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RESEARCH ARTICLE

EVALUATION OF THE IMPACT OF NUTRITIONAL EDUCATION ON THE EVOLUTION OF GOUT: THE CASE OF THE "LA PROVIDENCE" CLINIC IN N'DJAMENA

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Abstract

Gout is a fairly common problem in middle-aged men. Despite the high prevalence of the condition, there are few clinical studies of gout patients in Chad. The management of gout is based on the treatment and prevention of very painful gout attacks. In general, treatments for gout attacks are not based on rigorous scientific data. The present study aims to contribute to the nutritional management of gout. We used socio-demographic and cultural characteristics of gout patients, patients' knowledge of the aetiology and management of their disease before assessing the effect of nutrition education on disease progression. A total of 50 patients suffering from gout and attending the clinic "La providence" in N'Djamena were included in the study from 20 June to 26 July 2016. Our study shows that environmental factors play a significant role in the occurrence of gout: overweight, regular alcohol consumption, diet rich in purines (red meat and offal in particular). The effectiveness of dietary advice such as avoiding purine-rich foods and increasing fluid intake (2 to 3 litres of water per day) has been demonstrated, but these measures are recommended. It is advisable to limit alcohol consumption and to reduce body weight.

Introduction:-

Gout is the most common type of inflammatory arthritis and has been known for thousands of years (Bardin and Richette, 2011). The most clinical characteristic manifestation is acute monoarthritis of the big toe. This is associated, albeit inconstantly, with an increase in the circulating level of uric acid (hyperuricemia). It progresses untreated to the deposition of uric acid in several body sites with a predilection for the joints (gouty arthritis), skin (tophus) and kidneys (uratic nephropathy) (Bardin and Richette, 2011).

Recently, analysis of the National Health and Nutrition Examination Survey (NHANES) estimated the prevalence of gout in the United States in the population aged 20 years to be over 3.9% in 2007-2008, compared to an estimated 2.7% in the same database in 1988-1994. This suggests that the prevalence of the disease continues to increase in the United States. Gout is more common in blacks, which may be related to a higher incidence of high blood pressure,

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or lower incomes, affecting diet and the incidence of obesity (Bardin and Richette, 2011; Chalès, 2011). The prevalence of this disease has almost doubled in the last 30 years, probably due to the increase in the general standard of living in our population (richer diet). It is estimated to be 1-5% in men and 2% in women over 40. It is noted that 15% of patients with hyperuricaemia will develop gout, with these figures increasing to 30-50% if the uricaemia is above 100mg/L (Bardin and Richette, 2011; Chalès, 2011). It increases with age and with degrees of hyperuricaemia and with gender. If left untreated, it progresses to multi-site damage and joint destruction and potentially fatal end-stage renal disease, for which a dietary regimen is required.

Several factors influence the manifestation of the disease. Diet plays a major role. The role of diet has long been recognised in the pathogenesis of gout. Several studies have recently re-examined the relationship of gout with diet (Bardin and Richette, 2011; Chalès, 2011). These risk factors should not be ignored by gout patients. Their personal involvement in the nutritional management of gout is a considerable asset on this regard. In general, treatments for gout attacks are not based on rigorous scientific data. During a treatment, the effectiveness of the medication, the side effects and diet must be taken into account for each individual attack and for each individual patient (Bardin and Richette, 2011; Chalès, 2011).This is the contribution of the nutritional management of gout is important to aset. Objectively, we want to evaluate the impact of nutritional education on the evolution of gout in patients at the "La providence" clinic in N'djamena. More specifically, we want to

1. Determine the socio-demographic and cultural characteristics of patients suffering from gout;
2. Evaluate the knowledge of patients on the etiology and management of their disease;
3. Conduct nutritional education with gout patients;
4. To assess the effect of nutrition education on disease progression.

After presenting the review of the literature on gout, we will highlight the materials and method, the results and discussion before concluding and proposing perspectives.

Methodology:-

1. **Setting and location of the study**

The study took place at the medical-surgical clinic "La PROVIDENCE" in N'Djamena.

2. **Period**

The study were conducted from 20 June to 26 July 2016.

3. **Study population**

all gout patients presenting to the providence clinic during the study period are used for this study. Before the start of the interviews, a consent form is submitted to each patient.

4. **Type of study**

The study was conducted on the basis of a prospective and retrospective analytical method:

- Prospective method: i.e., the work was done in the field, by exchanging with patients during consultation and those who are hospitalised at the research site (clinic)
- Retrospective method: consists of working on the patients' files at the site without having direct contact with them but through their telephone contact.

Nutritional education

Nutritional education includes both diet and aspects of physical activity as a factor of well-being and health. As gout is a relapsing disease, we advise patients to reduce or even avoid the consumption of certain foods such as: alcohol (especially beer), meat broths, fatty fish, chicken, offal (liver, brains, kidneys, tongue, heart....), foods rich in purine, vegetables that smell strong when cooked such as cabbage, asparagus, spinach, sorrel, champions; oleaginous fruits: dates, dried figs, dried fruits (nuts, grapes...) (Young et al.,2011; Guerra et al., 2014). All aperitifs (champagne, acidic white wines, sweet wines, liqueurs, spirits, strong beer, red wines rich in tannins), diuretic drugs (furosemides), consumption of animal proteins in large quantities are prohibited. Patients should avoid a sedentary lifestyle (idleness), as this reduces the body's expenditure but aggravates the effects of overeating) (Young et al., 2011; Guerra et al., 2014). They should seek an active life in the open air by doing moderate physical exercises without overexerting their muscles. The consumption of water is to be preferred, at least 2 to 3 litres to be distributed throughout the day. Preferably it is good to drink lemon juice for its alkalising and diluting role of the uric acid crystals which are in the blood and to be eliminated by the urinary way. In the absence of diabetes, to ensure abundant diuresis, which is essential to eliminate excess urates, increase the consumption of glucose (sugar) (Young et al., 2011; Guerra et al., 2014).

Materials:-

These are the documents and instruments needed to carry out this study:

1. A questionnaire;
2. A GET memory blood pressure monitor
3. A balance of the name Axions medical UK Chinese brand RGZ 160;
4. A Spectrophotometer of French brand
5. Access software;
6. The patient's medical file.

Data collection and analysis

The data were recorded on survey forms (see model in the appendix) and then analysed using Access.

Results And Discussion:-

The analyses were carried out largely on pivot tables, while allowing for an in-depth analysis of the details. The processed data were collected on a sample of 50 people, given the time constraints and the difficulty of obtaining the availability of patients for the surveys. This data analysis work is a statistical work and comparison of results.

Socio-demographic and socio-cultural characteristics

a) Distribution by sex, age and marital status

Table 1 shows that in the sample studied, there were 36 men and 14 women suffering from gout. This represents a rate of 72% for men and 28% for women.

The average age is 40 years for women and 48 years for men. Women have a minimum age of 28 and a maximum age of 65. Men have a minimum age of 28 and a maximum age of 98. The minimum age for men and women is equal to 28 years for both.

The surprising observation regarding marital status is that married people are more affected than single, widowed and divorced people. Among women there are 10 married and 4 single. Among men there are 32 married, 3 divorced and 1 widowed.

Table 1:- Distribution by gender, age and marital status

The pie chart below illustrates the standard gender gap for gout.

Sex	Number	Average Age	Max Age	Min Age
Female	**14**	**40**	**65**	**28**
Single	4	28	28	28
Married	10	45	65	38
Male	**36**	**48**	**98**	**28**
Divorced	3	36	36	36
Married	32	48	98	28
Widowed	1	98	98	98
Overall average	**50**	**46**	**98**	**28**

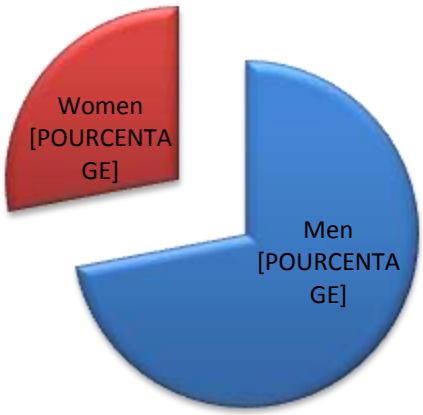

Figure 1:- Gender distribution.

Figure 2 summarises the rates of gout by gender and marital status. Married men have a rate of 64%, divorced men have a rate of 6% and widowed men have a rate of 2%. Married women have a rate of 20% and single women have a rate of 8%. Fewer women than men suffer from gout in the sample studied.

According to epidemiological studies, the risk of developing gout is 3 to 6 times higher in men than in women. Physiologically, this is due to the uricosuric effect of oestrogen, which leads to lower uricemia levels in women than in men until the menopause.The results of this study are similar to those of Suzanne P. et al. (1996); in their study, they showed that men have more gout than women in the Paris region.

These results are in agreement with the work of Bardin and Richette (2011). The prevalence of gout increases with age, which is probably due to the slow formation of pathogenic crystalline monosodium urate deposits. The risk of gout therefore depends on the degree and duration of hyperuricaemia. In the 1996 Nhanes, the prevalence was found to be 1.8% between the ages of 18 and 44 years, 22% between the ages of 18 and 44 years and 11% between the ages of 18 and 44 years. 1000 between the ages of 18 and 44, 22.4 p. 1000 between 45 and 64 years of age, and 30.8 p. 1000 in the population aged 65 and over. In all age groups, it was higher in men than in women, although this male predominance diminished with age. Due to the uricosuric effect of estrogen, adult women have lower uricemia than men until the menopause (Hak et al., 2008), when women's uricemia returns to male values.

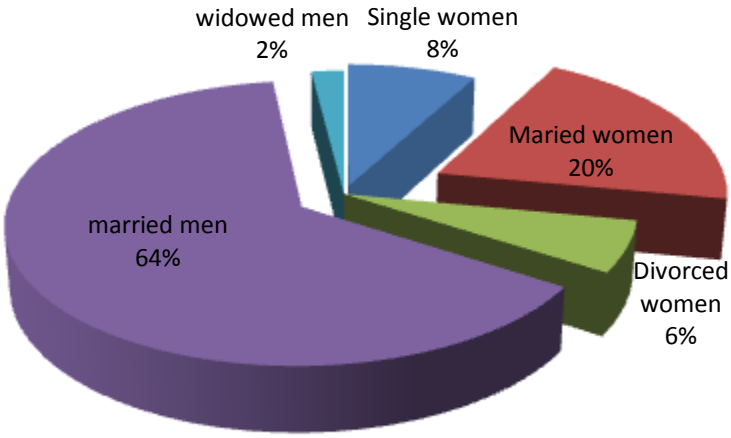

Figure 2:- Distribution by marital status.

b) Educational level

Figure 3 summarises the men and women according to their level of education. There are 4% of women and 4% of men who have had no schooling. At primary level, 4% of women and 2% of men have no education. At secondary level the rate is 16% for men and 2% for women. At the higher level, 50% are men and 18% are women.

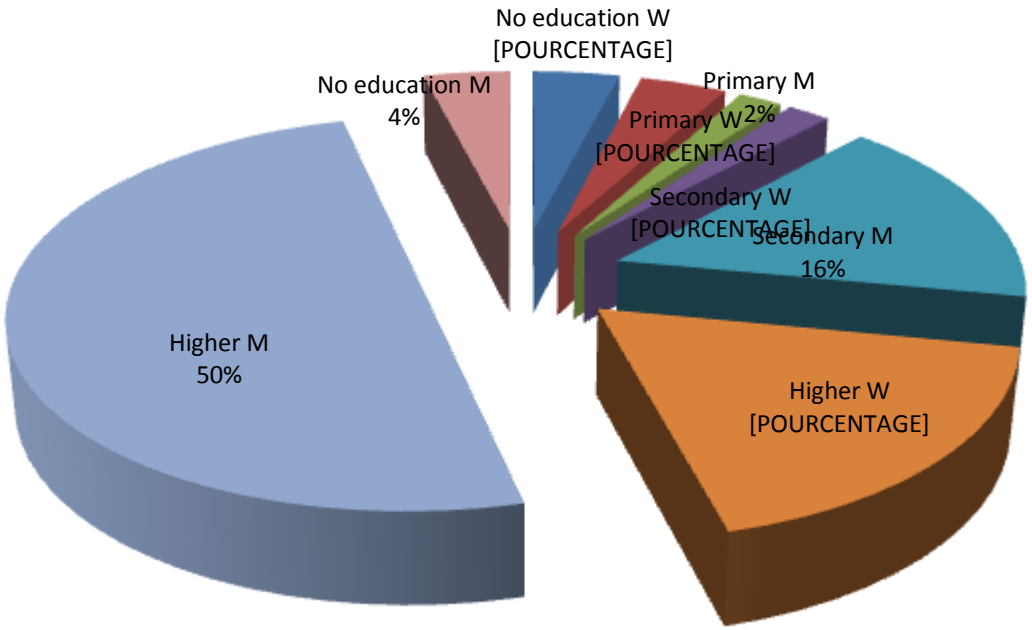

Figure 3:- Distribution by level of education.

Patients' knowledge of the etiology and management of their disease

a) Diet and sedentary lifestyle

Table 2:- Distribution according to diet or sedentary lifestyle as cause.

Cause	Patients
Diet	37
Sedentary lifestyle	1
No answer	12
Total	**50**

The majority of patients stated that diet is the main cause of gouty disease. Table 2 shows that 37 people or 74% (see Figure 4) confirmed that it was diet. A number of 12 people or 24% gave no answer, and only one person mentioned a sedentary lifestyle, which represents 2% of the population.

Dietary habits seem to have a negative impact on uric acid levels. Among the sources of exogenous uric acid, certain foods contain large amounts of purine, which must be metabolised and which will lead to a higher production of uric acid. This result underlines the major role of diet as a determining factor in the development of gout. This result is underline with the data obtained on the causes of gout, 74% of which are the result of diet. These results are in line with the observations of Ea (2011). These results allow us to formulate recommendations and guidelines with regard to an unhealthy diet, which is a source of implantation and proliferation of the disease.

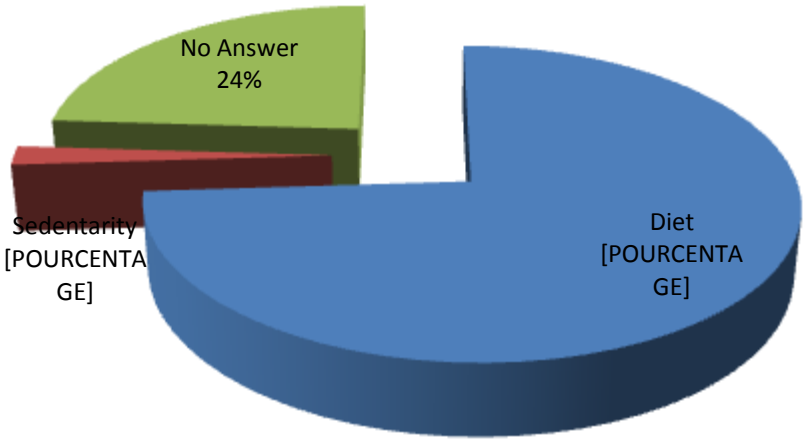

Figure 4:- Causes of gout by physical inactivity or diet.

b) Knowledge of foods that cause gout

Table 3:- Distribution of foods that cause gout.

Food	**Patients**
Alcohol	1
Alcohol Meat Slaughter	3
Meat	34
No response	12
Total	**50**

Table 3 shows that 34 people say that meat is responsible for gout. 12 people gave no answer on this subject. 3 people blamed the trio slaughter-meat-alcohol. 1 person blamed alcohol.

Table 3 highlights the local population's knowledge of the main foods responsible for the disease, gout. Jemaa et al. (2021) suggest that improving patients' knowledge and beliefs about gout requires more attention to the time spent informing patients and explaining the treatment and its side effects.

Graph 5 breaks down the rates as follows: 68% for meat, 24% no opinion, 6% for alcohol-slaughter-meat and 2% for alcohol only.

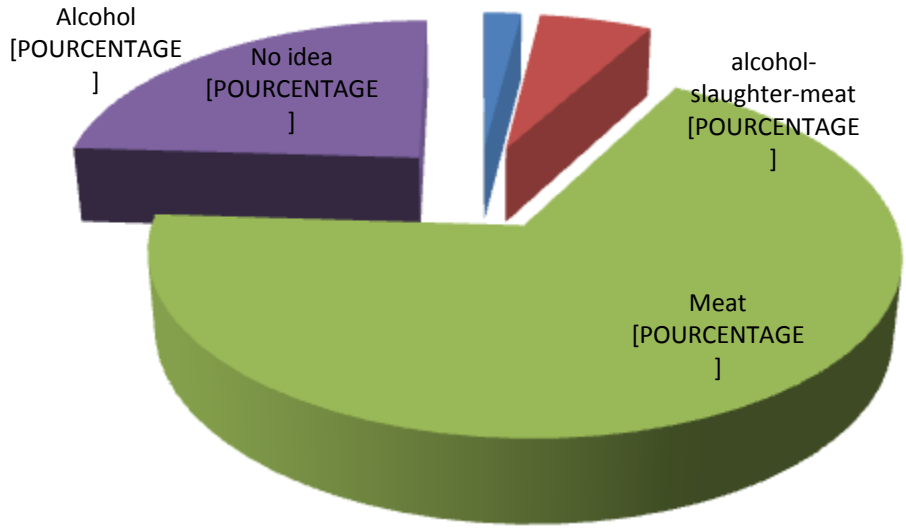

Figure 5:- Foods causing gout.

The low percentage of response related to alcohol is due to lack of awareness among patients. Heavy drinkers have an apparent predisposition to an attack of gout, but the type of alcoholic beverage may play a major role.

For example, beer is rich in purine, particularly guanotine. The more beer a patient consumes, the higher the risk of hyperuricaemia and gout.

Similarly, the consumption of meat and offal increases the occurrence of gout in all people. This can be explained by the fact that the foods are proteinous in nature and therefore rich in purine. Lekpa F. K et al(2016), in a study done on gout patients in Douala, Cameroon found a similar result to this study.According to these results, meat consumption appears to be the primary culprit in gout. The role of diet has long been recognised in the pathogenesis of gout. Several studies have recently re-examined the relationship of gout to diet. In a 12-year prospective study of 47,150 male health professionals without gout at baseline, the risk of developing gout was correlated with high intakes of animal purines (meat or seafood) and consumption of beer and spirits (Bardin and Richette, 2011).

c) Foods to avoid during a gout attack

Table 4:- Distribution of foods to be avoided during a gout attack.

Foods	**Patients**
Alcohol	1
Alcohol Meat Slaughter	3
Meat	34
No response	12
Total	**50**

It is very remarkable that the test results are identical for item (table) 3 and 4. In this respect, the patients faithfully reported the foods considered to cause gout in the list of foods to be avoided during the gout attack. Table 3 and graph 6 show the same values as table 4 and graph 5. 34 people, i.e., a rate of 68% for meat, 12 people, i.e., a rate of 24% for no response (people who did not give an opinion), 3 people, i.e., a rate of 6% for the trio of alcohol, meat and meat and finally 1 person, i.e., a rate of 2% for alcohol.

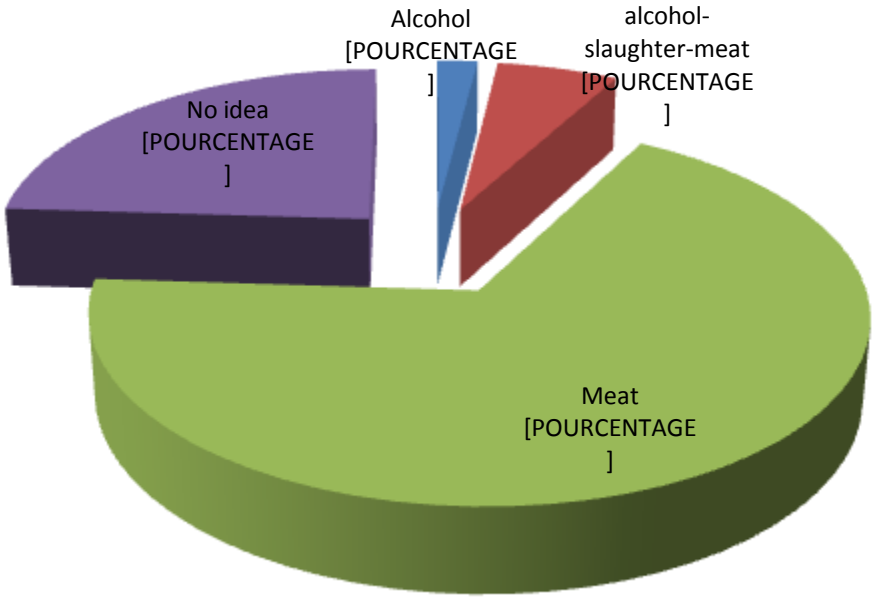

Figure 6:- Foods to avoid.

These results show us the value of nutrition education in gout patients, as the change in dietary behaviour recommended for gout is an accompanying measure and often also the focal point of the therapy. However, it is not a substitute for medical care. Its aim is to reduce the concentration and crystallisation of uric acid in the blood.

A study conducted in the United States by Singha (2012) shows that a well-followed nutritional education can decrease the number of gout patients.

d) Preferred food during gout attack.

Table 5:- Distribution of preferred foods during gout attack.

Food	Patients
Fruit and vegetables	17
Fish	17
Chicken	4
No response	12
Total	**50**

Concerning the foods to be favoured during a gout attack, Table 5 and Graph 7 give a total of 17 people for fruit and vegetables, i.e., a rate of 34%; a total of 17 people for fish, i.e., also a rate of 34%; a total of 12 people who did not give their opinion, i.e., a rate of 24%; a total of 4 people for chicken, i.e., a rate of 8%.

Recommendations can be made for better management of the disease. Certain foods can trigger a gout attack, even though they do not in themselves produce uric acid, through complex mechanisms: some foods are specific to each patient (chocolate, mushrooms, cabbage, asparagus, sorrel, spinach, rhubarb, dried figs, etc.) (Létard et al., 2009).

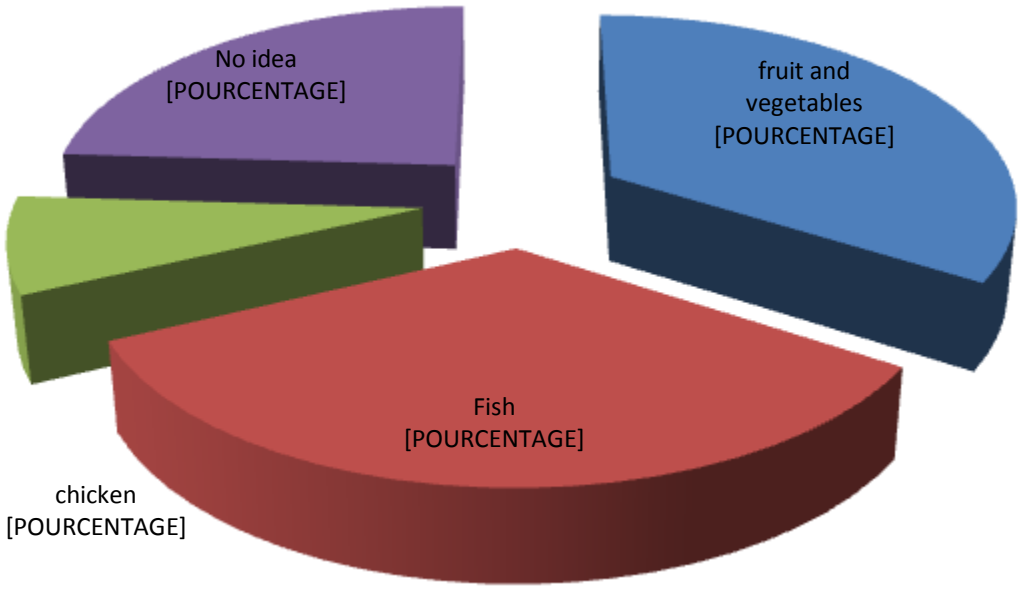

Figure 7:- Preferred foods.

Disease progression after nutrition education

a) Uric acid level

In all other organisms, uric acid is further processed before excretion(Fig. 8). Mammals other than primates oxidize it to their excretory product, allantoin, in a reaction catalyzed by the Cu-containing enzyme urate oxidase. Afurther degradation product, allantoic acid, is excreted by teleost (bony) fi sh.Cartilaginous fi sh and amphibia further degrade allantoic acid to urea prior toexcretion. Finally, marine invertebrates decompose urea to NH+4.

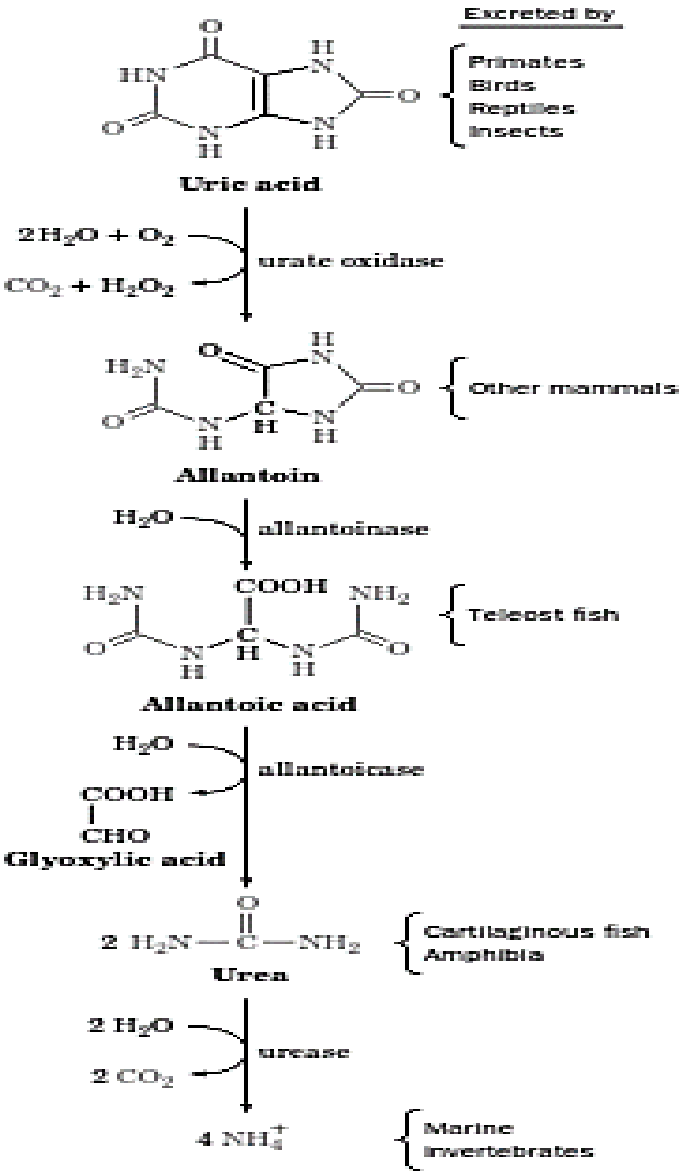

Figure 8:- Degradation of uric acid to ammonia(Voet et al., 2016).

The most prevalent cause of gout is impaired uric acid excretion (although usually for reasons other than lead poisoning). Gout may also result from a number of metabolic insufficiencies, most of which are not well characterized. One well-understood cause is HGPRT deficiency (Lesch–Nyhan Syndrome in severe cases), which leads to excessive uric acid production through PRPP accumulation (Voet et al., 2016).

Table 6:- Mean, minimum, maximum distribution of uric acid in mm/L.

Average	Min	Max
T1 450,6	348	624,7
T2 376,3	202,4	634,3
T3 286,9	240,1	577,1
T4 274,3	252,3	600,6

Table 6 summarises the mean, minimum and maximum values of the four uric acid levels taken from T1 to T4 for both types of treatment combined. In fact, the levels decrease most often from T1 to T4, whether for the average, minimum or maximum value. This decrease would explain the effectiveness of nutritional education for patients.

Graph 8 shows the variation curve of the mean uric acid levels in the patients subjected to this analytical study (Desideri et al., 2014).

Indeed, uric acid is the final product of purine metabolism in humans, who have lost the uricase that allows them to oxidise it into allantoin during evolution (Ea, 2011). Its plasma concentration depends on a balance between its synthesis and elimination which are regulated by genetic and dietary factors. Uric acid is derived from dietary purines, catabolism of cellular nucleic acids and de novo purino synthesis (Ea, 2011). Two thirds of uric acid is eliminated by the kidney and the remaining third by the digestive tract. Renal regulation of urate is complex and depends on numerous organic anion transporters (OATs) such as URAT1, the Glut9 transporter, the ATP-binding cassette family G2 (ABCG2) transporter and the sodium phosphate co-transporters (NTPs) 1 and 4. Polymorphisms in the genes coding for these different proteins have been associated with gout. Gout results from chronic hyperuricaemia (Ea, 2011).

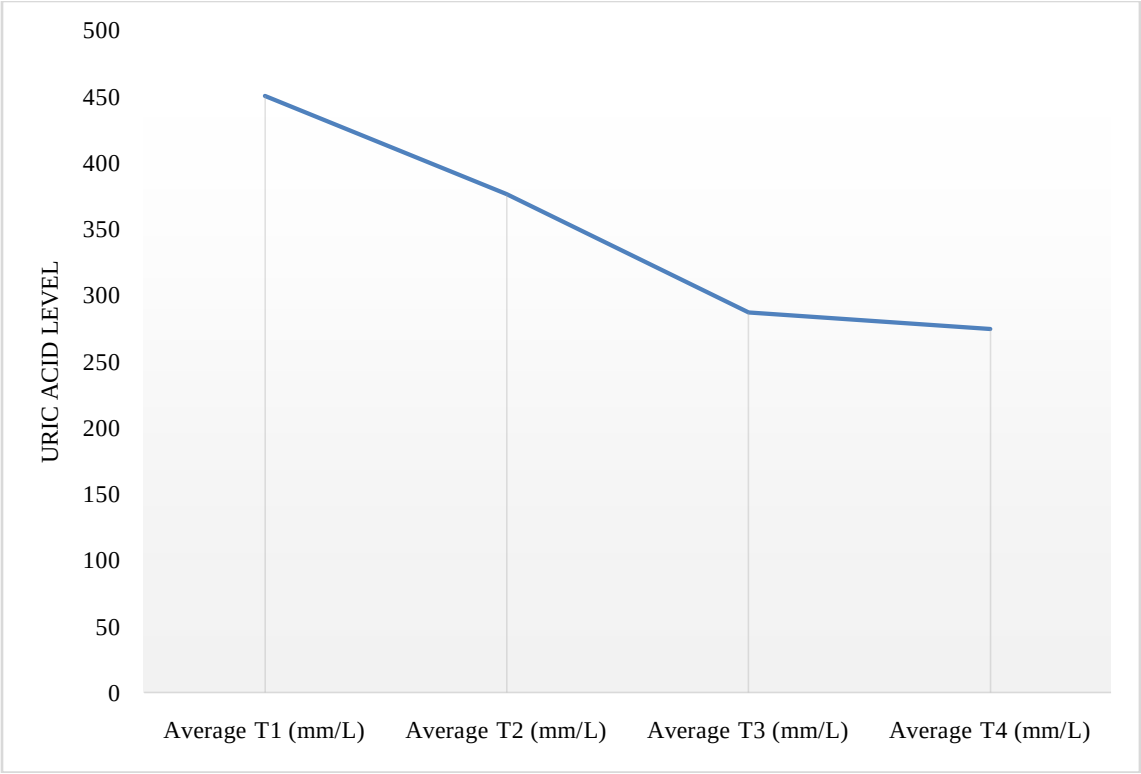

Figure 9:- Variation curve of the mean T1-T4.

The further analysis at the level of treatment type will focus on the comparison of the respective rate curve for the therapeutic treatment and the dietary regime.

b) Type of treatment used

Table 7:- Distribution according to whether patients received nutrition education or not.

Nutritional education	**Patients**
No	4
Yes	46
Total	**50**

The majority of patients had combined a diet with drug treatment. A total of 46 people had completed nutrition education and 4 had not. That is, 92% of the total number of patients completed nutrition education and 8% of the total number of patients did not complete nutrition education.

c) Comparison of the average uric acid levels of patients who received nutrition education with those who did not

Table 8:- Comparative distribution of uric acid levels of patients who received nutrition education and those who did not.

Nutritional education	T1	T2	T3	T4
Yes	434,05	296,07	234,43	251,50
No	452,05	383,27	379,02	377,56
Average	**450,61**	**376,30**	**306,72**	**314,53**

Table 8 compares the mean uric acid levels according to whether or not patients received nutrition education in combination with drug treatment. Unfortunately, this study did not have information on patients who received conventional treatment. This would have allowed a comparison of the two types of treatment. The mean values of the uric acid levels show that in both cases the uric acid level falls gradually (Desideri et al., 2014).

Figure 9 shows that the uric acid level curve of the patients who received nutrition education falls faster than those who did not. In this analytical study, the statistics show that nutrition education is effective in lowering uric acid levels.

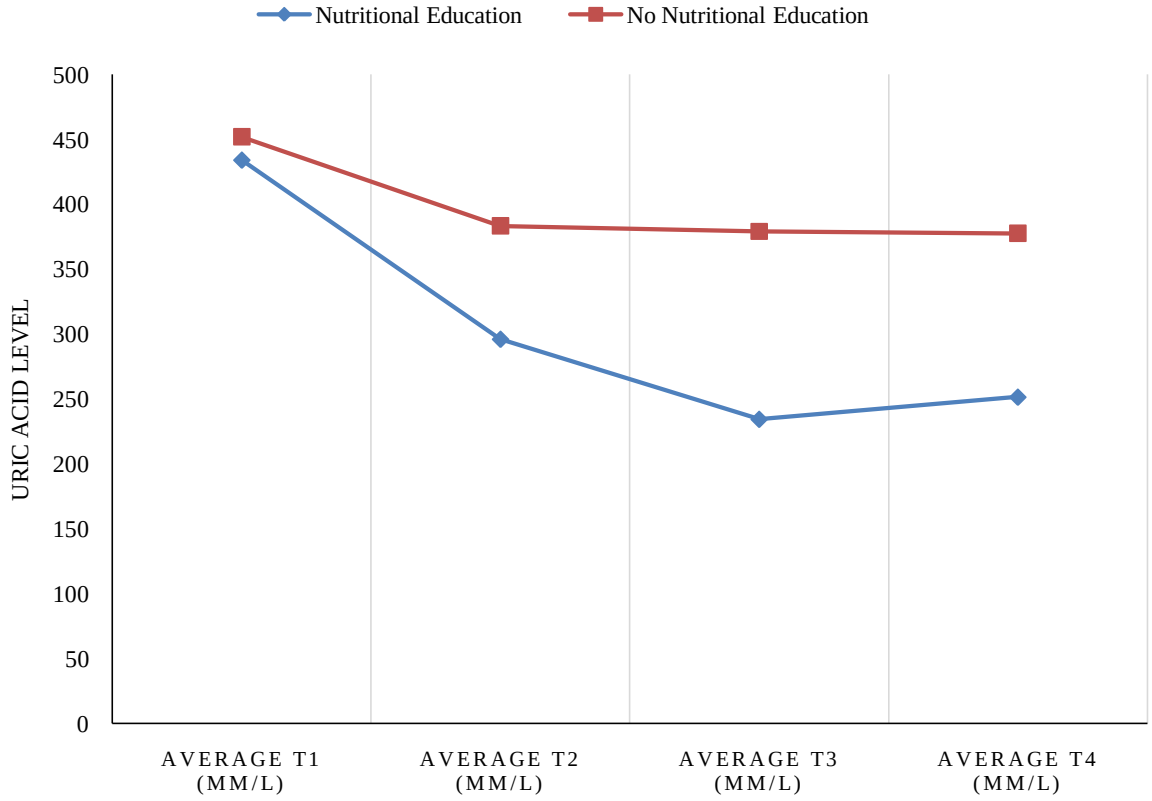

Figure 10:- Uric acid levels evolution curves.

In fact, uric acid is the end product of the catabolism of purine bases (Table 26.1), which are molecules present in nucleic acids and which therefore participate, alongside pyrimidine bases, in the functions of DNA duplication and protein biosynthesis. In most animal species (except primates), uric acid is itself transformed into allantoin, then into urea, a highly soluble product that is easily eliminated in the urine, under the effect of a final enzyme: uricase or urate oxidase. This uric acid is dissolved in body fluids, including blood, in the form of a salt (sodium urate), which is much more soluble at the body's pH than the acid itself. The amount of sodium urate in the body is about 1200 mg in men and 600 mg in women. About half of this is renewed every day (miscible pool of uric acid), distributed 1/5 in plasma and 4/5 in extracellular fluids (Constantin et al., 2018).

Conclusion And Perspectives:-

Gout has been on the rise for several decades in most countries of the world, and particularly in Chad. The causes of this increase are multiple: dietary changes, the increasing use of hyperuricemic drugs such as diuretics and the increase in life expectancy.Gout usually occurs after several years of hyperuricemia. Only a few cases develop into the disease. Currently, it is recommended that patients be managed with simple nutritional education measures. It is associated, albeit inconstantly, with an increase in the circulating level of uric acid (hyperuricemia). Without treatment, it progresses to the deposition of uric acid in several sites of the body, with a predilection for the joints (gouty arthritis), the skin (tophus) and the kidneys (uratic nephropathy), leading at most to disabling joint destruction and potentially fatal end-stage renal failure.

Generally, treatments for gout attacks are not based on rigorous scientific data. When choosing a treatment, not only the effectiveness of the drug but also its side effects must be considered for each individual attack and for each individual patient.The exact cause of gout remains unknown. A genetic component is likely due to the frequency of familial forms and the prevalence in males, but environmental factors play a significant role in its occurrence: overweight, regular consumption of alcohol, diet rich in purines (red meat and offal in particular). However, not all causes of hyperuricaemia are causes of gout (there is one subject with gout for every ten with hyperuricaemia in the population).The change in eating and drinking behaviour recommended for gout is an accompanying measure and often also the focal point of therapy, but it is not a substitute for medical care. Its primary aim is to reduce the concentration and crystallisation of uric acid in the blood. The amount of fluid to be drunk daily should be at least 2 litres to help eliminate uric acid via the kidneys and urine. Drink preferably unsweetened and alcohol-free drinks.

Avoid as much as possible the consumption of drinks containing fructose (lemonades), as too much fructose seems to increase the concentration of uric acid in the blood. Alcoholic beverages, especially beer and spirits, should be completely banned. Alcohol increases the concentration of uric acid in the blood because it increases the body's own production and at the same time impedes its elimination. In addition to alcohol, alcohol-free beer also contains purines. This is also the case with alcohol-free beer, which is therefore not a sensible alternative. Coffee with or without caffeine seems to have a positive effect on uric acid levels, but not so for other caffeine-containing drinks such as tea. In relation to gout, it is therefore not necessary to limit coffee consumption. Consumption of meat, offal, fish and seafood increases uric acid levels and should therefore be kept to a minimum. Vitamin C has been identified as a protective factor against hyperuricaemia and metabolic studies have shown that this vitamin has a uricosuric effect. In the light of this research, we propose:

1. To know more about the main etiologies associated with gout in the world, in particular in Chad;
2. To know the natural plants, present in Chad that can treat gout;
3. To raise awareness among the Chadian population about the consumption of meat, offal, fish and seafood.

Références:-

1. Bardin, T., Richette, P. (2011) : Épidémiologie et génétique de la goutte. La Presse Médicale. 40(9):830-835.
2. Bardin, T., Richette, P. (2010) : Gout,The Lancet,Vol. 375, Issue 9711,Pages 318-328, ISSN 0140-6736,
3. Becker, M.A., Schumacher H.R., Wortmann, R.L., Patricia, M.D., MacDonald, B.S.N., Denise Eustace, N.P., William, B. A., Palo, M.S., Janet Streit, M.S., and Nancy Joseph-Ridge, M.D. (2005): Febuxostat compared with allopurinol in patients with hyperuricemia and gout. N Engl J Med.,353: 2450-2461
4. Campion, E.W., Glynn, R.J., DeLabry, L.O. (1987): Asymptomatic hyperuricemia. Risks and consequences in the normative aging study. Am J Med., 82: 421–426.
5. Chalès, G. (2011) : De l'hyperuricémie à la goutte : épidémiologie de la goutte. Revue du rhumatisme. 78: 109-115.
6. Choi, H.K., Atkinson, K., Karlson, E.W., Willet, W.C., Curhan, G.(2004) : Alcohol intake and risk of incident gout in men: a prospective study. Lancet., 63: 1277-1281.
7. Choi, H.K., Curhan, G. (2007): Independent impact of gout on mortality and risk for coronary heart disease. Circulation., 116: 894–900.
8. Choi, H.K., Curhan, G. (2008): Soft drinks, fructose consumption, and the risk of gout inmen: prospective cohort study. BMJ., 336: 309–312.
9. Choi, H.K., Ford, E.S. (2007): Prevalence of the metabolic syndrome in individuals with hyperuricemia. Am J Med., 120: 442–447.

10. Choi, H.K., Liu, S., Curhan, G. (2005): Intake of purine-rich foods, protein, and dairy products and relationship to serum levels of uric acid: the Third National Health and Nutrition Examination Survey. Arthritis Rheum., 52: 283–289.
11. Choi, H.K., Mount, D.B., Reginato, A.M. (2005): Pathogenesis of gout. Ann Intern Med., 143: 499–516.
12. Choi, H.K., Atkinson, K., Karlson, E.W., Willet, W.C., Curhan, G. (2004) :Purine-rich foods, dairy and protein intake, and the risk of gout in men. N Engl J Med., 350: 1093-1103.
13. Coffer, (2002) : La goutte, in Rhumatologie. Masson, Paris., 228.236.
14. Constantin, A., Cantagrel, A., Laroche, M., Mazières, B. (2018) : Rhumatologie pour le praticien. Elsevier Health Sciences. 712 pages
15. Desideri, G., Castaldo, G., Lombardi, A., Mussap, M., Testa, A., Pontremoli, R., Punzi, L., Borghi, C. (2014): Is it time to revise the normal range of serum uric acid levels. Eur Rev Med Pharmacol Sci.,18(9):1295-1306.
16. Ea, H.K.(2011) : De l'hyperuricémie à la goutte: physiopathologie. Revue du rhumatisme., 78:S103-108.
17. Guerra, P.H., Nobre, M.R., da Silveira, J.A., Taddei, J.A. (2014) : School-based physical activity and nutritional education interventions on body mass index: A meta-analysis of randomised community trials—Project PANE. Preventive medicine., 61:81-89.
18. Hak, A.E., Choi, H.K. (2008): Menopause, postmenopausal hormone use and serum uric acid levels in US women˜the Third National Health and Nutrition Examination Survey. Arthritis Res Ther., 10:R116.
19. Janssens, H.J., Lucassen, P.L.B.J., Van de Laar, F.A., Janssen, M., Vand de Lisdonk, E.H. (2008) : Systemic corticosteroids for acute gout (Review). Cochrane Database Syst Rev.,: CD005521.
20. Jemaa, S.B., Benchehida, R., Feki, A., Mnif, I., Akrout, R., Ezzedine, M., Kallel, M.H., Fourati, H., Baklouti, S. (2021) : Connaissances et croyances de la goutte chez des patients goutteux: à propos de 79 cas. Revue du Rhumatisme., 88:A41.
21. Lekpa F. K., Mbiatat H.D.G and Luma N. H (2016). AB0809 Efficacy and Safety of A Personalized Nutritional Diet in Gouty Patients in Cameroon: A Pilot Study: Table 1. Annals of the Rheumatic Diseases 75(Suppl 2):1180.3-1181
22. Létard, J.C., Ludot, T., Costil, V., Tarrerias, A.L., Bonnaud, G., Boustière, C., Canard, J.M., Constantini, D., Coulom, P, Devulder, F., Gay, G. (2009) : Fiche de recommandations alimentaires.
23. Liu-Bryan, R., Scott, P., Sydlaske, A. (2005): Innate immunity conferred by Toll-like receptors 2 and 4 and myeloid differentiation factor 88 expression is pivotal to monosodium urate monohydrate crystal-induced inflammation. Arthritis Rheum.52 (9): 2936-2946.
24. Richette, P., Briere, C., Hoenen-Clavert, V. (2007) :Rasburicase for tophaceous gout not treatable with allopurinol : an exploratory study. J Rheumatol., 34: 2093-2098.
25. Roddy, E., Mallen, C.D., Hider, S.L., Jordan, K.P. (2010): Prescription and comorbidity screening following consultation for acute gout in primary care. Rheumatology., 49: 105–111.
26. Roddy, E. (2008): Hyperuricemia, gout, and lifestyle factors. J Rheumatol., 35:1689–1691.
27. Suzanne P. Murphy, Maradee A. Davis, John M. Neuhaus, David Lein (1996). Dietary quality and survival among middle-aged and older adults in the NHANES I epidemiologic follow-up study, Nutrition Research, 16, Issue 10: 1641-1650.
28. Terkeltaub, R., Zelman, D., Scavulli, J. (2009) :Gout Study Group: update on hyperuricemia and gout. Joint Bone Spine., 76: 444–446.
29. Terkeltaub, R.A. (2009): Colchicine Update 2008.Semin ArthritisRheum., 38:411–419.
30. Voet, D., Voet, J.G., Pratt, C.W. (2016) : Fundamentals of biochemistry: life at the molecular level. John Wiley & Sons.
31. Young, K., Bunn, F., Trivedi, D., Dickinson, A. (2011): Nutritional education for community dwelling older people: a systematic review of randomised controlled trials. International journal of nursing studies., 48(6):751-780.
32. World Health Organization. (2003): Adherence to long term therapies: evidence for action. Genève: WHO Library Cataloguing-in-Publication Data., p.11.
33. Zhang, W., Doherty, M., Bardin, T., Pascual, E., Barskova,V., Conaghan, P.Gerster, J., Jacobs, J., Leeb, B., Lioté, F., Mccarthy, G., Netter, P., Nuki, G., Perez-Ruiz, F., Pignone, A., Pimentão, J., Punzi, L., Roddy, E., Uhlig, T., Zimmermann- Gòrska, I. (2006): EULAR evidence based recommendations for gout. Part II: Management. Report of atask force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics (ESCISIT).Ann Rheum Dis., 65: 1312–1324.