ORIGINAL ARTICLE

INTERNATIONAL JOURNAL OF HEALTH MEDICINE AND CURRENT RESEARCH

EFFECT OF GEDI LEAF DECOCTION TO DECREASE URIC ACID LEVEL IN PATIENTS WITH GOUT ARTHRITIS

Siti Sultoni [1], Arend L. Mapanawang [2], Averous Faraby Budiadji [3], Sumiati Badoa [4], Roberto Cabu [5]

[1] Nursing Department of STIKES Halmahera
[2] Yayasan Medika Mandiri

ABSTRACT

Gout Arthritis is an inflammation of the joints as a manifestation of the accumulation of monosodium urate crystals precipitate, which is collected in the joints as a result of the high uric acid levels in the blood (hyperuricemia). However, not all people with hyperuricemia suffer from gout arthritis. However, the risk of gout arthritis is greater with the increasing concentration of uric acid in the blood (Noor ZH, 2013).

The purpose of this study is to analyze the effect of Gedi *(Abelmoschus manihot* (L.) leaf decoction on uric acid levels in patients with gout arthritis in Ngoali Village, Kao Barat Subdistrict, Halmahera Utara District. This research is a quantitative research with *quasi design experiment with pre - posttest control group.* The sampling technique used is *non-probability, consecutive sampling* with a total samples of 10 respondents divided into an experimental group and a control group.

The results of SPSS analysis using T-Test showed that the experimental group T-count value was 3,942 (> T-table of 2,776) with a value of $p = 0.017$ (<0.05), whereas the control group T-count value was 0,925 (< T-table of 2,776) with a value of $p = 0.408$ (> 0.05).
It can be concluded that Ho is rejected and Ha is accepted if the $p < 0.05$, which means that there is significant influence of Gedi leaf decoction in decreasing uric acid levels in patients with gout arthritis.

Citation: **Siti Sultoni[1], Arend L. Mapanawang[2], Averous Faraby Budiadji[3], Sumiati Badoa[4], Roberto Cabu[5], 2016** "*Effect Of Gedi Leaf Decoction To Decrease Uric Acid Level In Patients With Gout Arthritis*", *International Journal of Health Medicine and Current Research,* 1, (02), 194-200.

INTRODUCTION

Gout arthritis is a disease that is often found around the world. Gout arthritis is a heterogeneous group of diseases as a result of the deposition of monosodium urate crystals in the tissues or supersaturation of uric acid in the extracellular. Metabolic disorders that trigger gout is hyperuricemia, which is an increase in uric acid levels over 7.0 mg / dl and 6.0 mg / dl (Edward Stephen, 2010).

The prevalence of uric acid (gout) in the United States has doubled in the 75 years between 1990 and 1999, from 21 per 1000 to 41 per 1000. Meanwhile, in the UK, the prevalence of gout in the adult population is estimated at around 1.4%, with the highest prevalence (7%) in men aged 75 years (Alexander, 2010).

The prevalence of gout in the world varies. Epidemiological studies found that the incidence of uric acid has increased, especially in developed countries, whose population consumes fatty foods that contain high levels of purines.

The prevalence of hyperuricemia varies in different population of approximately 2.6 to 47.2% and the prevalence of gout also varies about 1 to 15.3%. One study found the incidence of gout at 4.9% in blood uric acid levels of > 9 mg / dL, 0.5% in the levels of 7 - 8.9, and 0.1% in the levels of <7 mg / dL. The cumulative incidence of gout reaches 22% after 5 years in the uric acid levels of > 9 mg / dL. In Indonesia, gout arthritis ranks as the second highest of osteoarthritis disease. Research results showed that most patients (65%) with gout arthritis experienced hyperuricemia (Alifiasari 2010).

The prevalence of joint disease nationwide is 30.3% (based on health professionals diagnosis and symptoms). However, the prevalence in 11 provinces, namely Nanggroe Aceh Darussalam, West Sumatra, Bengkulu, West Java, Central Java, East Java, Bali, Nusa Tenggara Barat, Nusa Tenggara East, South Kalimantan, and Papua Barat, was above the national average (Riskesdas, cited 2007 May 15, 2014). According to Riskerdas 2013, the prevalence of joint disease based on diagnosis by health workers in Indonesia was 11,9% while based on symptoms it was 24.7%. Seen from age characteristics, the highest prevalence occurs at age $\geq$ 75 years (54.8%). More women (27.5%) than men (21.8%) suffered from this diseasi. The prevalence of gout in Indonesia that occurred at ages under 34 years old was 32% with the highest incidence found in Minahasa (29.2%) (Buraerah in Kodim 2010).

Research by Ervi Diantari, Aryu Candra " Pengaruh Asupan Purin dan Cairan Terhadap Kadar Asam Urat Wanita Usia 50-60 Tahun " showed that uric acid levels of most subjects (95%) are in the normal category with 82.5% of the subjects having low purine intake, i.e. <500 mg per day and 85% of the subjects having enough fluid intake, i.e.> 1500 ml per day. Statistical test results showed the absence of influence between the liquid and uric acid levels (p> 0.05) and the positive influence between the intake of purines and uric acid levels (p <0.05). It was concluded that **a** purine intake influenced uric acid levels, while the fluid had no effect on uric acid levels in women aged 50-60 years.

Gedi is a tropical plant that belongs to Malvaceae family. It is a *Perrenial* plant that grows in Indonesia and the South Pacific region. The leaves have a soft texture and sweet taste, which can be eaten fresh or steamed, but should be eaten immediately after being picked as these leaves easily wither.

Jain *et al,* (2011) reported on the research by Gupta, *et al,* (2008) that phytochemical analysis of Gedi extracts shows that it contains steroids, triterpenoids and flavonoids that have analgesic activity. In addition, the research by Oyedapo, *et al.* (2008) found that Gedi show antioxidant and anti-inflammatory activities.

METHODS

The design of this study is Q*uasi Experiment Design Control Group* with *pre* and *posttest* with two groups selected at random. The first group is given treatment while the second is not. The treated group is called experimental group and the untreated group is called control group.

Pretest is done for the two groups, and then the first group *(experimental group)* is given treatment while the second group *(control group)* is not. After that, *posttest is* done on both groups to compare the results from the treated group *(experimental group)* and the untreated group *(control group).*

The study design is described below.

Pretest		Posttest
O_1	x	O_3
O_2		O_4

Notes:

O_1 = The measurement results of the uric acid levels before consuming Gedi leaf decoction in the intervention group (experimental)

O_2 = The measurement results of the uric acid levels in the control group

X = Gedi leaf decoction intervention

O_3 = The measurement results of the uric acid levels after consuming Gedi leaf decoction in the intervention group (experimental)

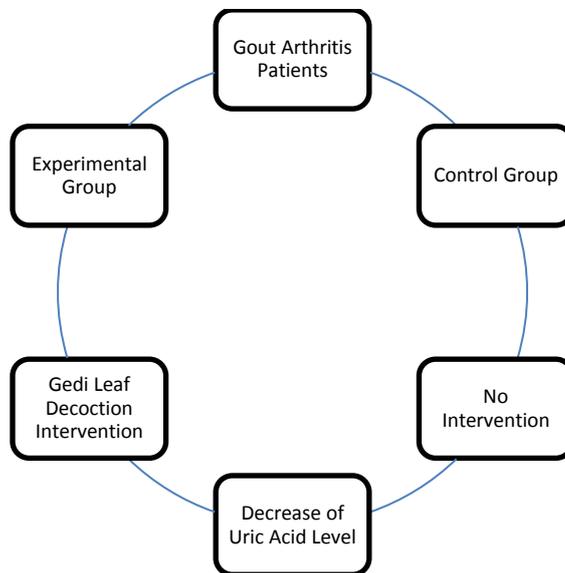O_4 = The measurement results of the uric acid levels in the control group

Figure 1. Research Framework

SAMPLE

The sample is part of the number and characteristics possessed by population (Sugiyono, 2010). Samples proportion was obtained from a population based on the number of gout patients who will undergo the intervention to decrease their uric acid levels in Ngoali village, Kao Barat subdistrict, Halmahera Utara district.

The sampling technique used is saturated sampling, where all members of the population are sampled. This is done when the population size is small, less than 30, so all members of this population are taken to be research samples (Hidayat, 2010). The samples used were 10 people, divided into two groups of five. The characteristics of the sample used are inclusion and exclusion criteria.

INTERVENTION PROCEDURE

a. **Intervention group**

1. Researchers ensure that respondents accept intervention procedures and introduce themselves to the respondents.

2. Researchers explain to the respondents about the definition, purpose, method, and the benefits of Gedi leaf decoction and the implementation time and procedures of the intervention.

3. Researchers give the respondents opportunities to ask questions and give *informed consent,* request a signature as proof of consent for respondents willing to participate in research activities.

b. **Control group**

Patients in the control group receive only health education. The questionnaire is done twice *(pre and post).*

RESULTS

Patients with gout arthritis in Ngoali village consisted of two men and eight women. Based on age, four people aged 21-42 years, two people aged 43-53 years, and four people aged > 60 years. The data was analyzed using univariate and bivariate. The univariate analysis in this study is the characteristics of respondents by sex, age, and education.

Table 1. Occupation

		Frequency	Percentage
Valid	Housewives	8	80.0
	Private	1	10.0
	Retired Teacher	1	10.0
	Total	10	100.0

Table 2. Education

		Frequency	Percentage
Valid	SD	8	80.0
	SMP	1	10.0
	PGSD	1	10.0
	Total	10	100.0

Table 3. Frequency Distribution of Uric Acid Levels In Control group (pretest)

No.	Levels of Uric Acid in Control Group (pretest)	Frequency	Percentage
1	10.3 mg/dL	1	10%
2	8.4 mg/dL	1	10%
3	13.0 mg/dL	1	10%
4	8.6 mg/dL	1	10%
5	7.4 mg/dL	1	10%
	Total	5	50%

Table 4. Frequency Distribution of Uric Acid Levels in Control group (posttest)

No.	Levels of Uric Acid in Control Group (posttest)	Frequency	Percentage
1	9.4 mg/dL	1	10%
2	9.5 mg/dL	1	10%
3	12.1 mg/dL	1	10%
4	8.5 mg/dL	1	10%
5	6.5 mg/dL	1	10%
	Total	5	50%

Table 5. Frequency Distribution of Uric Acid Levels in Experiment group (Pretest)

No.	Levels of Uric Acid in Experiment Group (posttest)	Frequency	Percentage
1	11.6 mg/dL	1	10%
2	10.4 mg/dL	1	10%
3	13.0 mg/dL	1	10%

No.		Frequency	Percentage
4	7.5 mg/dL	1	10%
5	9.7 mg/dL	1	10%
	Total	5	50%

Table 6. Frequency Distribution of Uric Acid Levels in Experiment group (Posttest)

No.	Levels of Uric Acid in Experiment Group (posttest)	Frequency	Percentage
1	5.4 mg/dL	1	10%
2	7.4 mg/dL	1	10%
3	6.3 mg/dL	1	10%
4	6.3 mg/dL	1	10%
5	6.4 mg/dL	1	10%
	Total	5	50%

The test results using the SPSS statistical T-test (Paired Sample T-Test) are:

1. In the experiment group, t-count value was 3,942 and the value of $\alpha = 0.017$
2. In the control group, t-count value of 0.925 and the value of $\alpha = 0.099$

Decision-making based on the comparison of the value of α, the value of T count and T table

a. If the value of $\alpha > 0.05$ and t-count is smaller than t-table, H0 is accepted and Ha rejected (No effect).
b. If the value of $\alpha < 0.05$ and t-count larger than t-table, H0 is rejected and Ha accepted (There is an effect).

DISCUSSION

In this research, it is found that there is a significant influence in the experimental group which receives Gedi leaf concoction intervention. It can be seen from the analysis of SPSS 16.0, with a value of T-test $p = 0.017$.

According to Misnadiarly (2007), normal uric acid levels in men range from 3.5-7 mg/dL while in women they range from 2.6-6 mg/dL. Average of uric acid levels in the blood depends on age and gender. After puberty, men uric acid levels increase gradually and reach 5.2 mg/dl. In women, uric acid levels are usually low, but then began to increase in the premenopausal age in the range of 4.7 mg/dl or higher.

According to Anjarwati (2010), increased levels of uric acid are caused by several factors such as foods high in purines, age, gender, certain drugs and alcohol. Production of uric acid in the body can be increased by eating foods high in purines such as meat, offal, spinach, beans, scallops, cauliflower, and crab. The food metabolism will form uric acid which ultimately increases the levels of uric acid in the blood.

Based on the data obtained from the experimental group and the control group, the cause of respondents' high uric acid levels is a result of the consumption of foods high in purines such as cassava leaves, spinach, peanuts, spinach, and bacon.

According to Dalimartha (2008), at >50 years old, women experience a decrease in the estrogen, which is useful to help excrete the uric acid through the urine. When estrogen decreases, the removal of uric acid decreases, and consequently uric acid levels increase. Women are more at risk of uric acid after premenopausal.

With this research, it is expected that communities may use Gedi not only as a food-complementary vegetable, but also herbal medicine therapy.

Figure 1. Red Gedi

Figure 2. Selection & Washing Process

Figure 3. Boiling Process

Figure 4. Water Filtration Process

CONCLUSION

It can be concluded that there is a significant effect between the experimental group and the control group, in which the experimental group who consume Gedi leaf decoction decreases their uric acid levels because the leaves contain compounds such as Trimethyl, Hydroxyl and flavonoids that play a role in decreasing the uric acid levels.

ACKNOWLEDGMENTS

REFERENCES

1. Bourdy,G. and A .Walter. *Maternity and Medicinal Plants in Vanuatu I.The Cyle of Reproduction* .J. Ethnopharmacology Anjarwati, W. Tulang Dan Tubuh Kita. Yogyakarta: Getar Hati; 1992.
2. Moris,R. *Plant For A Future : Edibye And Useful Plants For A Health hier Word (online).* 2006.
3. Misnadiarly. Rematik (Asam Urat Hiperurisemia, arthritis Gout). Jakarta: Pustaka Populer onor; 2007.
4. Notoatmodjo. Metodologi Penelitian Kesehatan. Jakarta: Rineka Cipta; 2007.
5. Tim Redaksi Viva Health. Asam Urat, Informasi Lengkap untuk Penderita dan Keluarganya, Jakarta: Gramedia Pustaka Utama; 2007.
6. Dalimartha, D. Resep Tumbuhan Obat untuk Asam Urat Edisi Revisi. Jakarta: Penebar swadaya; 2008.
7. Kee, Joyce LeFever. *Pedoman Pemeriksaan Laboratorium dan Diagnostic.* Jakarta: Edisi 6 Cetakan I EGC; 2008.
8. Khomsan A.S. Halinawati. *Terapi jus untuk rematik dan asam urat.* Jakarta: Cetakan V Puspa Swara; 2008.
9. Purwantoro,R.S. Prospek engembangan keragaman Tanaman Koloeksi Kebun Raya bogor Untuk menunjang Ketahanan Pangan.Makalah Pusat Konservasi Tumbuhan Kebun Raya Bogor. 2009.

http//ub.ac.id/PDFFILES/BSS 319.pdf (accessed 2 mei 2016).

10. Edward,Elizabeth J,. Buku saku Patofisiologi. Jakarta: Edisi 3 EGC; 2009.

11. Jain, P. S., S. B. Bari. Anti-inflammatory Activity of *Abelmoschus manihot* Extracts International Journal of Pharmacology. 2010;6(4):505-509.

12. Alifiasari, D. Komplikasi asam urat. 2010. http://e-bookspdf.org (accessed 2 mei 2016).

13. Damayanti,D. *Mencegah dan Mengobati Asam Urat*. Yogyakarta: Araska; 2012.

14. Hidayat, A.Aziz Alimul. Metode Penelitian Kesehatan Paradigma Kuantitatif. Surabaya: Health Books Publishing; 2010.

15. Mamahit, L dan N. H. Soekamto. Satu Senyawa Asam Organik Yang Diisolasi dari Daun Gedi (*Abelmoschus Manihot* L. Medik) Asal Sulawesi Utara. *Chem. Prog.* (Wikipedia, 2009; Plantamour, 2010).

16. Sari, M. Sehat dan Bugar tanpa asam urat. Yogyakarta: Araska Publisher; 2010.

17. Sugiyono. Metode Penelitian Pendidikan. Bandung: Alfabeta; 2010.

18. Plantamour (Situs Dunia Tumbuhan). Informasi Spesies Daun gedi (*Abelmoschus manihot L*) 2010. http://plantamour.com/ index.php?/Planta . http.www//Riset Kesehatan Dasar, di kutip 15 Mei , 2014. www.litbang.depkes.go.id/sites/download/lapnas Riskesdas 2014.pdf (accessed 2 mei 2016).

19. Lingga, L. Bebas Penyakit Asam Urat Tanpa obat. Jakarta: Agro Media Pustaka; 2012.

20. Junadi,I. Rematik dan Asan Urat. Jakarta: PT Bhuana Ilmu Populer; 2012.

21. Ni Putu Rahayu dkk. Eksrak Daun Sirsak (Annona muricata L) sebagai antioksidan pada penurunan kadar asam urat pada tikus Wistar. Jurnal Kimia. 2012. (6).

22. Junadi,I. Rematik dan Asan Urat. Jakarta: PT Bhuana Ilmu Populer; 2013.

23. South et.al. Evaluasi Kandungan total Polifenol dan isolasi senyawa flavonoid pada daun Gedi Merah (Abelmoschus Manihot L). Chemistry Progress; 2013.

24. Noor Helmi, Zairin. Buku Ajar Gangguan Muskuloskeletal. Jakarta: Salemba Medika; 2013.

25. Smart Aqila. Rematik dan Asam Urat. Pengobatan Sampai Sembuh Total. Jogjakarta: A Plus Books; 2015.

26. Saraswati,S. Buku Diet Sehat Untuk Penyakit Asam Urat,diabetes,hipertensi dan stroke. Jogjakarta: 2015.

27. Mapanawang A. Riset Di Bidang Kesehatan. Tobelo: Medika Mandiri Halmahera; 2016.

****