Orthopedics and Rheumatology
Open Access Journal
ISSN: 2471-6804

Review Article
Volume 6 Issue 5 – May 2017
DOI: 10.19080/OROAJ.2017.06.555700

Ortho & Rheum Open Access J

Performance and Atipical and Generalized Location of Urato Crystal Deposits in a Young Patient with Drop

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Submission: April 24, 2017; **Published:** May 11 , 2017

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Summary

Patient (JPG) 43 years old, mestizo, married, attended in consultation of Traumatology and Orthopedics of the Hospital of Specialties of Guayaquil "Dr. Abel Gilbert Ponton", Ecuador in 2015, with APP of Hypertension controlled with drugs and diet, and 10-year history with gout symptomatology and non-acceptance of its image generated by tumors, requires surgery. High blood urea and uric acid levels, normal renal, hepatic andendocrine function was found in laboratory tests.

Keywords: Gouty arthritis; Gout

Introduction

The disease is known since antiquity, being recorded of different historical personages who presented it [1]. It is mentioned in ancient Egyptian texts, such as the Ebers Papyrus, where it is stated that it affects the big toe and mentions the use of colchicum for its healing [2,3]. The Greek physician Hippocrates (Cos, c.460 BC-Thessaly v. 370 BC), quoted her in his writings and made some assertions in his aphorisms that continue to be valid today, as the predominant condition in Males after reaching puberty [4,5]. Galen (Pergamon, Greece, 130-Rome, 200), described the gouty tophs and manifested the importance of diet in the appearance of evil. Much later in the seventeenth century, the English physician Thomas Sydenham (1624, Wynford Eagle (County of Dorset) -1689, London) conducted a detailed study of the symptoms of the disease which he detailed in writing in his works with great accuracy.

In the eighteenth century, uric acid was isolated by the Swedish chemist Carl Wilhelm Scheele, and shortly afterwards, in 1797, chemist and physicist William Hyde Wollaston found his presence in the tophos of an affected patient, thus demonstrating that Uric acid was the causative substance of the condition [5]. In 1848, physician Alfred Baring Garrod (1819-1907) devised a practical use test to check the levels of uric acid in the blood of the patients, which was a very important advance to facilitate an accurate diagnosis, clearly differentiating the disorder of other types of arthritis [6]. Another important contribution was that of Hermann Emil Fischer, who in 1898 showed that uric acid comes from the catabolism of purines of nucleic acids, receiving for his research in this field the Nobel Prize in chemistry in 1902 [7].

Epidemiology

Gout affects between 1% and 2% of the general population at some point in their life. It is more frequent in men, it is estimated that they suffer between five and eight men for each woman. It usually appears in the middle ages of life, usually after the age of 30 [8]. There are differences according to ethnic origin. It is more common in Pacific Islander peoples, and in the Maori population of New Zealand, but rarely appears in the

Australian Aboriginal, despite the latter has a higher mean concentration of serum uric acid [9]. In the United States, gout is twice as common in African Americans as in Caucasians [10].

Etiology

Although all cases of gout are caused by elevation of uric acid levels, the causes of this elevation may be multiple; they are classified into two groups: primary gout and secondary gout [11]. Primary drop corresponds to the vast majority of cases and there is no other disease that is the cause of the problem. Two situations can be established [11]:

a. Increased production of uric acid. The exact defect is usually unknown (idiopathic) although some enzymatic failures are known to cause it, such as hyperactivity of the enzyme phosphoribosyl-pyrophosphate synthetase.

b. Decreased elimination of uric acid from the kidney. There is a renal defect that causes less secretion of uric acid by the renal tubules [11]. Secondary gout. It receives this name the drop originated by another disease or reason, can be distinguished several causes [5]:

c. Increased production of uric acid. As occurs in polycythemia, leukemia, multiple myeloma, hemolytic anemia, extensive psoriasis and malignant tumors. Many of them are due to increased bone marrow activity and intense cellular turnover with increased catabolism of DNA molecules. Also in Lesch-Nyhan disease (juvenile drop choreoathetosis syndrome and mental retardation) due to deficiency of the enzyme hypoxanthine-guaninephosphoribosyltransferase (HPRT) [5].

d. Decreased elimination of uric acid from the kidney. It occurs in renal insufficiency, polycystic kidney, hypothyroidism, diabetes insipidus and dehydration, among others.

e. Originated by medicines. Like diuretics, ethambutol, nicotinic acid and some chemotherapeutics used for the treatment of cancer.

Pathogeny

Purines are part of the DNA and RNA molecules and are generated by the catabolism of nucleic acids. Another source is the intake of foods rich in these substances, such as viscera and shellfish. The metabolism of purines results in hypoxanthine and xanthine which is transformed by the enzyme xanthine oxidase into uric acid. Uric acid is a waste substance that is expelled by the kidneys through the urine in 70%, while through the intestine the remaining 30% is eliminated [12]. High levels of uric acid in the blood are favored by the consumption of foods rich in purines and the intake of alcoholic beverages. Hereditary factors may contribute to elevation of uric acid. Gout occurs when joints, tendons, and surrounding tissues deposit monosodium urate crystals. Uric acid crystals are more likely to form when hyperuricemia occurs, but elevated levels of uric acid in the blood do not necessarily imply that gout is present. People with gout are often obese and have diseases associated with obesity, such as diabetes and hypertension, and are therefore at high risk for heart disease. Gout is more common in opulent societies, due to a diet rich in protein, fat and alcohol. However when it occurs as a consequence of other diseases, such as hemolytic anemia, it is often regardless of the person's lifestyle.

Clinical Picture

Do not confuse gout with hyperuricemia. Hyperuricemia is the elevation of the level of uric acid in the blood; Most people with moderately elevated uric acid levels do not have gout episodes; In contrast it appears in 49% of individuals with very high levels, higher than 9 mg per deciliter. Occasionally there are episodes of gout with normal uric acid levels in the blood [13-16].

Gout presents three clinical phases (Figure 1):

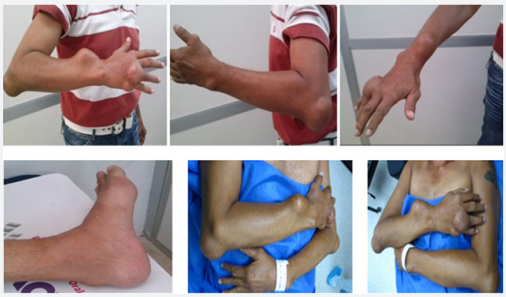

Figure 1: Initial and pre-operative images

a) Acute attack of gout: It can occur in any joint, but in more than 50% of cases it affects the metatarsophalangeal joint (MTT) of the big toe, called podagra. It is also common on the dorsum of the foot, knees, ankles and shoulders. It causes inflammation of the joint and is very painful. The pain is sudden onset, reaching its maximum intensity on the first or second day, disappearing progressively in two or three days, although in severe cases the symptomatology may persist for several weeks. Usually the first attack affects only one joint, but in successive attacks usually affect several, most patients suffer a second attack within less than two years from the first [11].

b) Intercritical drop: This name is the period that exists between two phases of acute gout and during which the patient has no symptoms. This phase can last for months or years depending on the evolution of the disease and the affected individual to perform the appropriate treatment [11] (Figure 2).

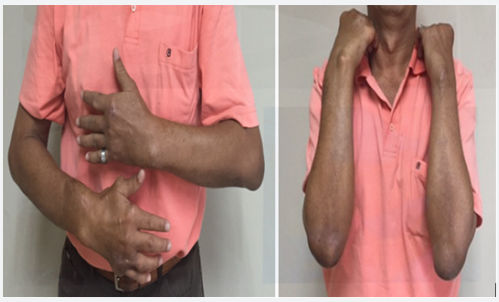

Figure 2: Post Surgical Images.

c) Chronic gout: After a long evolution of the disease, tophans are formed which consist of painless nodules located under the skin, sometimes of large size, in which uric acid is slowly deposited and progressively increase in size if uric acid levels Remain high in blood; On the other hand tend to disappear little by little when the disease is well controlled [11]. Observed under the microscope are characterized by

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an accumulation of urate crystals surrounded by an intense inflammatory reaction, formed in turn by macrophages, giant cells and lymphocytes [17].

Treatment

a. Non-steroidal anti-inflammatory drugs (NSAIDs): They are the most commonly used drugs for the treatment of the acute gout crisis and their efficacy has been demonstrated in different clinical trials, can be administered orally and are also useful for pain relief. However, the use of aspirin is not advised [1].

b. Colchicine: It is a drug widely used for the treatment of acute crisis and is especially useful if given early. It has, however, the disadvantage of a very short half-life and should be avoided if there is diarrhea, hepatic insufficiency or renal failure. It is also used at low doses, between 0.5 and 1 mg daily, as preventive medication [18-20].

c. Hyperhydric drugs: They have the property of reducing the levels of uric acid in the blood, reason why they facilitate the desaturation and the dissolution of the deposits of this substance that are formed in the tissues. The most commonly used drug in this therapeutic group is allopurinol which acts by inhibiting the enzyme xanthine oxidase and is administered at a dose of between 100 and 300 mg daily [1]. An alternative is febuxostat which has the same mechanism of action [21,22].

d. Uricosuric: They are medicines that increase the expulsion of uric acid by the urine. The most used is probenecid [1].

e. Glucocorticoids: Glucocorticoids have been shown to be as effective as NSAIDs [23] and may be used in case of contraindications for NSAIDs [24]. They also cause improvement when injected into the joint; However, infectious arthritis should be ruled out as steroids worsen this disease [24,25].

f. Peglotica: Pegloticasa (Krystexxa), approved in the United States in 2010, for the treatment of gout. [26]. It is considered a therapeutic option for 3% of people who are intolerant of other medications [26]. It is administered by intravenous infusion every two weeks, [26] and has been found to reduce uric acid levels in these people [27].

Prophylaxis

The concentration of uric acid in the blood is the main risk factor for the appearance of gout and is conditioned by its production by the organism and elimination by the kidney [28,29]. The uric acid threshold for gout attacks is approximately 6.7mg/dl. Above this threshold crystals may form. To avoid gout, it is advised to keep blood uric acid levels below 6 mg / dl. The average level of uric acid in males is 5 mg / dl. A low purine diet reduces serum uric acid levels, unless these levels are caused by other health conditions. On the other hand a low calorie diet in obese men decreases on average 100µmol / l (1.7 mg / dl) levels of uric acid in blood [30]. The major sources of purines are DNA and RNA, through their bases adenine and guanine. Some foods such as red meats, seafood and blue fish are rich in purines. People with gout should restrict their use of: [14,31].

a. Meats, mainly red meats and game meats such as wild boar, deer and hare [29,31].

b. Fish, mainly sardines, anchovies, tuna, caviar and fish roe [29,31].

c. Viscera, including gizzards, kidneys, liver and brain [29,31].

d. Seafood, prawns, crabs, crayfish, mussels, others [29,31].

e. Alcohol. Alcohol is a determinant factor in the increase of uric acid at the plasma level and in the peripheral tissues since it favors the production of monosodium urate and induces a deficient excretion of uric acid, especially beer [31,32].

Presentation of a case

We present a patient (JPG) 43 years old, mestizo, married, attended in consultation of Traumatology and Orthopedics with APP of essential hypertension controlled with drugs and diet and history of 10 years with symptomatology of gout in clinical phase intercritica and to facea Chronic and the non-acceptance of his image generated by the tumors, he asked us for surgery. High blood urea and uric acid levels, normal renal, hepatic and endocrine function were found in laboratory tests. APF of Gout in father and paternal grandfather.

The indicated drug treatment consisted of Aines and Uricosurics with Probenecid at recommended pharmacological doses, associated with low purine diet and alcohol withdrawal. Preoperative evaluation and evaluation by anesthesiology, internal medicine and cardiology with assessment suitable for surgery; We offer you excision and biopsy of gouty tophi for outpatient surgery [33-36]. The patient and family accept the proposal and sign informed consent for surgery. Ambulatory surgeries are scheduled in two surgical times, with an interval of 3 weeks between each of them, due to the numerous and scattered tumors of gouty tophi in the upper and lower extremities. Systematic clinical control at 15 days, at one month and at three months, follow-up by specialty of Rheumatology and Internal Medicine is indicated by their essential AHPs.

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