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IMPLEMENTATION OF THE 2012 AMERICAN COLLEGE OF RHEUMATOLOGY GUIDELINES FOR MANAGEMENT OF GOUT IN A SAMPLE OF MIDWESTERN NATIVE AMERICANS

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IMPLEMENTATION OF THE 2012 AMERICAN COLLEGE OF RHEUMATOLOGY GUIDELINES FOR MANAGEMENT OF GOUT IN A SAMPLE OF MIDWESTERN NATIVE AMERICANS

BY

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GUIDELINES FOR MANAGEMENT OF GOUT

IN A SAMPLE OF MIDWESTERN NATIVE AMERICANS

This DNP Scholarly Project by Myrth C. Condon is recommended for approval by the student's Faculty Chair, Committee and Department Head in the School of Nursing

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ABSTRACT

IMPLEMENTATION OF THE 2012 AMERICAN COLLEGE OF RHEUMATOLOGY GUIDELINES FOR MANAGEMENT OF GOUT IN A SAMPLE OF MIDWESTERN NATIVE AMERICANS

By

Myrth C. Condon

Gout is a well understood, yet poorly treated condition that is associated with many co-morbidities including hypertension, renal and cardiac disease, metabolic syndrome, central obesity and type-2 diabetes. The American Indian population is at risk for gout due an increased incidence of type-2 diabetes, chronic kidney disease, hypertension, and alcohol abuse. The purpose of this scholarly project was to implement the 2012 American College of Rheumatology (ACR) guidelines in a sample of Tribal members within mid-western portions of the United States. This scholarly project utilized a quasi-experimental research design with pretest – posttest methods to measure uric acid levels at baseline and at six months after implementation of the ACR guidelines. The Cox (2003) interaction model of client health behavior was used as a theoretical framework to guide the scholarly project. A paired *t*-test was used to compare mean uric acid levels (6.41 mg/dL) prior to the intervention with uric acid levels (6.36 mg/dL) after the intervention. Results from the statistical analysis did not yield statically significant results ($p = 0.52$). The findings of the study support the conclusion that gout is poorly managed by healthcare providers and additional support and resources are needed to improve patient care outcomes.

DEDICATION

This scholarly project is dedicated to my loving, supportive husband and to my adoptive Tribe and my patients who suffer from gout.

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Chapter One

Gout is one of the most common inflammatory conditions that exists in the United States (Hilaire & Wozniak, 2010). Gout is caused by monosodium urate crystals that form in the joints and other tissues of the body (Krishnan et al., 2013). The serum level of uric acid must be elevated for these crystals to form (Krishnan et al., 2013). Elevation of serum uric acid levels greater than 6.8 mg/dL defines the condition of hyperuricemia and can lead to gout (Hilaire & Wozniak, 2010).

The prevalence of both gout and hyperuricemia has increased over the past 20 years, likely due to increased obesity rates and hypertension (Zhu, Pandya, & Choi, 2011). Current gout prevalence in the United States is 3.9%, which correlates to approximately 8 million people within the general population (Centers for Disease Control and Prevention, 2015). There are no prevalence rates available in the literature of gout in the American Indian population. However, according to the 2010 census data, there are 5.2 million people in the United States that self-identified as American Indian or Alaska Native so one can deduce approximate prevalence rates, using the 3.9% figure, for this population to be roughly 0.2 million affected by gout (Norris, Vines, & Hoeffel, 2012).

Of the inflammatory conditions, gout is the most well understood; yet it remains one of the most poorly treated conditions (Doherty et al., 2012). Gout flares tend to be the focus of treatment with little attention given to preventative therapy, consisting of lowering uric acid levels (Doherty et al., 2012). Furthermore, gout has historically been

considered a self-induced disease caused by excessive intake of alcohol and rich foods containing purines (Zhang et al., 2006; Zhang, Chen, et al., 2012). Purine rich foods include meats, organ meats, gravies, seafood, beans, peas, lentils, oatmeal, spinach, asparagus, and mushrooms (Zhang, Chen, et al., 2012). More recently, studies have shown that gout can also be triggered by excessive intake of high fructose beverages (Choi & Curhan, 2008; Choi, Willett, & Curhan, 2010). Both gout and hyperuricemia have been linked to other comorbid diseases such as hypertension, renal disease, cardiac disease, metabolic syndrome, central obesity, and diabetes (Dehghan, van Hoek, Sijbrands, Hofman, & Witteman, 2008; Hilaire & Wozniak, 2010; Sheikhbahaei, Fotouhi, Hafezi-Nejad, Nakhjavani, & Esteghamati 2014).

Background and Significance

Type 2 diabetes mellitus (T2DM) is one of many co-morbid conditions associated with gout and high uric acid levels. Recent studies have shown a positive association between elevated uric acid levels and the development of impaired fasting glucose which may lead to T2DM (Kodama et al., 2009; Lv et al., 2013; Meisinger et al., 2012; Miyake et al., 2014). Chronic kidney disease (CKD) is another comorbid condition that is associated with both hyperuricemia, gout, and diabetes. Researchers recently found that elevated serum uric acid led to renal damage both at the cellular level and in the tissues of the kidneys (Viazzi, Leoncini, Ratto & Pontremoli, 2014). Other studies demonstrated an association between elevated uric acid levels as an independent risk factor and the worsening of CKD and subsequent progression to dialysis (Nacak, van Diepen, de Goeij, Rotmans, & Dekker, 2014; and Sheikhbahaei et al., 2014).

Management of Gout

Over the years, gout has been poorly treated by health care providers despite current understandings about the pathophysiology of gout and hyperuricemia (Simkin, 2008). Improved patient education and communication about the causes of gout and treatment options are essential to improving management of this long term disease (Zychowicz, Pope, & Graser, 2010). In 2012, the American College of Rheumatology published new guidelines for the management of gout (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). These guidelines include recommendations for patient education about diet, lifestyle modifications and medication use for the management of gout and hyperuricemia (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012).

Statement of the Problem

The American Indian population is afflicted by T2DM and associated comorbidities at a much higher rate than that of the general population. American Indians have a 2.3 times higher occurrence of T2DM than White, non-Hispanics in the United States (Indian Health Service, 2012). Approximately one in eight (13.2%) American Indians aged 18 or older has diabetes; and in the 18 to 35 age group, diabetes incidence has increased by 46% compared to increases of only 14% in the general population (Acton et al., 2002; American Kidney Fund, 2017. Incidence of kidney disease and kidney failure secondary to diabetes in American Indians is 1.9 times higher than that of the general population (Indian Health Service, 2012). A greater incidence of T2DM and CKD results in higher death rates for American Indians compared to the White individuals (Espey et al., 2014).

Alcoholism remains a significant problem for American Indians (Beauvais, 1998). In 2009, American Indian adults experienced increased rates of binge drinking in comparison to prior years and an increased number of drinks per episode of binge drinking (Centers for Disease Control and Prevention, 2011). American Indians also have an elevated incidence of alcohol related accidents causing injury in comparison to the White population (Green, McNulty-Eitle, & Eitle, 2014).

Since the American Indian population is so adversely affected by T2DM, CKD, and alcoholism and given that these factors are so closely associated with gout; it is therefore likely that American Indians are also afflicted by gout. However, there is a paucity of literature devoted to this topic.

Statement of Purpose

The purpose of this scholarly project was to implement the 2012 American College of Rheumatology (ACR) guidelines in a sample of Tribal members within a mid-western portion of the United States (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). Inclusion criteria consisted of Tribal members, aged 18 years and older, who receive care at a mid-western Tribal health center and have a diagnosis of gout. A pretest - posttest, quasi-experimental design was utilized; consisting of baseline assessments of uric acid levels and other measures obtained through chart reviews which was followed by implementation of the ACR guidelines. Then, six months following implementation of the guidelines, uric acid levels were obtained and compared to baseline measures. In addition, a patient education handout was distributed that provided information about causes of gout, dietary triggers, and lifestyle changes that can be used to lower uric acid levels. The educational handout also included information for patients

about medications that are used for treatment of gout flares and chronic hyperuricemia. During the sixth month implementation phase, patients were offered the educational handout and health care professionals provided education about the importance of lifestyle modifications and medication use. Implementing the ACR guidelines may assist health care providers to better manage the treatment of acute gout flares and to lower uric acid levels long term; thereby reducing the incidence and severity of gout flares for Tribal member patients.

Theoretical Framework

For this scholarly project, the interaction model of client health behavior (IMCHB) was implemented. The IMCHB is considered to be a middle-range nursing theory and is useful in primary care (McEwen, 2014). The IMCHB is comprised of three main elements: client singularity, the client-professional interaction, and health outcomes (Cox, 2003). Client singularity includes background and dynamic variables. Background variables consist of demographic characteristics, social influence, previous healthcare experience, and environmental resources, while the dynamic variables include cognitive appraisal, affective response, and motivation. The client-professional interaction incorporates concepts of affective support, providing health information, decisional control, and professional or technical competencies. Finally, health outcomes include concepts of healthcare utilization, health status indicators, problem-severity indicators, adherence to recommended care regimen, and satisfaction with care (Cox, 2003).

The IMCHB posits that patient health outcomes are influenced in response to health care provider interventions and from interactions between health care providers and individual clients (Cox, 2003). In addition, each client is influenced by his/her

background and dynamic variables which can affect the client-professional interaction. Reciprocity exists between the client's three dynamic variables and the four concepts of client-professional element. So, in other words, providers can play a role in helping patients effectively manage disease states and optimize their health outcomes. According to Cox (2003), the IMCHB can be used in research by examining the client singularity variables to help explain health behaviors and their consequential outcomes and to help formulate interventions that target these variables.

Chapter Two

Literature Review

Gout occurs as a result of urate under-excretion, overproduction, or both (Dincer, Dincer, & Levinson, 2002). Urate under-excretion is the most common cause of gout in approximately 90% of cases (Choi, Mount, & Reginato, 2005). Individuals with gout tend to excrete 41% less uric acid than those without gout due to deficiency in renal function (Dincer et al., 2002). Uricase is an enzyme that converts uric acid to allantoin, a more soluble substance that is readily excreted in urine (Zychowicz et al., 2010). Genetic loss of this enzyme causes under-excretion (Zychowicz et al., 2010). Other causes of under-excretion include excess alcohol intake, decreased glomerular filtration rate in the kidney, use of certain medications like diuretics, low-dose aspirin, and niacin (Zychowicz et al., 2010). Over production of uric acid is related to dietary factors, such as excessive intake of purine rich foods, particularly of animal origin, and genetic factors, such as increased cell turn-over which occurs with psoriasis and certain hematologic cancers (Choi et al., 2005).

The progression of gout is often categorized into four stages: (1) asymptomatic hyperuricemia, (2) acute gouty arthritis, (3) inter-critical periods, and (4) advanced gout (Zychowicz et al., 2010). In the first stage, asymptomatic hyperuricemia, uric acid levels are elevated and monosodium urate (MSU) crystals begin to form and deposit into the tissues and joints (Zychowicz et al., 2010). Patients at this point are symptom free (Dalbeth & Stamp, 2014). It has been demonstrated that only about 20% of people with hyperuricemia will go on to develop symptomatic gout (Dalbeth & Stamp, 2014). By the time there are sufficient numbers of MSU crystals in the tissues, an inflammatory

response is triggered, causing a “classic” gout attack. Second stage, acute gouty arthritis, typically involves a single joint and the first metatarsophalangeal joint is the most commonly affected (Zychowicz et al., 2010). As the disease progresses, acute gout attacks can occur more frequently and involve multiple joints. Tophi, which consist of MSU crystals in the soft tissues, can form in the acute gout stage as well. In the third stage, inter-critical periods, MSU crystals continue to form and deposit and more than 50% of people will have noticeable joint changes evident on ultrasound exams. During this stage, acute gout attacks continue, becoming more severe, lasting longer, and may include multiple joint involvement. If third stage gout is left untreated, fourth stage advanced gout will lead to chronic disfiguring arthritis with joint damage, erosions, and visible tophaceous nodules (Zychowicz et al., 2010).

In contrast to Zychowicz et al. (2010) commonly held assumption that gout occurs as a series of symptomatic, recurrent flares, Dalbeth and Stamp (2014) argue that important pathological changes may be missed if clinical diagnosis is only based upon symptomatology. For example, through advanced imaging and microscopic evaluation, researchers identified individuals with asymptomatic hyperuricemia who were found to have MSU crystal formation but had not experienced gout flares (Dalbeth & Stamp, 2014). In addition, other individuals who previously experienced gout flares within certain joints were found to have MSU crystals in other joints that seemingly were thought to be unaffected. As a result, Dalbeth and Stamp (2014) contend that gout is a chronic disease of MSU crystal deposition.

In response to their findings, Dalbeth and Stamp (2014) developed a revised staging system for hyperuricemia. Individuals in Stage A have hyperuricemia but do not

have MSU crystal deposition. In Stage B, individuals with hyperuricemia do not have gout flares, but there is evidence of MSU crystal deposition on microscopy of joint aspirate and on advanced imaging. In Stage C, MSU crystal deposition remains evident on imaging and joint aspirate and acute gout attacks are present. In Stage D, advanced gout is demonstrated by tophi, chronic gouty arthritis, and joint erosions. Referral to specialist care is indicated for individuals with Stage D (Dalbeth & Stamp, 2014).

Hyperuricemia Related to Type 2 Diabetes

Type 2 diabetes mellitus (T2DM) is one of many co-morbid conditions associated with gout and high uric acid levels. The following studies examine association between high uric acid levels in patients and risks for the development of T2DM. In a meta-analysis, Kodama et al. (2009) found that hyperuricemia was associated with T2DM development and that uric acid levels might be used as a predictor for T2DM development. In a population-based, prospective cohort study among 55 year and older subjects, Dehghan et al. (2008) showed that uric acid levels were a strong and independent risk factor for the development of T2DM. According to Krishnan et al. (2013), in a retrospective cohort study examining 1,923 United States veterans, high uric acid levels were associated with risks for developing T2DM. In a meta-analysis of prospective cohort studies, Lv et al. (2013) determined that uric acid served as a predictor for the development of T2DM in middle aged and older people. Miyake et al. (2014) observed in their retrospective, community-based, longitudinal cohort study population of 3,194 male subjects, that high uric acid levels were associated with increased risks for developing impaired fasting glucose. Meisinger et al. (2012) studied 2,970 German subjects using a population-based health survey and demonstrated that persons with pre-

diabetes and newly diagnosed diabetes had higher uric acid levels compared to normal glycemic persons. Laughon, Catov, Provins, Roberts, and Gandley (2009), in a longitudinal study of 2,215 first trimester gravid women, established that those with hyperuricemia had an increased risk of developing gestational diabetes, independent of body mass index. Rho et al. (2016) found in a matched cohort study of 35,339 individuals with gout, of which 72.4% were male, that gout was an independent risk factor that was found to be associated with the development of diabetes. This risk for diabetes was greater in the female population in comparison to males. Finally, in a review of studies, Li, Hsieh, and Chang (2013), found evidence that insulin resistance played a role in the progressive relationship between hyperuricemia and metabolic syndrome, and subsequent development of T2DM. The authors further suggested that hyperuricemia and insulin resistance may share a bi-directional causal effect. In other words, insulin affects uric acid clearance by the kidney leading to hyperuricemia, while hyperuricemia induces insulin resistance through inhibiting the supply of endothelial nitric oxide (Li et al., 2013).

Hyperuricemia Related to Chronic Kidney Disease

In the middle of the nineteenth century, gout and hyperuricemia were considered by scientists to cause renal disease, although eventually, this idea fell out of favor (Johnson et al., 2013). Recently there has been renewed interest in the study of hyperuricemia as a causal agent for renal disease (Johnson et al., 2013). Chronic kidney disease (CKD) is considered to be a comorbid condition that is associated with hyperuricemia, gout, and diabetes. The following studies examine associations between hyperuricemia and CKD.

In a review of literature, Viazzi et al. (2014) found that elevated serum uric acid led to renal damage both at the cellular level and in the tissues of the kidneys. In an observational prospective cohort study, Nacak et al. (2014) examined pre-dialysis patients, and showed that elevated serum uric acid was a risk factor for earlier progression to dialysis compared to patients with normal uric acid levels. In a cross-sectional study of 1,463 subjects, of whom 661 were male, Sheikhbahaei et al. (2014) determined that patients with hyperuricemia had a higher prevalence of CKD. In an experimental design with 50 subjects, Talaat and El-Sheikh (2007) found that asymptomatic hyperuricemia had an adverse effect on the progression of CKD. Zoppini et al. (2012) followed 1,449 subjects with T2DM in an observational longitudinal study and reported that in people with T2DM, higher uric acid levels were an independent risk factor leading to the progression to CKD.

Currently, there are no recommendations for treating asymptomatic hyperuricemia as a means to prevent gout or renal disease (Graf et al., 2015). However, in Japan, researchers using an observational study found that several nephrologists were treating asymptomatic hyperuricemia in patients who had pre-dialysis CKD in an attempt to prevent progression to dialysis (Nakaya et al., 2011). Several researchers have examined the use of allopurinol for urate lowering therapy (Bayram et al., 2014; Liu et al., 2014; Ng et al., 2014; Santhosh Pai, Swarnalatha, Ram, & Dakshinamurty, 2013; Sezer, Karakan, Atesagaogle, & Acar, 2014).

In one retrospective cohort study, Santhosh et al. (2013) observed that treatment with allopurinol lowered uric acid levels and was associated with improved blood pressure control and decreased progression to more severe forms of CKD in patients with mild

CKD and hyperuricemia. Liu et al. (2015) conducted a randomized controlled study of 176 patients and demonstrated that in comparison to usual treatment, allopurinol therapy lowered uric acid levels and increased glomerular filtration rates which may have provided kidney protection for T2DM patients with hyperuricemia. In a cross-sectional, observational study of 422 patients, Ng et al. (2014) reported that use of allopurinol in patients with CKD was independently associated with lower levels of arterial stiffness which plays a role in cardiovascular mortality in chronic renal patients. Sezer et al. (2014) conducted a cross-sectional study of 96 patients and concluded that use of allopurinol lowered uric acid which was found to control progression to CKD in pre-dialysis patients. A prospective cohort study by Bayram et al. (2014), involving 30 patients, determined that use of allopurinol lowered uric acid and improved endothelial (the interior lining of the artery) function and slowed the progression to CKD.

Diet and Lifestyle Contributing Factors

There is much evidence in the literature that supports associations between dietary factors and the development of hyperuricemia and gout (Choi & Curhan, 2008; Choi, Gao, & Curhan, 2009; Choi & Curhan, 2010; Choi et al, 2010; Zhang, Chen et al., 2012; Zhang et al., 2006; Zhang, Neogi et al., 2012). In an Internet-based, case-crossover study of 179 subjects, Zhang et al. (2006) found that alcohol intake elicited recurrent acute gout flares, with symptoms most frequently occurring within 24 hours of consumption. In another more recent crossover study, of 633 subjects, Zhang, Chen, et al. (2012) concluded that intake of purines increased risks for recurrent gout attacks in patients that were predisposed to gout. Several researchers have explored associations between the intake of fructose rich beverages and gout (Choi & Curhan, 2008; Choi et al., 2010). In a

prospective cohort study, Choi and Curhan (2008) followed participants over 12 years and reported that intake of sugar sweetened soft drinks, fruit juice, and/or other fructose rich fruits increased risks for the development of gout; although the researchers did not identify any increased risk in relation to intake of diet soft drinks. These results continued to be replicated within the same prospective cohort after 22 years (Choi et al., 2010).

Choi, Willet, and Curhan (2007) studied a sample of 45,869 males over a 12-year period and concluded that long-term coffee intake was associated with lower risks for gout occurrence. In another study, Choi and Curhan (2010) followed 89,433 females over 26 years and determined that long-term coffee intake lowered the risk for gout in this sample. In a prospective study of 46,994 males, Choi et al. (2009) examined relationships between vitamin C intake and risks for the development of gout and found that intake of higher amounts of vitamin C, greater than 1500 mg/day, lowered the risk of gout development. Zhang, Neogi et al. (2012) conducted a case-crossover study of 633 subjects and demonstrated that intake of cherries and cherry extract lowered risks for the development of gout flares.

Treatment Guidelines

Historically, treatment of acute gout, based on symptomatology, has been a main focus for health care providers. However, recently researchers have suggested that long-term management of gout through lowering of uric acid levels is important and may assist in the prevention of tophi, joint erosions, and disfiguring arthritis (Dalbeth & Stamp, 2014). In 2006, a small group of researchers and rheumatologists from the European League Against Rheumatism (EULAR) provided recommendations for the treatment of

gout (Bardin & Richette, 2013). More recently, in 2012, the American College of Rheumatology (ACR) established guidelines for the treatment and management of gout (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). These guidelines were produced by health care providers with specialization in nephrology, primary care, and rheumatology (Bardin & Richette, 2013). The guidelines are comprised of the following domains: (a) urate-lowering therapy (ULT), (b) chronic tophaceous gouty arthropathy, (c) analgesic and anti-inflammatory management of acute gout, and (d) pharmacologic and anti-inflammatory prophylaxis of acute gout (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). The first two domains are referred to as Part I and focus on non-pharmacologic and pharmacologic treatments for hyperuricemia. The third and fourth domains are referred to as Part II and consist of pharmacologic treatment approaches and anti-inflammatory prophylaxis for acute gout flares. The guidelines were published as separate manuscripts (Parts I and II) (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). Specific information about the ACR guidelines is provided in Appendix B of this manuscript.

Theoretical Framework

For this scholarly project, the interaction model of client health behavior (IMCHB) (Cox 2003) was used as a theoretical framework (see Appendix A). The IMCHB is considered to be a midrange nursing theory and is useful in primary care (McEwen, 2014). After reviewing the literature, there was no evidence of the IMCHB being used previously to guide the study or management of gout. The IMCHB consists of three main elements: client singularity, client-professional interaction, and health outcomes (Cox, 2003). The client singularity element includes the following concepts:

(a) background variables, such as demographic characteristics, social influence, previous health care experience, and environmental resources, and (b) dynamic variables, such as cognitive appraisal, affective response, and motivation. The client-professional interaction element includes the concepts of affective support, providing health information, decisional control, and professional or technical competencies. The third element, health outcomes, includes the concepts of health care utilization, health status indicators, problem-severity indicators, adherence to recommended care regimen, and satisfaction with care (Cox, 2003).

Using the IMCHB, patient health outcomes may be positively or negatively impacted by the health care provider's interventions and interactions with the client. Each client is influenced by his/her background and dynamic variables which can affect the client-professional interaction (Cox, 2003). Cox (2003) identifies a reciprocal relationship between the three dynamic variables included within the client singularity element and those of the client-professional element. According to Cox (2003), the IMCHB can be used to achieve two goals: to provide an example of using client singularity to explain health behaviors and their consequential outcomes and to help formulate interventions that target these variables.

For the purposes of this scholarly project, the IMCHB was utilized. Adult Tribal members demonstrated aspects of the client singularity concept. These individuals were involved in client-professional interactions with Tribal health care providers and the assumption was that these interactions positively impacted health outcomes. For the sample identified in this project, background variables included: (a) demographic characteristics including race and cultural influence; (b) environmental resources that

provide access to free health care (i.e. rural Tribal health care centers); (c) social influences associated with increased alcohol abuse and prevalence of T2DM and CKD; and (d) previous health care experiences (i.e. treatment for acute gout flares in comparison to starting ULT). Dynamic variables included the following: (a) cognitive appraisal of the treatment for gout based on knowledge, beliefs, and attitudes; (b) motivation for taking medications and/or making diet and lifestyle changes; and (c) affective responses based upon emotion. For the client-professional interaction element, provision of health information included patient education regarding gout, dietary and lifestyle changes, and pharmacological treatments. Professional or technical competencies could include statements like "I would like to see you get professional help to quit drinking alcohol, but the decision is yours…" Health outcomes included utilization of health care services of alcohol abuse counseling and diabetes clinic care. Clinical health status indicators included uric acid screenings in appropriate adults and monitoring of uric acid levels if pharmacologic treatment is implemented. Adherence to the recommended care regimen may depend upon proper use of ULT to prevent future gout flares. Patient satisfaction with care was expected to depend upon the patient's perception of treatment outcomes.

Chapter Three

Purpose and Sample

The purpose of this scholarly project was to implement the 2012 American College of Rheumatology (ACR) guidelines using a convenience sample of Tribal members from a mid-western portion of the United States (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). Inclusion criteria consisted of Tribal members, ages 18 years and older, who received care at Tribal health centers and had a diagnosis of gout. The Tribe utilized a computer software program called i-Care Version 2.5 (Indian Health Service, 2016), a population management tool, that was capable of generating a list of patients who meet the inclusion criteria. A report using i-Care was performed and 134 prospective participants were identified. A sample size calculator was used to determine the minimum sample size necessary to obtain adequate power for the study. Using a confidence level of 95% and a 5% margin of error, it was determined that a minimum of 108 subjects was required. Therefore, a sample size of 134 was considered to be appropriate.

Project Approval

Institutional Review Board (IRB) approval by Northern Michigan University (NMU) was obtained in spring 2016, prior to implementation of this scholarly project (See Appendix A). Anonymous aggregate patient data were collected via electronic chart reviews; thereby eliminating the need for consent to be obtained from each participant. A request for an administrative review process was submitted to the NMU IRB committee. Prior to obtaining IRB approval, permission to conduct the project was requested through a mid-western Tribal Board. Information about the study was presented to medical staff,

administration within the health division, and Tribal Board members in a workshop-type meeting that allowed for medical staff, administrators, and board members to ask questions. After the workshop was completed and questions addressed, Tribal Board members voted on the resolution.

Design and Measures

This scholarly project utilized a quasi-experimental research design (Terry, 2015). Pretest - posttest methods were used to measure uric acid levels at baseline and six months after implementation of the ACR guidelines. In addition to uric acid levels, anonymous aggregate demographic, descriptive or other data were collected from electronic chart reviews using the EHR database and included the following: (a) age, (b) gender, (c) body mass index (BMI), (d) hemoglobin A1C level, and (e) glomerular filtration rate (GFR).

Uric acid labs are routinely drawn on Tribal patients yearly and more often if patients are incurring gout flares and require medication adjustments. Uric acid lab testing uses an assay that is internationally standardized and readily accessible for use in clinical practice (Stamp et al., 2011). Uric acid remains stable at variable temperatures, has long-term storage capability, and has been found to be a reliable outcome measure for the management of gout (Stamp et al., 2011).

Procedures

In a medical staff meeting prior to implementation of the scholarly project, a document containing instructions for implementation of the 2012 ACR guidelines (see Appendix B) was presented to the Tribal medical staff which included physicians, physician assistants, and nurse practitioners. These instructions served as the protocol

that was followed by the Tribal medical staff throughout the duration of the scholarly project. In addition, an evidence-based educational handout covering the causes of gout and diet and lifestyle factors that affect gout was developed and this was distributed to patients by Tribal health care providers during the six-month implementation phase of the project (see Appendix C). The handout contains information about medications use for the treatment of gout flares, long term hyperuricemia lowering treatments, and uric acid goals. The handout also includes an individualized section where health care providers can identify the following: (a) medications that should be taken to treat a gout flare; (b) medications recommended for long term urate lowering therapy; (c) suggestions for dietary and lifestyle modifications; (d) goals for uric acid levels; and (e) dates and time for follow-up laboratory visits to re-evaluate uric acid levels. Tribal health care providers were instructed to give the handout to all patients with gout that were seen in office visits during the six-month implementation phase. The Tribe employs certified dieticians and each dietician received the clinical guideline document and the patient educational handout to ensure consistency in patient education.

Data Analysis

Sample characteristics were determined using descriptive statistics consisting of means, standard deviations, and percent values. Paired *t*-tests were used to compare uric acid levels before and after implementation of the ACR guidelines using IBM SPSS Statistics 24 software. Study participants, identified by the i-Care (Indian Health Service, 2016) software program, were assigned a study identification number and names were not attached to the data. Patient demographic, descriptive, and other laboratory data (uric acid levels, hemoglobin A1C, and GFR) were collected by an information technology

(IT) data analyst and the information was placed on a spreadsheet and used by the researcher for statistical analysis. All research materials and documents will be kept in a locked file cabinet and destroyed after seven years.

Chapter Four

Results

This chapter begins with a description of demographic characteristics of the study participants and *t*-test results which were used to determine whether there were significant differences in uric acid levels after implementation of the 2012 ACR guidelines. The results will be followed by a discussion section which includes implications of the findings for nursing clinical practice, strengths and limitations of the scholarly project, and recommendations for theory and future research.

Demographic and Descriptive Data

Using i-Care (Indian Health Service, 2016) software, data were collected from 134 American Indians with a diagnosis of gout. However, due to missing pre-intervention uric acid levels in eight subjects, the final (pre-intervention) sample consisted of 126 subjects. Of these, 126 participants received uric acid testing prior to the intervention and 46 participants received uric acid testing after the intervention. In other words, the number of participants who underwent uric acid testing prior to the intervention was higher than the previously determined minimum sample size requirement of 108 subjects and the sample size for follow-up uric acid testing was lower than the previously determined requirement for minimum sample size. The overall sample consisted of 98 males and 28 females. The age of the entire sample ranged from 24 to 90 years (M = 61.5, SD = 12.7). The age of males ranged from 24 to 82 years (M = 59, SD = 12.1). The age of females ranged from 48 to 96 years (M = 69, SD = 12.4).

Since gout is linked with other comorbid diseases such as renal disease, metabolic syndrome, central obesity, and diabetes, data were collected in relation to some of these

common diseases (Dehghan et al., 2008; Hilaire & Wozniak, 2010; Sheikhbahaei et al., 2014). A diagnosis of T2DM was present for 56 of the total 126 subjects, which comprised 44% of the sample of Tribal members with a diagnosis of gout. Hemoglobin A1C levels ranged from 4.5% to 11.9% (*M* = 6.4%, *SD* = 1.3). A diagnosis of CKD was present in 22% of the sample. The GFR ranged from 7 mL/min to > 60 mL/min (*M* = 56 mL/min, *SD* = 10). The BMI ranged from 18.9 to 56.9 (*M* = 34.4, *SD* = 6.9), demonstrating the presence of obesity for the majority of cases. In fact, only 15 of subjects (11.9%) had a BMI < 27.

Table 1

Participant Characteristics with Means and Standard Deviations

Characteristic	Total *(N = 126)*
Age	
M (SD)	61.5 (12.7)
Age of males	
M (SD)	59 (12.1)
Age of females	
M (SD)	69 (12.4)
Hemoglobin A1C	
M (SD)	6.4 (1.3)
GFR	
M (SD)	56 (10)
BMI	
M (SD)	34.4 (6.9)

Differences in Uric Acid Levels

The mean uric acid level before implementation of the 2012 ACR Guidelines was 6.41 mg/dL (*SD* = 1.9) and the mean uric level after the implementation of the guideline was 6.36 mg/dL; both of which are higher than the 2012 ACR Guideline goal of < 6.0 mg/dL. Using a paired *t*-test analysis, it was found that differences between uric acid scores prior to and after the intervention were not statistically different ($t(126) = .65$, $p = 0.52$).

Table 2

Means Scores and t-Test Results for Uric Acid Levels Before and After Implementation of the 2012 ACR Guidelines

	M	*SD*	*t*	*p*
Uric acid Pre-intervention	6.41	1.86		
Uric acid Post-intervention	6.36	1.88	0.65	0.52

* Note. * $p < .05$. ** $p < .01$.

Discussion

The purpose of this scholarly project was to implement the 2012 American College of Rheumatology (ACR) guidelines in a sample of Tribal members within a mid-western portion of the United States (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). In respect to demographic characteristics, the mean age of females was approximately 10 years older than males. This finding is consistent with the literature because prior to menopause, the presence of estrogen promotes uric acid excretion, so that after menopause, women develop a rise in uric acid levels equal to that of males (Dincer et al., 2002). Thus, women tend to develop gout about a decade later than males.

In this sample, 44% of individuals were diagnosed with T2DM in addition to gout. This percentage of T2DM is higher than what Acton et al. (2002) using a sample of American Indians reported. In Acton et al. study, 13.2% of participants were diagnosed with T2DM in addition to gout. The sample in Acton et al. study was a general population, in which it would be expected that the prevalence of gout would be in the 4% range, unlike the population of this scholarly project in which all had gout (Centers for Disease Control and Prevention, 2015). Therefore since gout is linked to diabetes, it would be expected that the percentage of individuals with T2DM would be greater in this sample (Kodama et al., 2009). In this study, a diagnosis of CKD was present in 22% of the sample. This finding is consistent with the literature that indicates many with gout also have T2DM and CKD as comorbid diagnoses (Hilaire & Wozniak, 2010). This sample also exhibited an increased mean BMI of 34.4, demonstrating the presence of obesity for the majority of cases. This finding is not unusual, as all participants in the sample were diagnosed with gout and obesity is a comorbid condition that often occurs with gout (Hilaire & Wozniak, 2010). This finding of increased obesity in Native Americans has been supported in the literature (Story et al., 1999).

The results of uric acid level testing showed a slight decrease in mean uric acid levels after implementation of the 2012 ACR guidelines. However, the differences in uric acid levels prior to and after the intervention were not statistically significant. After reviewing the data, there is a possible explanation for the lack of significance which will be explored in this section. After evaluating the data, it was found that there were only 46 cases in which a follow-up uric acid level was obtained after the six-month implementation phase. That left 80 cases, or patients, that were either not seen in the

Tribal centers or were seen by a provider but uric acid levels were not ordered and/or drawn during the six-month implementation phase. One possible explanation for this is that the providers may not have pursued follow-up testing because the patient did not report sufficient symptoms to trigger a discussion and subsequent uric acid level testing. It is possible that gout management was omitted in some of the follow-up health care visits because it was deemed less important by the providers in comparison to other health issues that needed to be addressed in a short amount of time during the patient encounter (Dincer et al., 2002).

Implications for Advanced Practice Nursing

The results of this scholarly project provide evidence to support the idea that gout remains one of the most poorly treated health conditions (Doherty et al., 2012). This finding is concerning, due to the fact that significant joint damage can occur as a result of lack of identifying and managing gout effectively (Dalbeth & Stamp, 2014). It is suggested that advanced practice registered nurses (APRN) focus on their patients' needs related to gout. Early identification and prompt treatment of gout attacks is imperative. In addition, consideration of ULT and timely follow up are suggested to ensure that uric acid levels reach recommended treatment goals (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). APRNs have long held a role as patient educators (Bastable, 2014). Therefore, it is suggested that APRNs provide education to patients about the different types of medications they receive and whether these medications are being used to treat an acute gout attack verses ULT. Also, APRNs need to educate their patients about dietary factors that influence gout management (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). As patients gain the knowledge to better

understand their condition, and treatments, levels of compliance tend to increase; thus improving health outcomes (Bastable, 2014).

Strengths and Limitations

Overall, this scholarly project had several strengths and there were some limitations which will be discussed in this section. First, implementation of the 2012 ACR guidelines provided patient benefits in relation to the opportunity to receive access to educational materials and improved gout management. In addition, the use of a quasi-experimental design was a strength. Finally, the Tribal board approved the project and the medical staff were willing to participate with implementation of the guidelines. There were some limitations identified with the scholarly project. The uric acid levels before and after implementation of the intervention remained above the treatment goal despite implementation of the 2012 ACR guidelines. Another limitation was identified in relation to the loss of study participants for follow-up testing after the implementation phase of the scholarly project. Uric acid levels were drawn on 126 patients prior to the intervention but only 46 patients received follow-up uric acid level testing. Therefore, the *t*-test results may not have reached significance due to a lack of power in relation to low sample size. A longer implementation phase and additional advertising within the Tribal health centers could have potentially increased the number of individuals that would have been able to participate in follow-up testing. In addition, the collection of prospective data may have provided a more accurate picture of the sample in comparison to using i-Care software for patient data retrieval. There could have been more buy-in by the medical staff which might have resulted in a greater effort to educate patients about gout during the six-month implementation phase. Finally, the sample was homogenous.

A more diverse American Indian sample would have improved external validity. The use of non-probability, convenience sampling is weak in comparison to probability-type sampling techniques in which participants are randomly selected from the population.

Recommendations for Future Research

This scholarly project fits nicely into the directives of the Triple Aim as developed by the Institute for Healthcare Improvement (Berwick, Nolan & Whittington, 2008). The Triple Aim consists of three goals which include improving overall population health, improving individual patient's healthcare experiences (quality and satisfaction), and improving cost effectiveness in relation to the healthcare dollars that are spent (Berwick et al., 2008). Future researchers can meet Triple Aim goals through application of theory and through implementation of the 2012 ACR guidelines for the management of gout. The first Triple Aim goal can be met by improving the health of all Tribal members by recognizing, managing, and treating gout and other comorbid diseases such as T2DM and CKD. In respect to the second Triple Aim goal, use of the IMCHB (2003) involves the health outcome of the client's satisfaction with care which can be readily evaluated as can the client's clinical health status indicators as measured by a uric acid level. Finally, the third goal of the Triple Aim can be met through proper gout management which may result in lower uric acid levels, leading to decreased gout flares and fewer subsequent clinic visits; thereby saving health care resources (Doherty et al., 2012).

Conclusion

The purpose of this scholarly project was to implement the 2012 American College of Rheumatology (ACR) guidelines using a sample of Tribal members from a

mid-western portion of the United States (Khanna, Fitzgerald, et al., 2012; Khanna, Khanna, et al., 2012). The study did not demonstrate statistically significant differences in uric acid levels prior to and after the intervention. The lack of significant findings may be attributed to the small number of participants who received post-intervention testing. Findings from this scholarly project indicates that more work needs to be done by APRNs and other healthcare providers to effectively manage gout. Clinical practice recommendations have been provided in this manuscript. The primary recommendation consists of implementation of the 2012 ACR guidelines. Use of the ACR guidelines may improve patient outcomes through early identification of gout, prompt treatment of gout attacks, consideration of ULT, patient education, and timely follow-up with periodic uric acid level testing. With proper management and education about dietary and lifestyle modifications, burdensome healthcare costs may be reduced and patients may experience enhanced health outcomes.

References

Acton, K. J., Burrows, N. R., Moore, K., Querec, L., Geiss, L. S., & Engelgau, M. M. (2002). Trends in diabetes prevalence among American Indian and Alaska Native children, adolescents, and young adults. *American Journal of Public Health, 92*(9), 1485-1490. https://doi.org/10.2105/AJPH.92.9.1485

American Kidney Fund. (2017). Race/ethnicity and kidney disease. Retrieved March 3, 2017, from http://www.kidneyfund.org/prevention/are-you-at-risk /race-ethnicity.html

Bardin, T., & Richette, P. (2013). New ACR guidelines for gout management hold some surprises. *Nature Reviews Rheumatology, 9*(1), 9-11. https://doi.org/10.1038/nrrheum.2012.216

Bastable, S. B. (2014). *Nurse as educator: Principles of teaching and learning for nursing practice* (4th ed.). Burlington, MA: Jones & Bartlett Learning.

Bayram, D., Tugrual Sezer, M., Inal, S., Altuntas, A., Kidir, V., & Orhan, H. (2014). The effects of allopurinol on metabolic acidosis and endothelial functions in chronic kidney disease patients. *Clinical and Experimental Nephrology, 19*(3), 443-449. https://doi.org/10.1007/s10157-014-1012-z

Beauvais, F. (1998). American Indians and alcohol. *Alcohol Health & Research World, 22*(4), 235-259. Retrieved from http://www.worldcat.org/title /alcohol-health-and-research-world/oclc/1785965

Berwick, D. M., Nolan, T. W., & Whittington, J. (2008). The triple aim: Care, health, and cost. *Health Affairs, 27*(3), 759-769. https://doi.org/10.1377/hlthaff.27.3.759

Centers for Disease Control and Prevention. (2011). CDC health disparities & inequalities report. Retrieved from https://www.cdc.gov/minorityhealth/chdir/2011/factsheet.pdf

Centers for Disease Control and Prevention. (2015). Gout. Retrieved from http://www.cdc.gov/arthritis/basics/gout.html

Choi, H. K., & Curhan, G. (2008). Soft drinks, fructose consumption, and the risk of gout in men: Prospective cohort study. *BMJ, 336*(7639), 309-312. https://doi.org/10.1136/bmj.39449.819271.BE

Choi, H. K., & Curhan, G. (2010). Coffee consumption and risk of incident gout in women: The Nurses' Health Study. *American Journal of Clinical Nutrition, 92*(4), 922-927. https://doi.org/10.3945/ajcn.2010.29565

Choi, H. K., Gao, X., & Curhan, G. (2009). Vitamin C intake and the risk of gout in men— A prospective study. *Archives of Internal Medicine, 169*(5), 502-507. https://doi.org/10.1001/archinternmed.2008.606

Choi, H. K., Mount, D. B., & Reginato, A. M. (2005). Pathogenesis of gout. *Annals of Internal Medicine, 143*(7), 499-516. https://doi.org/10.7326/0003-4819-143-7-200510040-00009

Choi, H. K., Willett, W., & Curhan, G. (2007). Coffee consumption and risk of incident gout in men: A prospective study. *Arthritis and Rheumatism, 56*(6), 2049-2055. https://doi.org/10.1002/art.22712

Choi, H. K., Willett, W., & Curhan, G. (2010). Fructose-rich beverages and the risk of gout in women. *Journal of the American Medical Association, 304*(20), 2270-2278. https//doi.org/10.1001/jama.2010.1638

Cox, C. L. (2003). A model of health behavior to guide studies of childhood cancer survivors. *Oncology Nursing Forum, 30*(5), E92-E99. https://doi.org/10.1188/03.ONF.E92-E99

Dalbeth, N., & Stamp, L. (2014). Hyperuricaemia and gout: Time for a new staging system? *Annals of the Rheumatic Diseases, 73*(9), 1598-1600. https://doi.org/10.1136/annrheumdis-2014-205304

Dehghan, A., van Hoek, M., Sijbrands, E. J. G., Hofman, A., & Witteman, J. C. M. (2008). High serum uric acid as a novel risk factor for type 2 diabetes. *Diabetes Care, 31*(2), 361-362. https://doi.org/10.2337/dc07-1276

Dincer, H. E., Dincer, A. P., & Levinson, D. J. (2002). Asymptomatic hyperuricemia: To treat or not to treat. *Cleveland Clinic Journal of Medicine, 69*(8), 594-608. https://doi.org/10.3949/ccjm.69.8.594

Doherty, M., Jansen, T. L., Nuki, G., Pascual, E., Perez-Ruiz, F., Punzi, L., … Bardin, T. (2012). Gout: Why is this curable disease so seldom cured? *Annals of the Rheumatic Diseases, 71*(11), 1765-1770. https://doi.org/10.1136/annrheumdis-2012-201687

Espey, D. K., Jim, M. A., Cobb, N., Bartholomew, M., Becker, T., Haverkamp, D., & Plescia, M. (2014). Leading causes of death and all-cause mortality in American Indians and Alaska Natives. *American Journal of Public Health, 104*(Suppl. 3), 303-311. https://doi.org/10.2105/AJPH.2013.301798

Graf, S. W., Whittle, S. L., Wechalekar, M. D., Moi, J. H. Y., Barrett, C., Hill, C. L., … Zochling, J. (2015). Australian and New Zealand recommendations for the diagnosis and management of gout: Integrating systematic literature review and

expert opinion in the 3e initiative. *International Journal of Rheumatic Diseases, 18*(3), 341-351. https://doi.org/10.1111/1756-185x.12557

Greene, K. M., McNulty-Eitle, T., & Eitle, D. (2014). Adult social roles and alcohol use among American Indians. *Addictive Behaviors, 30*(9), 1357-1360. https://doi.org/10.1016/j.addbeh.2014.04.024

Hilaire, M. L., & Wozniak, J. R. (2010). Gout: Overview and newer therapeutic developments. *Formulary, 45*(3), 84-90. Retrieved from http://www.formularyjournal.com

Indian Health Service. (2012). Diabetes in American Indians and Alaska Natives: Facts at-a-glance. Retrieved from https://www.ihs.gov/MedicalPrograms/Diabetes /HomeDocs/Resources/FactSheets/Fact_sheet_AIAN_508c.pdf

Indian Health Service. (2016). iCare (Version 2.5) [Computer software]. Retrieved from https://www.ihs.gov/icare/

Johnson, R. J., Nakagawa, T., Jalal, D., Sanchez-Lozada, L. G., Kang, D.-H., & Ritz, E. (2013). Uric acid and chronic kidney disease: Which is chasing which? *Nephrology Dialysis Transplantation, 28*(9), 2221-2228. https://doi.org/10.1093/ndt/gft029

Khanna, D., Fitzgerald, J. D., Khanna, P. P., Bae, S., Singh, M. L., Neogi, T., … Terkeltaub, R. (2012). 2012 American College of Rheumatology guidelines for management of gout. Part 1: Systematic nonpharmacologic and pharmacologic therapeutic approaches to hyperuricemia. *Arthritis Care & Research, 64*(10), 1431-1446. https://doi.org/10.1002/acr.21772

Khanna, D., Khanna, P. P., Fitzgerald, J. D., Singh, M. K., Bae, S., Neogi, T., … Terkeltaub, R. (2012). 2012 American College of Rheumatology guidelines for management of gout. Part 2: Therapy and anti-inflammatory prophylaxis of acute gouty arthritis. *Arthritis Care & Research, 64*(10), 1447-1461. https://doi.org/10.1002/acr.21773

Kodama, S., Saito, K., Yachi, Y., Asumi, M., Sugawara, A., Totsuka, K., … Sone, H. (2009). Association between serum uric acid and development of type 2 diabetes. *Diabetes Care, 32*(9), 1737-1742. https://doi.org/10.2337/dc09-0288

Krishnan, E., Akhras, K. S., Sharma, H., Marynchenko, M., Wu, E. Q., Tawk, R., … Shi, L. (2013). Relative and attributable diabetes risk associated with hyperuricemia in US veterans with gout. *Quarterly Journal of Medicine, 106*(8), 721-729. https://doi.org/10.1093/qjmed/hct093

Laughon, S. K., Catov, J., Provins, T., Roberts, J. M., & Gandley, R. E. (2009). Elevated first-trimester uric acid concentrations are associated with the development of gestational diabetes. *American Journal of Obstetrics & Gynecology, 201*(4), 402.e1-402.e5. https://doi.org/10.1016/j.ajog.2009.06.065

Li, C., Hsieh, M.-C., & Chang, S.-J. (2013). Metabolic syndrome, diabetes, and hyperuricemia. *Current Opinion Rheumatology*, *25*(2), 210-216. https://doi.org/10.1097/BOR.0b013e32835d951e

Liu, P., Chen, Y., Wang, B., Zhang, F., Wang, D., & Wang, Y. (2015). Allopurinol treatment improves renal function in patients with type 2 diabetes and asymptomatic hyperuricemia: 3-year randomized parallel-controlled study. *Clinical Endocrinology*, *83*(4), 475-482. https://doi.org/10.1111/cen.12673

Lv, Q., Meng, X.-F., He, F.-F., Chen, S., Su, H., Xiong, J., … Zhang, C. (2013). High serum uric acid and increased risk of type 2 diabetes: A systemic review and meta-analysis of prospective cohort studies. *PLoS One, 8*(2), 1-7. https://doi.org/10.1371/journal.pone.0056864

McEwen, M. (2014). Introduction to middle range nursing theories. In M. McEwen, & E. Wills, *Theoretical basis for nursing* (4th ed.,pp. 213-228). Philadelphia, PA: Lippincott Williams & Wilkins.

Meisinger, C., Doring, A., Stockl, D., Thorand, B., Kowall, B., & Rathmann, W. (2012). Uric acid is more strongly associated with impaired glucose regulation in women than in men from the general population: The KORA F4-study. *PLoS One, 7*(5), 1-7. https://doi.org/10.1371/journal.pone.0037180

Miyake, T., Kumagi, T., Furukawa, S., Hirooka, M., Kawasaki, K., Koizumi, M., … Hiasa, Y. (2014). Hyperuriecmia is a risk factor for the onset of impaired fasting glucose in men with a high plasma glucose level: A community-based study. *PLoS One, 9*(9), 1-11. https://doi.org/10.1371/journal.pone.0107882

Nacak, H., van Diepen, M., de Goeij, M. C. M., Rotmans, J. I., & Dekker, F. W. (2014). Uric acid: Association with rate of renal function decline and time until start of dialysis in incident pre-dialysis patients. *Nephrology, 15*, 91. https://doi.org/10.1186/1471-2369-15-91

Nakaya, I., Namikoshi, T., Tsuruta, Y., Nakata, T., Shibagaki, Y., Onishi, Y., & Fukuhara, S. (2011). Management of asymptomatic hyperuricaemia in patients with chronic kidney disease by Japanese nephrologists: A questionnaire survey. *Nephrology, 16*(5), 518-521. https://doi.org/10.1111/j.1440-1797.2011.01446.x

Ng, K. P., Stringer, S. J., Jesky, M. D., Yadav, P., Athwal, R., Dutton, C., … Cockwell, P. (2014). Allopurinol is an independent determinant of improved arterial stiffness in chronic kidney disease: A cross-sectional study. *PLoS One, 9*(3), 1-8. https://doi.org/10.1371/journal.pone.0091961

Norris, T., Vines, P. L., & Hoeffel, E. M. (2012). The American Indian and Alaska Native population: 2010 Census briefs. Retrieved from http://www.census.gov /prod/cen2010/briefs/c2010br-10.pdf

Rho, Y. H., Lu, N., Peloquin, C. E., Man, A., Zhu, Y., Zhang, Y., & Choi, H. K. (2016). Independent impact of gout on the risk of diabetes mellitus among women and men: A population-based, BMI-matched cohort study. *Annals of Rheumatic Diseases, 75*(1), 91-95. https://doi.org/10.1136/annrheumdis-2014-205827

Santhosh Pai, B. H., Swarnalatha, G., Ram, R., & Dakshinamurty, K. V. (2013). Allopurinol for prevention of progression of kidney disease with hyperuricemia. *Indian Journal of Nephrology, 23*(4), 280-286. https://doi.org /10.4103/0971-4065.114499

Sezer, S., Karakan, S., Atesagaoglu, B., & Acar, F. N. O. (2014). Allopurinol reduces cardiovascular risks and improves renal function in pre-dialysis chronic kidney disease patients with hyperuricemia. *Saudi Journal of Kidney Diseases and Transplantation, 25*(2), 316-320. Retrieved from http://www.sjkdt.org

Sheikhbahaei, S., Fotouhi, A., Hafezi-Nejad, N., Nakhjavani, M., & Esteghamati, A. (2014). Serum uric acid, the metabolic syndrome, and the risk of chronic kidney disease in patients with type 2 diabetes. *Metabolic Syndrome and Related Disorders, 12*(2), 102-109. https://doi.org/10.1089/met.2013.0119

Simkin, P. A. (2008). Sharing decisions in gout: Better communication for better outcomes. The *Journal of Musculoskeletal Medicine, 25*(3), 116-118, 124-125. Retrieved from http://www.ubmmedica.com/

Stamp, L. K., Zhu, X., Dalbeth, N., Jordan, S., Edwards, N. L., & Taylor, W. (2011). Serum urate as a soluble biomarker in chronic gout: Evidence that serum urate fulfills the OMERACT validation criteria for soluble biomarkers. *Seminars in Arthritis & Rheumatism, 40*(6), 483-500. https://doi.org/10.1016/j.semarthrit.2010.09.003

Story, M., Evans, M., Fabsitz, R. R., Clay, T. E., Holy Rock, B., & Broussard, B. (1999). The epidemic of obesity in American Indian communities and the need for childhood obesity-prevention programs. *American Journal of Clinical Nutrition, 69*(Suppl. 4), 747-754. https://doi.org/10.2337/diacare.22.2.345

Talaat, K. M., & El-Sheikh, A. R. (2007). The effect of mild hyperuricemia on urinary transforming growth factor beta and the progression of chronic kidney disease. *American Journal of Nephrology, 27*(5), 435-440. https://doi.org/10.1159/000105142

Terry, A. J. (2015). *Clinical research for the doctor of nursing practice* (2nd ed.). Burlington, MA: Jones & Bartlett Learning.

Viazzi, F., Leoncini, G., Ratto, E., & Pontremoli, R. (2014). Hyperuricemia and renal risk. *High Blood Pressure Cardiovascular Prevention, 21*(3), 189-194. https://doi.org/10.1007/s40292-014-0042-7

Zhang, Y., Chen, C., Choi, H., Chaisson, C., Hunter, D., Niu, J., & Neogi, T. (2012). Purine-rich goods intake and recurrent gout attacks. *Annals of the Rheumatic Diseases, 71*, 1448-1453. https://doi.org/10.1136/annrheumdis-2011-201215

Zhang, Y., Neogi, T., Chen, C., Chaisson, C., Hunter, D., & Choi, H. K. (2012). Cherry consumption and the risk of recurrent gout attacks. *Arthritis Rheumatology, 64*(12), 4004-4011. https://doi.org/10.1002/art.34677

Zhang, Y., Woods, R., Chaisson, C. E., Neogi, T., Niu, J., McAlindon, T. E., & Hunter, D. (2006). Alcohol consumption as a trigger of recurrent gout attacks. *American Journal of Medicine, 119,* 13-18. https://doi.org/10.1016/j.amjmed.2006.01.020

Zhu, Y., Pandya, B. J., & Choi, H. K. (2011). Prevalence of gout and hyperuricemia in the US general population: The National Health and Nutrition Examination Survey 2007-2008.. *Arthritis & Rheumatism, 63*(10), 3136-3141. https://doi.org/10.1002/art.30520

Zoppini, G., Targher, G., Chonchol, M., Ortalda, V., Abaterusso, C., Pichiri I., … Bonora, E. (2012). Serum uric acid levels and incident chronic kidney disease in patients with type 2 diabetes and preserved kidney function. *Diabetes Care, 35*(1), 99-104. https://doi.org/10.2337/dc11-1346

Zychowicz, M. E., Pope, R. S., & Graser, E. (2010). The current state of care in gout: Addressing the need for better understanding of an ancient disease. *Journal of the American Academy of Nurse Practitioners, 22*, 623-636. https://doi.org/10.1111/j.1745-7599.2010.00556.x

Appendix A

Memorandum

TO: Myrth Condon
School of Nursing

CC: Melissa Romero
School of Nursing

FROM: Dr. Robert Winn
Assistant Provost/IRB Administrator

DATE: April 4, 2016

SUBJECT: IRB Proposal HS16-751
"Implementation of the 2012 American College of Rheumatology Guidelines for Management of Gout in a Sample of Midwestern Native Americans"
IRB Approval Dates: 4/4/2016-4/4/2017**
Proposed Project Dates: 4/4/2016-12/30/2016

Your proposal has "Implementation of the 2012 American College of Rheumatology Guidelines for Management of Gout in a Sample of Midwestern Native Americans" been approved under the administrative review process. Please include your proposal number (HS16-751) on all research materials and on any correspondence regarding this project.

Any changes or revisions to your approved research plan must be approved by the Institutional Review Board (IRB) prior to implementation.

**If you do not complete your project within 12 months from the date of your approval notification, you must submit a Project Renewal Form for Research Involving Human Subjects. You may apply for a one-year project renewal up to four times.

All forms can be found at the NMU Grants and Research website:
http://www.nmu.edu/grantsandresearch/node/102

Appendix B

Clinical Guideline: Management of Gout

The following guideline contains recommendations for pharmacologic and non-pharmacologic treatments for the management of gout. This guideline was adapted from the 2012 American College of Rheumatology Guidelines for Management of Gout Part 1: Systematic Non-pharmacologic and Pharmacologic Therapeutic Approaches for Hyperuricemia and Part 2: Therapy and Anti-inflammatory Prophylaxis of Acute Gouty Arthritis

Causes of Gout:

Approximately 20% of patients with hyperuricemia (uric acid level of >6.8 mg/dL) will progress to gout (Dalbeth & Stamp, 2014). Gout occurs due to either urate under-excretion, overproduction, or both (Dincer et al., 2002). Under-excretion is the most common cause of gout (90% of cases) (Choi, Mount, & Reginato, 2005) due to: chronic kidney disease, genetic deficiency of an enzyme, uricase, that converts uric acid to allantoin, increased alcohol intake, or use of certain medications like thiazide and loop diuretics, low-dose ASA therapy, and niacin (Zychowicz et al., 2010). Overproduction of uric acid is mainly diet related due to over-intake of purine rich foods and genetic factors (Choi et al., 2005). After a diagnosis of gout has been made, health care providers should identify whether any offending medications such as thiazide or loop diuretics, niacin, or low dose aspirin* should be changed or discontinued.

*Note: there is no recommendation to stop low dose aspirin therapy in patients for whom it is deemed appropriate, such as diabetics

I. Recommendations for Pharmacological Urate Lowering Therapy (ULT)

ULT should be initiated when a patient has a diagnosis of gout and any one of the following condition:

- Tophus or tophi seen on physical exam or imaging study
- Two or more acute gout flares annually
- Chronic kidney disease, stage 2 or worse
- Past history of urolithiasis

ULT Pharmacologic Agents:

- **Allopurinol** (*Zyloprim*): Starting dose is 100 mg daily, with gradual titration by 100 mg increments every 2-5 weeks to obtain a goal of uric acid level of < 6.0 mg/dL (maximum FDA approved dose is 800 mg/day)
    - Currently, there is no recommended lower level of dosing due to renal impairment. Monitor for signs and symptoms of drug toxicity (pruritis, rash and elevated liver enzymes)
- **Febuxostat** (*Uloric*): **S**tarting dose, 40 mg daily, with increase to 80 mg daily after 2 weeks (maximum international dose is 120 mg daily)
- **Probenecid**, **Fenofibrate** (*Tricor*) and **Losartan** (*Cozaar*) can also be used to lower uric acid alone or in combination

The goal of ULT is to reduce serum uric acid levels to < 6.0 mg/dL (for some patients, the goal may need to be < 5.0 mg/dL in order to achieve improvements in gout signs and symptoms). Uric acid levels should be tested in 2-5 weeks after starting ULT and medication doses can be titrated up as recommended above to achieve this goal. ULT is then continued indefinitely to maintain a goal uric acid level of < 6.0 mg/dL. Once ULT begins, it is recommended to combine ULT with anti-inflammatory medications and to continue these as long as necessary to provide acute gout flare prophylaxis

II. Therapy and Anti-inflammatory Prophylaxis of Acute Gouty Arthritis:

Therapy should start within the first 24 hours of gout flare. Ongoing ULT should not be interrupted during an acute gout flare. If symptoms are mild to moderate (only a few small joints or one to two large joints affected) then monotherapy can be used. If symptoms are severe (polyarticular involvement), then combination therapy can be used

Monotherapy:

- **NSAIDs:**
    - **Naproxen** (*Naprosyn*) 750 mg once, then 250 mg every 8 hours prn
    - **Indomethacin** (*Indocin*) 50 mg TID prn
- **Cox-2 Inhibitors:**
    - **Celecoxib** (*Celebrex*) 100-200 mg BID prn

- **Colchicine,** Starting dose**,** 1.2 mg, followed by 0.6 mg one hour later if needed for pain control. After 12 hours, it can continue to be dosed at 0.6mg daily or BID until symptoms are controlled

- **Oral corticosteroids:**
  - **Prednisone** or **Prednisolone:** 0.5 mg/kg per day for 5-10 days and discontinue, or continue dosing for 2-5 days, then tapered for 7-10 days and discontinue

- **Intramuscular and Intraarticular Corticosteroids:**
  - **Triamcinolone acetonide:** 60 mg IM injection, followed by oral prednisone or prednisolone
  - **Intraarticular corticosteroid injections** may be used in one or two large joints if they are the only joints affected

Combination Therapy Options:

If monotherapy agents are not effective in relieving acute gout symptomatology, drug therapy can be changed or combined to achieve an adequate response. Combination therapy options are listed below:

- Colchicine can be combined with NSAIDs, or oral corticosteroids
- Intraarticular steroids can be combined with any of the monotherapy options

Application of **ice** to the affected joint(s) is also recommended

Dietary and Lifestyle Recommendations:
Health care providers are encouraged to identify factors that contribute to gout and encourage patients to make the following dietary and lifestyle modifications:

Dietary Recommendations:
- Avoid foods high in purines such as: organ meats (liver, kidney, sweetbreads)
- Limit serving sizes of beef, pork, lamb and seafood (sardines, shellfish)
- Avoid high fructose corn syrup sweetened beverages or foods
- Limit fruit juices and other sweetened beverages and desserts
- Avoid all alcohol during an acute gout flare
- Limit alcohol intake to 1 to 2 drinks for males and 1 drink for females per day
- Limit daily use of table salt; especially in sauces and gravies,
- Encourage the intake of vegetables and low-fat or non-fat dairy products

Lifestyle Recommendations:
- Weight loss for obese patients
- Smoking cessation
- Regular exercise to achieve physical fitness
- Regular fluid intake to achieve adequate hydration status

Clinical Guideline References

Choi, H. K., Mount, D. B., & Reginato, A. M. (2005). Pathogenesis of gout. *Annals of Internal Medicine*, *143*(7), 499-516. https://doi.org/10.7326/0003-4819-143-7-200510040-00009

Dalbeth, N., & Stamp, L. (2014). Hyperuricaemia and gout: Time for a new staging system? *Annals of the Rheumatic Diseases, 73*(9), 1598-1600. https://doi.org/10.1136/annrheumdis-2014-205304

Dincer, H. E., Dincer, A. P., & Levinson, D. J. (2002). Asymptomatic hyperuricemia: To treat or not to treat. *Cleveland Clinic Journal of Medicine, 69*(8), 594-608. https://doi.org/10.3944/ccjm.69.8.594

Khanna, D., Fitzgerald, J. D., Khanna, P. P., Bae, S., Singh, M. L., Neogi, T., … Terkeltaub, R. (2012). 2012 American College of Rheumatology guidelines for management of gout. Part I: Systematic nonpharmacologic and pharmacologic therapeutic approaches to hyperuricemia. *Arthritis Care & Research, 64*(10), 1431-1446. https://doi.org/10.1002/acr.21772

Khanna, D. Khanna, P. P., Fitzgerald, J. D. , Singh, M. K., Bae, S., Neogi, T., … Terkeltaub, R., (2012). 2012 American College of Rheumatology guidelines for management of gout. Part II: Therapy and anti-inflammatory prophylaxis of acute gouty arthritis. *Arthritis Care & Research, 64*(10), 1447-1461. https://doi.org/10.2337/dc09-0288

Zychowicz, M. E., Pope, R. S., & Graser, E. (2010). The current state of care in gout: Addressing the need for better understanding of an ancient disease. *Journal of the American Academy of Nurse Practitioners, 22*, 623-636. https://doi.org/10.1111/j.1745-7599.2010.00556.x

Appendix C

Information on Gout for Patients

Gout is an inflammatory disease in which uric acid levels in the blood are too high. When the uric acid level runs too high, crystals form and deposit in the joints and soft tissues of the body and can even form kidney stones. These crystals are very tiny and are sharp, needle-like. When enough crystals are present, they will cause a classic gout attack, sometimes called a gout flare. The most common joint involved in a gout attack is the great toe, but other joints can be affected, like hands, knees, or ankles. Symptoms of a gout attack typically include: redness, swelling and pain. Often it is difficult to put weight on the affected joint or to walk.

Medications are often used to prevent and treat gout. Long term medicine treatment is often used to help prevent future gout attacks and physical problems. A medicine called Zyloprim (allopurinol) is often used, long term, to lower uric acid in the blood. There are some other medications that may be prescribed such as anti-inflammatory medications which can be used to help prevent a gout attack. If you have gout and/or are receiving medications to treat gout, you will need to have your blood tested periodically to see if the uric acid level has been lowered.

You can make changes in your diet and lifestyle to help lower the risk of gout attacks and to help lower your overall uric acid level. Avoid organ meats such as kidney, liver, and sweetbreads, in addition to other meats like seafood (sardines, shellfish, anchovies), beef, lamb, and pork. Studies have shown that intake of high sugary beverages (soda pop, fruit juices, sport drinks) and desserts can also cause gout attacks. In addition, avoid increased table salt, especially in sauces and gravies. Alcohol has been found to be associated with gout attacks. Limit your daily alcohol intake to less than one to two drinks for men and one drink for women. Always avoid all alcohol during gout attacks. In order to prevent gout attacks, drink low-fat or non-fat dairy products, drink at least one liter of fluid daily, and eat plenty of vegetables. Coffee, cherries, cherry extract, and vitamin C (commonly found in citrus fruits) have been shown to lower risks for gout. Gout has been found to be associated with obesity and diabetes, so weight loss is encouraged in addition to daily exercise.

This worksheet is intended to be used by you and your provider to track your uric acid levels.

Name: _____

Current uric acid level: _____ Goal uric acid level: _____

Return to clinic for follow up appointment/blood testing on: _____

Current uric acid lowering medication plan: _____

_____

_____

Medication plan to treat an acute gout attack or flare: _____

_____

_____

_____

Dietary/lifestyle goals: _____

_____

_____

_____

_____