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A REVIEW : ANTIGOUT MEDICINAL PLANTS

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ABSTRACT
It is a disorder of purine metabolism associated with increase level of serum uric acid (hyperuricaemia) above 6.8 mg/dl. Uric acid crystallizes in the form of monosodium urate and deposits in joints, tendons and in the surrounding tissues that manifested as a sudden burning pain, swelling, redness and tenderness in joints. Gout is the common cause of arthritis in men aged over the fifty. Incidence of gout in men is three to four times more common than women because before menopause, estrogen promotes urate wasting in the urine. The male to female ratio is 3.6: 1 but rare in pre-menopausal women and its incidence and prevalence increases with age. However, in patients over the age of 60, gout affects both men and women equally. The major objectives in gout management are to keep the serum uric acid level towards normal, prevent joint damage due to hyperuricemia and further occurrence as well as to promote the dissolution of existing uric acid crystals as well as prevent new crystal formation. However, the main reason for gout and hyperuricemia is related to the overproduction and hypo-excretion of renal uric acid. The aim of present review is to gather collective information of medicinal plants which are used in the therapeutic management of gout with respect to its parts used. This summerised information will be beneficial for further research.

KEYWORDS: Uric acid, Hyperuricemia, Xanthine Oxidase Inhibitor, Gout, Medicinal Plants.

INTRODUCTION
Gout was described by Hippocrates as “the disease of kings” due to its association with a rich diet.[1] It is a disorder of purine metabolism associated with increase level of serum uric acid (hyperuricaemia) above 6.8 mg/dl. Uric acid crystallizes in the form of monosodium urate and deposits in joints, tendons and in the surrounding tissues that manifested as a sudden burning pain, swelling, redness and tenderness in joints. Initially, hyperuricemic persons have no prominent symptoms and they remain asymptomatic for long time. Gout is the common cause of arthritis in men aged over the fifty. Incidence of gout in men is three to four times more common than women because before menopause, estrogen promotes urate wasting in the urine. The male to female ratio is 3.6:1 but rare in pre-menopausal women and its incidence and prevalence increases with age.[2] However, in patients over the age of 60, gout affects both men and women equally.[1] Gout has both modifiable (diet, alcohol, medications, co-morbidities, body mass index, physical fitness) and non-modifiable (genetics, age and gender) risk factors. As the level of uric acid [in men (≤ 7 mg/dl) and women (≤ 6 mg/dl)] crosses its saturation thresholds in physiological fluids, urate crystals precipitation started in the joints and other tissues. The major objectives in gout management are to keep the serum uric acid level towards normal, prevent joint damage due to hyperuricemia and further occurrence as well as to promote the dissolution of existing uric acid crystals as well as prevent new crystal formation.[3] However, the main reason for gout and hyperuricemia is related to the overproduction and hypo-excretion of renal uric acid.[2] Xanthine oxidase (XO) is responsible for oxidation of hypoxanthine to xanthine and finally xanthine to uric acid. Over activity of this enzyme and increased intake of dietary food rich in nucleic acids (e.g. meat, leguminous seeds) impair renal excretion of uric acid and result in hyperuricemia and gout.[4]

PATHOPHYSIOLOGY
Pathogenesis of Hyperuricemia
Urate is the ionized form of uric acid present in the body. Uric acid is a weak acid with pH of 5.8. Urate crystals deposition in tissues starts to occur when serum uric acid level rises above the normal threshold. Pathological threshold of hyperuricemia is defined as 6.8 mg/dL. Some factors may affect the solubility of uric acid in the joints. These include synovial fluid pH, water concentration, electrolytes level and other synovial components such as proteoglycans and collagen. Serum Uric Acid level in the body is determined by the balance

between its production either from purine intake in diet or endogenous production by cellular turnover and its excretion by the kidneys and GIT. Increased production of Uric Acid is responsible for only 10% of cases of gout while the remaining 90% are caused by its renal under-excretion. Factors affecting Serum Uric Acid levels include age and gender. Serum Uric Acid is low in children. After puberty, Serum Uric Acid levels start to increase to reach their normal levels. In men, levels are higher than in women. However, Serum Uric Acid levels in postmenopausal women increase to reach men's levels. This explains why gout is usually a disease of middle aged and older men, and postmenopausal women. Rarely, it may happen in children and young adults in some rare inborn errors of purine metabolism. These enzymatic defects result in increased Serum Uric Acid with consequent production of Uric Acid crystals in kidneys and joints.

Overproduction of uric acid

Deficiency of enzymes involved in purine metabolism leads to overproduction of Uric Acid. For example, Lesch-Nyhan syndrome is an inborn error of metabolism resulting from deficiency of an enzyme involved in Uric Acid metabolism named hypoxanthine–guanine phosphoribosyl transferase. It is a genetic X-linked recessive disorder with varying degrees of severity according to the type of mutation. The clinical picture of this disease involves neurological abnormalities such as dystonia, chorea, cognitive dysfunction, compulsive injurious behavior, self-mutilation and articular manifestations (early onset gout) in addition to renal stones. If left untreated, it may lead to tophi formation and renal failure. Another enzymatic abnormality that causes gout in the young is the superactivity of phosphoribosyl pyrophosphate synthetase. It is an X-linked dominant inherited disorder. The syndrome has two clinical forms, a severe early onset form in children and a mild late juvenile or early adult onset form. Clinical picture includes neurological abnormalities such as sensorineural hearing loss, hypotonia and ataxia in the severe form. The mild form manifests as uric acid renal stones and arthritis. However, these enzymatic disorders constitute only less than 10% of cases of overproduction of urates.

Endogenous urate production

Increased endogenous production of uric acid occurs in accelerated cellular turnover such as in malignancies, heamatological and inflammatory diseases. Also, increased purine production may result from chemotherapy and tissue damage. Furthermore, increased body weight and obesity leads to enhanced production of uric acid aggravating the risk of hyperuriceamia. Leptin was found to increase serum levels of urate. So, weight loss and exercises are very useful in reducing Serum Uric Acid level and gout risk.

Decreased excretion of uric acid

Two thirds of urate excretion occurs in the kidneys while the rest is excreted through the gastrointestinal tract. Reduced secretory function of the transporter ABCG2 leads to decreased excretion of uric acid through the GIT resulting in rise of serum level of uric acid and enhanced renal excretion. Uric acid crystals are not soluble so require specific membrane transporters in order to cross cell membranes. Of these transporters are the urate transporter/channel (URAT) mainly URAT1 and the organic anion transporters (OAT1 and OAT3). Renal excretion of uric acid is the end result of 4 phases. The first phase is the passage of Uric Acid across the Bowman's capsule (glomerular filtration); followed by reabsorption of almost all urates passing in the proximal tubules. The third phase involves secretion of part of the reabsorbed Uric Acid ending with another reabsorption phase in the proximal tubules. The excreted UA is almost 10% of the filtered urate through Bowman's capsule and the rest is reabsorbed in the body. Reduced renal excretion of urate is associated with some autosomal dominant disorders. Uromodulin is a gene that is expressed in the thick ascending limb of the loop of henle. It is responsible for regulating water permeability. Mutations of uromodulin gene result in decreased fractional excretion of Uric Acid, which in turn increases Serum Uric Acid. URAT1 transports Uric Acid in the filtered fluid passing through the proximal tubules into the tubules by an active transport process. Uricosuric drugs such as probenecid, benzbromarone and sulfinpyrazone decrease URAT1 activity, and consequently Uric Acid reabsorption in proximal tubules. On the other hand, drugs such as pyrazinamide, nicotinate and lactate increase urate reabsorption by acting on URAT1, moving Uric Acid from the lumen into the tubular cells. They both increase glomerular filtration and tubular reabsorption of Uric Acid preventing its loss in urine and increasing UA levels in serum. Substances that affect URAT1 activity can both potentiate or inhibit its activity according to their dose. For example, low doses of aspirin have an anti-uricosuric effect while high doses have a uricosuric effect. High dose aspirin inhibits URAT1, hence its uricosuric effect. This process is called cis-inhibition of URAT1. The antiuricosuric effect is caused by trans-stimulation of URAT1 by aspirin.[120]

Table 1: Medicinal Plants Used in Therapy of Gout

Plant Name	Family	Parts used	References
Amaranthus spinosus	Amaranthaceae	Whole Plant	[5]
Acorus calamus	Araceae	Leaves	[6]
Andrographis paniculata	Acanthaceae	Roots	[7]
Adhatoda vasica	Apiaceae	Leaves	[8]
Asparagus racemosus	Asparagaceae	Roots	[9]
Ananas comosus	Bromeliaceae	Roots	[10]
Apium graveolens	Apiaceae	Leaves	[11]
Articum lappa	Asteraceae	Roots	[12]
Abrus precatorius	Fabaceae	Leaves & Seeds	[13]
Ajuga bracteosa	Labiatae	Whole Plant	[14]
Abutilon indicum	Malvaceae	Roots	[15]
Aconitum violaceum	Ranunculaceae	Roots & Rhizome	[16]
Aquilegia fragians	Ranunculaceae	Roots	[17]
Biota orientalis	Cupressaceae	Leaves	[18]
Blumea balsamifera	Compositae	Leaves	[19]
Brassica oleracea	Brassicaceae	Leaves & Roots	[20]
Coriandrum sativum	Apiaceae	Fruit	[21]
Chamomilla recutita	Asteraceae	Flowers	[22]
Cichorium intybus	Asteraceae	Whole plant	[23]
Conyza bonariensis	Asteraceae	Whole plant	[24]
Caesapinia bonduc	Caesalpiniacae	Seeds	[25]
Cappris spinosa	Capparidaceae	Whole Plant	[26]
Cappris aphylla	Caesalpiniacae	-	[27]
Caesalpinia sappan	Caesalpiniacae	Heartwood	[28]
Cannabis sativa	Cannabaceae	Leaves	[29]
Cassia fistula	Caesalpiniacae	Pulp	[30]
Cappris decidua	Capparidaceae	Seeds	[31]
Costus speciosus	Costaceae	Roots	[32]
Citrullus colocynthis	Cucurbitaceae	Leaves	[33]
Coccinia grandis	Cucurbitaceae	Leaves	[34]
Crotalaria Burhia	Fabaceae	Leaves	[35]
Crocus sativum	Iridaceae	Bulbs	[36]
Cinnamomum zeylanicum	Lauraceae	Whole Plant	[37]
Cassia senna	Leguminosae	Leaves & Pods	[38]
Cocculus hirsutus	Menispermaceae	Roots	[39]
Caryophyllus aromaticus	Myrtaceae	Flower buds	[40]
Coix lachryma-jobi	Poaceae	Seeds	[41]
Cymbopogan citrates	Poaceae	Leaves & Stalks	[42]
Cardiospermum halicacabum	Sapindaceae	Whole Plant	[43]
Capsicum annum	Solanaceae	Fruits	[44]
Christella parasitica	Thelypteridaceae	Whole Plant	[45]
Cissus quadrangularis	Vitaceae	Stem	[46]
Curcuma caesia	Zingiberaceae	Rhizomes	[47]
Curcuma longa	Zingiberaceae	Whole Plant	[48]
Curcuma amada	Zingiberaceae	Rhizomes	[49]
Dacus carota	Umbelliferae	Roots	[50]
Datura metel	Solanaceae	Leaves	[51]
Dodonaea viscose	Sapindaceae	Whole Plant	[52]
Delphinium denudatum	Ranunculaceae	Roots	[53]
Ecbolium linneanum	Acanthaceae	Whole Plant	[54]
Elephantopus scaber	Asteraceae	Leaves	[55]
Euphorbia antiquorum	Euphorbiaceae	Stem	[56]
Erythrila stricta	Fabaceae	Roots	[57]
Flacourtica indica	Flacourtiaceae	Bark	[58]
Ficus bungalensis	Moraceae	Leaves	[59]
Ficus carica	Moraceae	Whole Plant	[60]
Gylocardia odorata	Flacourtiaceae	Seeds	[61]
Gloriosa superba	Liliaceae	Tubers	[62]
Gossypium herbacrum	Malvaceae	Leaves	[63]

Helianthus annus	Compositae	Tubers	[64]
Hypericum perforatum	Hypericaceae	Aerial parts	[65]
Hibiscus sabdariffa	Malvaceae	Whole Plant	[66]
Hemidesmus indicus	Periocaceae	Roots	[67]
Indigofera tinctora	Fabaceae	Whole Plant	[68]
Imperata cylindrical	Poaceae	Roots	[69]
Juniperus communis	Cupressaceae	Berries & Leaves	[70]
Jatropha curcas	Euphorbiaceae	Roots	[71]
Kaempferia parviflora	Zingiberaceae	Rhizome	[72]
Launaea sarmentosa	Asteraceae	Whole Plant	[73]
Lepidium sativum	Cruciferae	Seeds	[74]
Momordica charantia	Cucurbitaceae	Fruits & Leaves	[75]
Mesua ferrea	Guttiferae	Stamens	[76]
Mentha Canadensis	Lamiaceae	Roots	[77]
Mollugo carviana	Molluginaceae	Whole Plant	[78]
Myristica fragrans	Myristicaceae	Nut	[79]
Nilgirianthus heyneanus	Acanthaceae	Whole Plant	[80]
Nigella sativa	Ranunculaceae	Seeds	[81]
Nicotiana tobacum	Solanaceae	Leaves	[82]
Olea europaea	Oleaceae	Leaves	[83]
Pistacia integerrima	Anacardiaceae	Leaves	[84]
Polyalthia longifolia	Annonaceae	Stem bark	[85]
Petroselinum crispum	Apiaceae	Seeds & Leaves	[86]
Piper longum	Piperaceae	Fruits	[87]
Pipar nigrum	Piperaceae	Fruits	[88]
Plantago ovate	Plantaginaceae	Seeds	[89]
Physalis alkekengi	Solanaceae	Leaves & Fruits	[90]
Physalis minima	Solanaceae	Fruits	[91]
Physalis peruviana	Solanaceae	Whole Plant	[92]
Pterospermum heyneanum	Sterculiaceae	Flowers & Fruits	[93]
Premna serratifolia	Verbenaceae	Whole Plant	[94]
Rhodiola rosea	Crassulaceae	Roots	[95]
Rosmarinus officinalis	Labiatae	Aerial Parts	[96]
Ranunculus arvensis	Ranunculaceae	Whole Plant	[97]
Semecarpus anacardium	Anacardiaceae	Whole Plant	[98]
Sambucus nigra	Caprifoliaceae	Fruits	[99]
Strychnos nux vomica	Loganiacae	Leaves	[100]
Schrebera swietenioides	Oleaceae	Roots	[101]
Salix alba	Saliaceae	Whole Plant	[102]
Schleichera oleosa	Sapindaceae	Seeds	[103]
Scoparia dulcis	Scrophulariaceae	Whole Plant	[104]
Solanum nigrum	Solanaceae	Leaves	[105]
Tecoma stans	Bignoniaceae	Whole Plant	[106]
Tragia involucrate	Euphorbiaceae	Roots	[107]
Tephrosia purpurea	Fabaceae	Roots	[108]
Trigonella foenumgraecum	Fabaceae	Seeds	[109]
Tinospora cardifolia	Menispermaceae	Whole Plant	[110]
Thalictrum foliolosum	Ranunculaceae	Whole Plant	[111]
Tribulus terrestris	Zygophyllaceae	Fruits & Seeds	[112]
Uraria picta	Papilionaceae	Whole Plant	[113]
Urtica dioica	Uritaceae	Aerial Parts	[114]
Viscum articulatum	Loranthaceae	Whole Plant	[115]
Viola odorata	Violaceae	Aerial Parts	[116]
Withania somnifera	Solanaceae	Roots & Stem	[117]
Ziziphus jujube	Rhamnaceae	Roots	[118]
Zingiber officinale	Zingiberaceae	Rhizomes	[119]

CONCLUSION

Hyperuricaemia, is one of the major risk factor for the development of gout and the main cause of gout is the imbalance between the rates of production and excretion of uric acid. An excess of uric acid thus supersaturates the body fluids, leading to the deposition of monosodium urate crystals in the tissues as well as in joints, which turns leads to initiation of an acute attack of gout. The aim of our study is to provide information of medicinal plants which are used in the therapeutic management of gout with respect to its parts used. This summerised information will be beneficial for further research. However, the additional studies are required to isolate the main active phytochemicals which are responsible for antigout effect as well as to reveal the mechanisms for antigout effect of these medicinal plants.

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