Because the mean of the dysphagia group was lower, it could imply that individuals with lower CD4 counts are more likely do develop dysphagia or an opportunistic disease in which dysphagia is a resulting symptom. We found no significant relationships between a diagnosis of dysphagia with and without cognitive impairment and CD4 count, but there is a relationship between these groupings and age and diet consistency recommendations. This finding helps confirm the observation that older individuals with HIV/AIDS are more likely to have the co-morbidities of dysphagia and cognitive impairment than younger individuals. These findings also help confirm the observation that individuals with cognitive impairment and dysphagia are more likely to be put on a restrictive diet than individuals with dysphagia only.

An interesting observation found in Group 1 is that three out of nine patients were put on a regular diet when dysphagia was not treated with medicine (as in the case of fungal dysphagia) and despite signs and symptoms of dysphagia. This could have a negative impact on the client's health. No follow-up could be found on these patients, so we are unsure of their success on these diets. If a person with dysphagia is put on a regular consistency diet, they may be more at risk for aspiration due to the varied textures and consistencies of the bolus. This observation suggests a need for the medical team and the SLP to devise better guidelines about HIV/AIDS care and

the speech pathologist's role in treatment. It is suggested that follow-up should be initiated to see how successful discharged patients are with these diets.

Aspiration was documented most frequently in Group 2. It is unclear whether it occurs more in this group because there is a neurological component that could cause a more severe dysphagia, or if it is just documented more frequently because individuals with a neurological event (e.g. stroke, TBI) are automatically seen by the SLP in this particular hospital.

Limitations:

There were obvious limitations to this study. First, this was a retrospective analysis of actual medical charts that were used in patient care and not annotated for the purposes of research. Therefore, there was no control over data in charts or in the examinations that were reported. Some charts were more detailed than others. In some cases, only the discharge report, physician's notes, and lab results were on file and in other cases, the complete file could be retrieved. Another limitation for the dysphagia subject data (Groups 1 and 2) is that not all were seen by the SLP; the diets may have been recommended by a doctor or nutritionist who did not consider the therapeutic implications of a dietary plan in the way that an SLP would. Many patients with infectious oral lesions who could have been referred to a SLP were not referred and were not given formal dysphagia assessments. Also, there was no way of knowing what occurred after discharge pertaining to success with recommended diet consistencies.

Clinical Implications:

This study provides more evidence that dysphagia and odynophagia are substantial complaints in the HIV/AIDS population and that more steps should be taken to educate professionals who work with HIV patients, (including physicians, nurses, and SLPs), in the diagnosis and management of swallowing disorders. This study also suggests that cognitive impairments can co-occur with dysphagia across the age range and that this small subset of the HIV/AIDS population may experience significant deficits which require more strict dietary guidelines and professional assistance in order to maintain an adequate quality of life. Better management and follow-up might lead to less need for acute care or re-hospitalization and could reduce the risk of aspiration pneumonia.

Future Research Needs:

The limitations of the current study are common in retrospective research. Prospective study could eliminate these limitations. A prospective study based upon this research would allow further exploration into how cognitive impairments affect dysphagia management and how the co-treatment of these disorders impacts the health of the individual. This study should have documentation of baseline severities of both dysphagia and cognitive impairments in order to have better control over subject selection and extemporaneous variables. Follow-up is needed with patients having HIV/AIDS to see how well they are adhering to recommended diet consistencies or prescribed oral motor exercises and compensatory strategies. Future research could also explore the need for more objective studies of dysphagia/odynophagia in this population. Prior research has shown risk of

aspiration, especially in the face of infectious/structural complications of HIV (e.g. (Halvorsen, Moelleken, & Kearney, 2003). In this study many of the patients were not seen by the SLP unless they were consulted following a stroke or other neurological events. However, evaluation of swallowing when the etiology is an oral lesion might be beneficial to the patient who may be silently aspirating because the oral infection has reduced their sensitivity in the pharynx. This may indicate a need for consistent referrals of these patients to a speech pathologist for further investigation of the patient's swallowing abilities. From more consistent referrals, SLPs can begin to develop standard set of measures of the risk of aspiration and penetration and on treatment for this population.

Future research should also focus on clarifying the most successful diets for this population and proper follow-up protocols after discharge. In conjunction with nutritionists, the SLP could focus on the safest diet consistencies while maintaining optimal nutrition, since nutrition is especially vital to wellness in this population. Future research may also help guide speech language pathologists and other professionals who help treat HIV and dysphagia to better understand the effects of diet consistencies on these patients, whether they have mild dysphagia from an oral lesion or severe dysphagia from a stroke.

Additional research should include patients who are not at their sickest point and require hospitalization. Rather, data are needed from outpatients in community treatment facilities. Examining patients who come in for regular check-ups to see if they have dysphagia/odynophagia, (and/or cognitive impairment) and documented CD4 count may help confirm or deny whether there is a relationship between CD4

and dysphagia. A wider population sample, including those in sub-acute care, rehabilitation, or those who are only under the care of the primary physician, could document how well compensatory measures are used in the treatment of dysphagia/odynophagia and cognitive impairment outside of the hospital setting. This research should also eliminate some of the limitations of the current study by controlling for variables and matching treatment methods to presenting symptoms.

Dysphagia and odynophagia are dangerous and uncomfortable disorders that are present in over 20% of the HIV/AIDS population, causing numerous problems including aspiration pneumonia and malnutrition. Dysphagia negatively affects a patient's quality of life and nutrition. If this disorder is treated properly, these patients may have a better chance to thrive by eliminating the chances of becoming malnourished or developing pneumonia. When coupled with cognitive deficits, which affected 5% of this population, a patient's ability to manage dysphagia or odynophagia may be more difficult. While the number of individuals with these concurrent diseases are small, this population will require special care and collaboration between medical professionals, which will only occur with further knowledge, and understanding of how these diseases impact the health and daily life of those affected.

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