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A case study: Obesity and hyperuricemia. A two-pronged program, targeted dietary intervention and intermittent fasting, significantly reduce, body weight and hyperuricemia

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Abstract

A 46-year-old woman of a height of 152 cms with body weight of 78 kg and a BMI of 33.8 g/m2 was seeking medical treatment for elevated levels of serum uric acid. The patient had anxiety issues when she reached out for treatment. She suffered from swelling over her bilateral ankles and difficulty in walking and chronic pain in legs in standing & resting position. Over the years she had sought multiple treatments for various ailments viz. asthma, pneumonia, and anxiety disorder. After examination and diagnosis, the decision was made to begin a two-pronged program by way of dietary intervention and intermittent fasting. The program was closely monitored by telephonic interaction daily for first week followed by follow up on every 3rd day for the balance period of treatment. After 5 weeks of treatment the woman has lost 8 kg of bodyweight and reduction in the serum uric acid levels by 2.1 mg/ dl. In conclusion, it is possible to successfully treat severe obesity and elevated levels of uric acid and return a patient to acceptable BMI with the combination of dietary intervention and intermittent fasting.

Keywords: Obesity, hyperuricemia, uric acid, Hb1AC, intermittent fasting, insulin resistance

Introduction

Obesity and the hyperuricemia are linked together [24]. When an individual gets severely obese, insulin resistance, hypertension and increased abdominal circumference follow as a natural cause due to the excess fat in the body. Obesity and hyperuricemia have been extensively researched and today clinical evidence implicates intra-abdominal adiposity as a powerful driving force for elevated serum uric acid risk [2,25]. This association appears to arise directly, via secretion of adipokines, and indirectly, through promotion of insulin resistance.

The hyperuricemia is defined as serum uric acid ≥7 mg/dL for men and ≥6.0 mg/dL for women [5], many factors can influence the concentrations of serum uric acid, e.g.: diet, obesity, and metabolic syndrome [6-8]. Hyperuricemia increases in 16% all causes of mortality and 39% of total cardiovascular disease [9].

Gout is an inflammatory arthritis caused by deposition of monosodium urate crystals within synovial joints. High plasma uric acid (UA) is a precipitating factor for gout and renal calculi as well as a strong risk factor for Metabolic Syndrome and cardiovascular disease. The main causes for higher plasma UA are either lower excretion, higher synthesis, or both [1-4].

Current recommendations support aggressive medical therapy to treat gout, whereas dietary intervention has become less emphasized.

This study argues for the absolute importance of dietary intervention and intermittent fasting in hyperuricemia and proves why this counselling may impact the long-term well-being of a patient [10,26].

Pharmacologic therapies to prevent recurrent flares have primarily focused on reduction of uric acid levels. However, urate-lowering therapy is not without adverse effects. In contrast, dietary interventions may be an attractive alternative to prescription drug therapy, particularly among those with mild gout, a population representing more than 3 million adults in the US.

Dietary approaches to reducing uric acid levels include lowering protein intake and consuming low purine foods. Evidence in support of these dietary recommendations is largely derived from observational studies.

However, these studies have not consistently demonstrated that lower dietary protein intake is associated with a reduction in uric acid levels [7-9]. Furthermore, there are few trials examining the effects of low intake of dietary protein on uric acid levels. Whether replacing protein with saturated fat or changing the type of carbohydrates in the diet has an impact on uric acid concentration is unknown [11].

Although there has been study that showed that moderate restriction of consumption of carbohydrate in reducing uric acid levels [12]; there is no detail evidence to support these results.

Intermittent Fasting (IF) is also called time bound eating with eating window restricted by time. IF is reported to improve the lipid profile, to decrease the inflammatory responses, reflected by change in serum adipokine levels; and to change the expression of genes related to inflammatory response and other factors [27].

In this study we have combined the dietary intervention with intermittent fasting to study the impact on obesity and hyperuricemia.

Aim of Study

The aim of this study was to investigate whether a dietary intervention of low carbohydrate, low protein diet in combination with intermittent fasting would enhance the weight loss and improve the serum uric acid levels in severe obese patient with hyperuricemia.

Methods

Patient: A 46-year-old woman with a history of obesity and with pain while walking & at rest, climbing up the stairs and swelling over her bilateral ankles and difficulty in walking since a year was seeking treatment for the weight loss and hyperuricemia. She was aware of the link between obesity and cardiovascular diseases. She had been normal weight as a child and adolescent. She continued to be of normal weight up until her marriage at the age of 20. After marriage she had to stay away from her husband and with her in-laws which led to chronic stress. After 6 months of marriage, she started getting allergic cough like symptoms on a frequent basis, which was not relieved by medications. From the time of start of allergic symptoms, she developed asthma within six months. SEROFLOW inhaler was prescribed by the doctor with dosage of twice a day, which she followed for the next 7 years.

From 2006 onwards she has a history of gradual increase in weight. In 2020, she gradually started having difficulty in breathing for which she was diagnosed with pneumonia. She was admitted in ICU for 15 days.

After discharge from the hospital, she started having generalised weakness, which she described as not having strength to stand or walk, along with swollen legs from thighs to the ankles & pain over bilateral ankles. This caused her difficulty in doing daily household chores. She reported to having difficulty in speaking because of weakness.

Looking at the severity of the weakness, the doctor advised to get the investigate HBA1c and serum uric acid.

Her readings at the beginning of the protocol on 21st November were:

Age: 46 years Height: 5' feet 1''inch (152 cms) Weight: 78kgs BMI: 33.8 kg/m^2

HBA1C: 6.3 % Serum Uric Acid:7.3mg/dl.

As per the readings she was obese, pre-diabetic and having elevated levels of serum uric acid (hyperuricemia)

Management

On the first consultation with the patient, the detailed protocol for the next 5 weeks was explained. It was decided to closely monitor the compliance for the first week. During the first week the patient was maintaining the daily notes on the effects of the suggested protocol. Counselling was provided daily for the first week to clear any queries and/ or difficulties faced by the patient.

The following protocol was followed:

Intermittent Fasting: Fasting window of 12 hours was implemented. This fasting window was observed between dinner and breakfast of the following day.

Diet Intervention

Proteins: all the pulses, milk, paneer, eggs, fish, chicken, and red meat were stopped completely.

Fat: She was asked to use cold pressed coconut oil for cooking.

Carbohydrates: Refined carbohydrates such as polished white rice, all wheat and wheat derived products were discontinued completely. All the bakery products such as biscuits, bread was discontinued. Any form of sugar such as white crystalline sugar, jaggery, honey was not allowed for the consumption. Instead of refined grains she was advised to eat Indian millets such as jowar, bajra, nachani.

Fruits with low Glycaemic Index were allowed such as pear, strawberry, guava, and fruits with high Glycaemic Index such as banana, apple, watermelon, mango were not allowed.

In vegetables cruciferous vegetables and greens were avoided.

Electrolytes: Refined White salt was replaced with either pink salt or sea salt.

Salt lemon water twice a day was consumed.

She was advised to drink apple cider vinegar 1 teaspoon -2 times a day in a glass of water.

She followed the diet for a month and on 28/12/2020 blood investigations were done, HbA1c and Serum Uric Acid.

Second week onwards the frequency of follow-up counseling and monitoring was changed to once every 3 days. The close monitoring of the patient helped to have early intervention of the any potential challenges faced by the patient.

During the period of the study the patient was on Seroflow (medication for asthma) and no medication for hyperuricemia.

Result

After following the protocol for 5 weeks the same tests were repeated to check the progress. There was significant reduction of 8kgs in bodyweight, which led her to move from obese (BMI 33.8 kg/m2) to being overweight (BMI 30.4 kg/m2) as per the BMI standards. Incredibly significant improvement was observed in HbA1c (decrease of 0.4%) and decrease of 2.2 mg/dl in serum uric acid levels. According to standard guidelines patient was pre diabetic and was going towards full blown diabetes. After following the treatment protocol, HbA1c level was reduced to the extent that she was diagnosed as non-diabetic or normal blood sugar level from pre-diabetes.

With serum uric acid levels, according to standard guidelines patient was diagnosed with gout because of much elevated uric acid level. Post treatment value dropped significantly from 7.3 mg/dl to 5.1 mg/dl which falls under normal range.

There was significant reduction in the values on account of the diet intervention and intermittent fasting protocol followed by the patient.

Discussion

Serum uric acid is the end-product of purine metabolism and has been shown to be associated with an increased risk of hypertension, cardiovascular disease, and chronic kidney disease in previous epidemiological studies [18]. In a large cohort study, there was an association between increasing serum uric acid levels and diabetes mellitus in the whole cohort. It was observed that there is an inverse association between serum uric acid levels and diabetes mellitus in both the age, sex-adjusted and the multivariable-adjusted models. In a supplementary analysis where they examined the association between uric acid and diabetes mellitus, in addition to fasting glucose defined diabetes mellitus as raised HbA1C (levels > 6.5%) [19]. Increased animal protein intake like animal meat, sea food and dairy products is well known to be associated with an increased risk of gout. A higher total intake of vegetable protein is not found to be associated with an increased risk of gout. Some studies suggest that the protein from vegetable sources may have a protective effect instead [20].

The insulin resistance syndrome may present clinically in many different ways, particularly as impaired glucose tolerance with moderate increase in the fasting blood sugar. It may also present as hypertension or as hyperlipidaemia with myocardial ischaemia. Now that hyperuricaemia has come to be recognised as an intrinsic part of the syndrome, it is being realised that one of the clinical presentations may be as gouty arthritis. Indeed, the degree of hyperuricaemia has been proposed as a simple marker of the degree of insulin resistance [21].

When insulin does not work correctly, we have a state called insulin resistance. Insulin resistance is defined where a normal or elevated insulin level produces an attenuated biological response, classically this refers to impaired sensitivity to insulin mediated glucose disposal [13].

At its simplest, insulin resistance is the reduced response to hormone insulin, when a cell stops responding to insulin it becomes insulin resistant. In this state, certain cells need more than usual amount of insulin to get the same response as before. Thus, the key feature of insulin resistance are the higher levels of insulin in blood than they used to be, condition called as hyperinsulinemia [14]. Hyperinsulinemia causes obesity. This point is crucial because it makes obvious that successful treatments to reduce obesity depends

upon lowering insulin. Highly refined and processed carbohydrates-sugars, wheat flour, bread, pasta, muffins, rice, and potatoes are well known to raise blood glucose and insulin production. This theory of obesity is known as carbohydrate- insulin hypothesis [15].

According to the carbohydrate-insulin model (CIM) of obesity, diets high in carbohydrate are particularly fattening due to their propensity to elevate insulin secretion, that promote calorie deposition in adipose tissue, exacerbate hunger, and lower energy expenditure. A large feeding study reported that total energy expenditure (TEE) was greater on a low- versus high-carbohydrate diet, supporting the carbohydrate-insulin model of obesity [16].

Hba1c reflects the body's average level of blood glucose over three months. The use of A1C for the identification of persons with undiagnosed diabetes has been investigated for several years. A1C better reflects long-term glycaemic exposure than current diagnostic tests based on point-in-time measures of fasting and post load blood glucose and has improved test-retest reliability. In addition to utility and convenience, A1C could help identify persons at increased risk of developing diabetes [17].

Dietary fat stimulates insulin the least. Pure saturated fat such as butter, olive oil, coconut oil, stimulate no insulin release [22]. Therefore, replacing refined carbohydrates with natural fat is a simple and natural method of reducing insulin.

Intermittent fasting is a well proven method to lower the insulin levels than calorie-restriction method [23].

Tables

Chart showing pre-treatment and post treatment differences in the markers:

Parameters	Pre-treatment 21/11/2020	Post-treatment 28/12/2020	Reference values
Weight	78 kg	70 kg	43-52Kg
BMI	33.8 kg/m2	30.4 kg/m2	Normal weight: 18.5-24.9 Overweight :25-29.9 Obesity: 30 or greater
HbA1c	6.3%	5.9%	Non-Diabetic: <= 5.6% Prediabetic : 5.7%-6.4% Diabetic: 6.5% or higher
Serum uric acid	7.3 mg/dl	5.1mg/dl	3.4 - 8.0 mg/dl

Conclusion

A two-pronged program, that targets dietary intervention and intermittent fasting, helps in significantly reduce, body weight and hyperuricemia. A treatment programme that included a low carbohydrate and low animal protein with intermittent fasting resulted in substantial drop in bodyweight, BMI, serum uric acid and HbA1c levels.

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