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LAPORAN KASUS ARTRITIS GOUT PADA SENDI JARI DAN TINJAUAN LITERATUR

CASE REPORT OF GOUT ARTHRITIS IN THE JOINTS OF THE FINGERS AND LITERATURE REVIEW

[1] Muhammad Yoga Dwi Anggara Sopiyan, [2] Wan Tisya Muhaira, [3] Fahmi Irsan Nasution

[1,2,3] Fakultas Kedokteran Institut Kesehatan Helvetia, Indonesia
*Koresponden Muhammad Yoga Dwi Anggara Sopiyan. Address: Fakultas Kedokteran Institut Kesehatan Helvetia, Alamat: Jl Kapten Sumarsono 107 Medan Indonesia , email : muhammadyoga@helvetia.ac.id

Abstrak

Gout adalah artritis inflamasi yang paling sering terjadi ketika hiperurisemia, yaitu peningkatan kadar asam urat serum secara terus-menerus, mengakibatkan kejenuhan berlebih jaringan tubuh dengan asam urat, yang menyebabkan pembentukan dan pengendapan kristal monosodium urat di dalam dan di sekitar sendi. Prevalensinya berkisar dari <1% hingga 6,8% dan insidensinya adalah 0,58–2,89 per 1.000 orang per tahun. Gout lebih umum terjadi pada pria daripada wanita, terutama seiring bertambahnya usia, dan pada kelompok etnis tertentu. Seorang pria berusia 49 tahun datang berobat di rumah sakit swasta di Medan dengan keluhan benjolan, pendarahan, dan nyeri pada jempol kaki kiri yang dialami selama 1 bulan. Pasien memiliki riwayat hipertensi, artritis gout, dan penyakit arteriosklerosis jantung (PJK). Kemudian, pasien dirujuk ke departemen ortopedi, di mana ia didiagnosis menderita artritis gout dan direkomendasikan untuk rawat inap. Setelah itu, dilakukan pembedahan dan pemeriksaan histopatologi spesimen tersebut di Laboratorium Patologi Anatomi. Diterima massa satu bagian dengan warna abu-abu kekuningan, konsistensi rapuh, dan dimensi 5 x 4 x 3 cm. Pemeriksaan mikroskopis menunjukkan bahwa massa tersebut sebagian terdiri dari jaringan otot, di beberapa tempat terlihat endapan gout yang dikelilingi oleh jaringan ikat fibrosa dan infiltrasi sel inflamasi mononuklear. Ditemukan histiosit dan sel raksasa secara lokal. Tidak ditemukan tanda-tanda keganasan pada preparat ini. Berdasarkan manifestasi data klinis, hasil laboratorium, pemeriksaan makroskopis dan mikroskopis, pasien ini didiagnosis menderita artritis gout.

Kata kunci: Artritis gout, hiperurisemia, kristal monosodium urat, sendi.

Abstract

Gout is the most common inflammatory arthritis, occurring when hyperuricaemia, a sustained elevation of serum urate levels resulting in supersaturation of body tissues with urate, leads to the formation and deposition of monosodium urate crystals in and around the joints. Range from a prevalence of <1% to 6,8% and incidence of 0,58-2,89 per 1000 person years. Gout is more prevalent in men than in women, particularly with increasing age, and in certain ethnic groups. A 49-year-old man came for treatment at a private hospital in Medan complaining of a bump, hemorrhage, and pain in the big toe on the left side, experienced for 1 month. Patient had a history of hypertension, gout arthtritis and cardiac arteriosclerotic diseases (CAD). Then, the patient was referred to the orthopaedic department, where he was diagnosed with gout arthritis and recommended for hospitalization. After that, the surgery and histopathological examination of that specimen in the Laboratory of Anatomical Pathology. A one-piece mass with yellowish grey colour, friable consistency, and dimensions of 5 x 4 x 3 cm was received. Microscopic examination showed

that it consists of partly muscle tissue; in some places, a gouty deposit is seen, surrounded by fibrous connective tissue and a mononuclear inflammatory cell infiltrate. Locally found histiocytes and giant cells. No sign of malignancy was found in this preparation. Based on clinical data manifestation, laboratory results, macroscopic and microscopic examinations, this patient was diagnosed with gouty arthritis.

Keywords: *Gout arthritis, hyperuricaemia, monosodium urate crystal, joints*

BACKGROUND

Gout is a common cause of inflammatory arthritis. Among the US adult population, the prevalence of gout is 3.9%. Monosodium urate (MSU) crystal deposition is the central pathophysiological cause of the disease. Acute onset of intensely painful monoarthritis, usually affecting the lower limb and most often the first metatarsophalangeal joint, is the classical clinical presentation of gout. The pain of the acute flare usually peaks within 24 h and gradually resolves over 7–14 days. In the setting of an acute inflammatory monoarthritis, concerns about other diagnoses, such as septic arthritis, may necessitate pathological examination of the affected tissue. Furthermore, although the presentation of gout is usually quite characteristic, patients may present with atypical symptoms such as subcutaneous nodules, prolonged joint inflammation, or acute inflammation at uncharacteristic sites. While microscopy of aspirated material for crystal confirmation or advanced imaging methods may assist with the diagnosis, pathological analysis of affected tissue may be required to confirm the diagnosis. We are reporting on a case report of gouty arthritis, which aims to describe the anatomical pathology of gout.[1,2,3,4]

Case report

A 49-year-old man came for treatment at a private hospital in Medan complaining of a bump, hemorrhage, and pain in the big toe on the left side, experienced for 1 month. Patient had a history of hypertension, gout arthtritis and cardiac arteriosclerotic diseases (CAD). Then, the patient was referred to the orthopaedic department, he was diagnosed with gout arthritis and recommended for hospitalization. After that, surgery was carried out in the form of excision and incision as well as exploration of the incision boundaries of the thopus tissue, a mass appeared around the Metacarpophalangeal joint (MCP) of the big toe sinistra and part of the MCP second toe. Thopus destroyed the joint capsule, proximal phalanx, and head of the metatarsal bone. After that, histopathological examination of that specimen in the Laboratory of Anatomical Pathology.

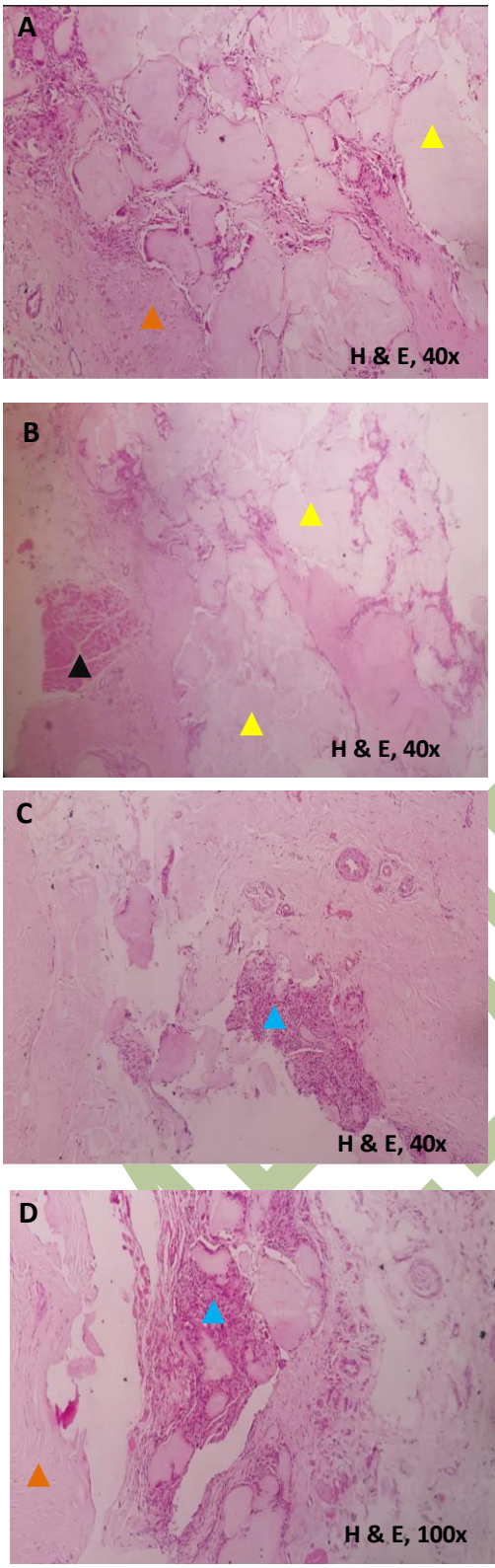


Figure 1. Microscopic feature. A, B, C, and D showed that consist of partly muscle tissue (arrow head black), in some places a gouty deposit (arrow head yellow) is seen surrounded by fibrous connective tissue and a mononuclear inflammatory cell infiltrate (arrow head orange and blue) (H&E, 40x, 100x).

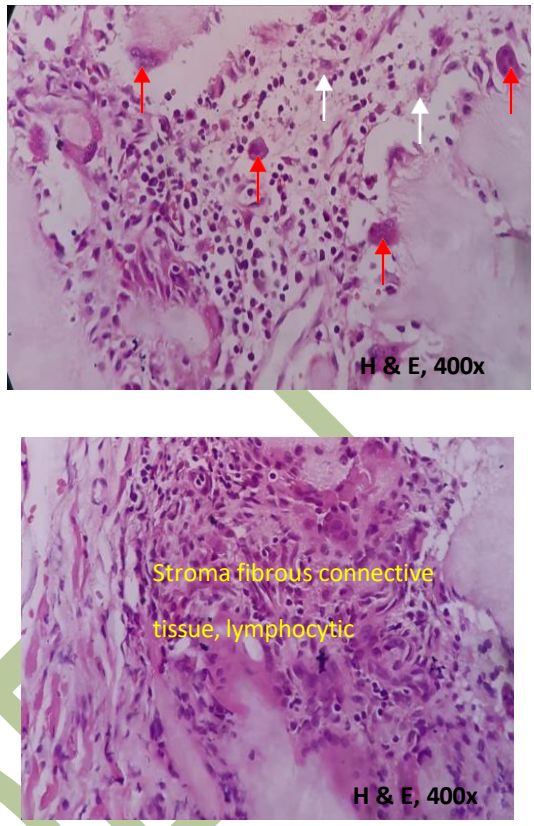


Figure 2. Microscopic feature: Locally found histiocytes and giant cells (arrow white and red) with fibrous connective tissue stroma and lymphocytes.

DISCUSSION

The most prevalent form of inflammatory arthritis is Gout, which results from hyperuricemia. Hyperuricemia is described by the elevated level of serum uric acid. The saturation level of SUA (Serum Uric Acid) at 37 ◦C and pH (power of hydrogen) 7 is 6.8 mg/dL (milligrams per deciliter).[5,6] *Above the saturation level, inflammatory MSU (monosodium urate crystals) are formed in the synovium and joint. Patients are classified as Hyperuricemic if their SUA level is greater than 7 mg/dL in men and 6 mg/dL in women. The frequency and severity of gout are found to be between 1-4% and 0.1–0.3%. Gout is more common in men than in women, by a factor of ratio 3.1 to 10.1. The prevalence of*

gout reported by the national BJD (Bone and Joint Decade) India COPCORD (Community-oriented program from the control of rheumatic diseases) survey of 2006–2011 is 0.05%. Gout incidence and prevalence have been increased in recent decades, with prevalence reaching 11–13% and incidence reaching 0.4% in people over the age of 80. Gout is more common in ethnic minorities in the United States, Han Chinese, New Zealand Maori, and some Asian racial groups. Gout can affect the quality of life and sometimes lead to irreversible joint damage.[3] In this case the patient's uric acid level was 9,5 mg/dl and he was male. According to the literature, uric acid levels >7 mg/dl are classified as hyperuricemia which can lead to gout arthtritis and usually occurs more in men than in female.[7,8,9]

The risk factor of gout arthtritis are obesity (BMI ≥30 kg/m2), lifestyle factors, comorbidities and genetics (dietary patterns explained ≤0.3% of the variance in serum urate levels whereas common, genome wide single nucleotide variants explained 23.9% of the variance, suggesting that diet has less impact on hyperuricaemia risk than genetics). Dietary factor is consuming alcoholic drinks (particularly beer), meat (especially red meat, wild game, and organ meat), some seafood (e.g., shellfish, some large saltwater fish), fruit juice, and beverages sweetened with high fructose corn syrup increases the risk of gout. Purine-rich foods such as nuts, oatmeal, asparagus, legumes, and mushrooms do not seem to increase the risk.[10,11,12] Consumption of dairy products appears to confer slight protection from gout (Table 1).

The Dietary Approaches to Stop Hypertension (DASH) diet comprises fruit, vegetables, low fat dairy products and reduced saturated and total fat content.[4,2]

Table 1 : Risk Factors of Gout

Risk factor	Notes	Relative risk (95% confidence interval)
Diuretic use*	—	3.37 (2.75 to 4.12)
Alcohol intake	≥ 50 g per day vs. none	2.53 (1.73 to 3.70)
Beer	≥ 2 drinks per day vs. none	2.51 (1.77 to 3.55)
Spirits	≥ 2 drinks per day vs. none	1.60 (1.19 to 2.16)
Wine	≥ 2 drinks per day vs. none	1.05 (0.64 to 1.72)
Hypertension	—	2.31 (1.96 to 2.72)
Body mass index	≥ 30 kg per m² at 21 years of age	2.14 (1.37 to 3.32)
Sweetened beverage consumption	≥ 2 drinks per day vs. none	1.85 (1.08 to 3.16)
Fructose intake	Highest vs. lowest quintile	1.81 (1.31, 2.50)
Seafood consumption	Highest vs. lowest quintile	1.51 (1.17, 1.95)
Meat consumption	Highest vs. lowest quintile	1.41 (1.07, 1.86)
Dairy product consumption	Highest vs. lowest quintile	0.56 (0.42, 0.74)
Vitamin C intake	≥ 1,500 mg vs. < 250 mg per day	0.55 (0.38, 0.80)
Coffee consumption	≥ 6 cups per day vs. none	0.41 (0.19, 0.88)

*—Adjusted for age.

Adapted with permission from Roddy E, Doherty M. Epidemiology of gout. Arthritis Res Ther. 2010;12(6):223, with additional information from reference 12.

Comorbidities are complex, with some diseases predisposing to hyperuricaemia and/or gout and others arising as a consequence of gout. Recent research has confirmed earlier observations about traditional cardiovascular risk factors (such as hypertension and hyperlipidaemia), Cardiac Vascular Disease (CVD) and Chronic Kidney Diseases (CKD). A large study of data from the UK Clinical Practice Research Datalink (CPRD) investigated temporal relationships between the occurrence of comorbidities before and after a first diagnosis of gout. This study confirmed hypertension,

hyperlipidaemia and renal disease as risk factors for gout, as well as the well recognized association of gout with sub sequent CVD and renal disease (figure 3).[2] *In accordance with the literature that the patient has a history of previous diseases such as gout, arthritis, hypertension and coronary acute diseases as comorbid factors.*

Over time, the joint space can be irreversibly damaged, leading to chronic pain and disability with grossly deformed joints. Tophi (i.e., subcutaneous nodules comprised of monosodium urate crystals in a matrix of lipids, protein, and mucopolysaccharides) may also form at the joint space (Figure 4A).[5] *The first metatarsophalangeal joint is most commonly affected. Other common sites include the midtarsal joints, ankles, knees, fingers (Figure 4B), wrists, and elbows. Urate crystals may also be deposited throughout the body (e.g., vertebrae, skin, soft tissues), mimicking other disease states.*[4,5] *In this case report, the location of gout arthritis is in accordance with the literature, namely on the big toe sinistra which extends to the metacarpophalangeal joint area to part of the second toe.*

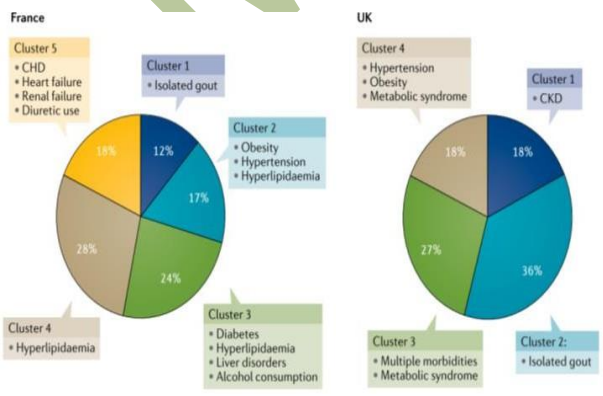


Figure 3. Patterns of comorbidity clustering in individuals with gout.[2]

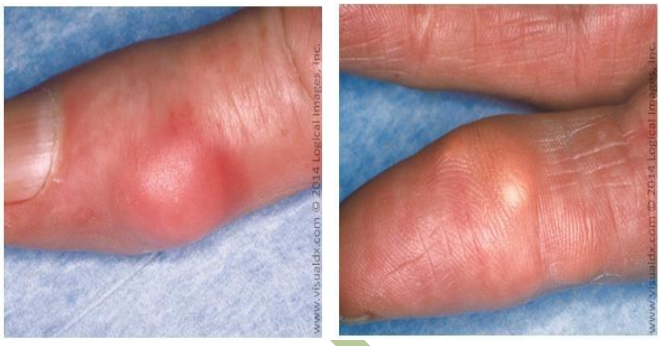


Figure 4. A. Hard nodules on distal digiti. B. Tender red papules on digit.[2]

The typical first presentation of gout is an intensely painful acute inflammatory arthritis (gout flare) affecting a lower limb joint.[1] *In the absence of treatment, the gout flare is typically self-limiting over a period of 7–14 days. After resolution, there is a pain-free asymptomatic period (intercritical gout), until another gout flare occurs. Over time, some people with persistent hyperuricaemia also develop tophi, chronic gouty arthritis (persistent joint inflammation induced by monosodium urate crystals), and structural joint damage.*[5,13,14]

Gout flares can occur in the joints or periarticular tissues (eg: bursae, tendons, and entheses). The pain can be described as stabbing, gnawing, burning, or throbbing. Another well described feature of the gout flare pain is its short time from onset to peak intensity (usually less than 12 h). Accompanying features of the flare are varying degrees of swelling, warmth, and erythema. Depending on location and intensity of the gout flare, tophi leads to restriction to joint movements/ limitation in the ability to use

the affected area, concerns about physical appearance, difficulty fitting shoes, difficulty walking, and a fear of even minimal physical contact. The pattern of joint involvement during a gout flare can greatly aid in its recognition. The lower limb (foot, ankle, and knee) is preferentially involved.[5,16,17] *In accordance with the symptoms and signs in literature, the patient complained of a bump, hemorrhage, and pain in the big toe sinistra.*[8]

Gout flares are usually monoarticular, although oligoarticular or polyarticular episodes do occur, typically in patients with poorly controlled disease or during hospitalisation. Polyarticular flares can be associated with pronounced systemic symptoms, including fever, chills, and even delirium. The recurrence of gout flares is difficult to predict, but the likelihood of recurrent flares is associated with the severity of hyperuricaemia. On palpation, tophi are firm or hard, but softening can occur during resolution with urate-lowering therapy. Although tophi can become acutely inflamed, these lesions are usually not tender, and without warmth or erythema.[4,5,15,17] *The American College of Rheumatology criteria are the most widely used for diagnosis of gout (Table 2).*[4] *In accordance with the literature, in this case also shows monoarticular (only attacking one part of the joint).*

Table 2. American College of Rheumatology Diagnostic Criteria for Gout.[4]

Table 2. American College of Rheumatology Diagnostic Criteria for Gout

Presence of characteristic urate crystals in the joint fluid
or
Presence of a tophus proven to contain urate crystals by chemical means or polarized light microscopy
or
Presence of six or more of the following clinical, laboratory, or radiologic findings:
- Asymmetric swelling within a joint on radiography
- Attack of monoarticular arthritis
- Culture of joint fluid negative for microorganisms during attack of joint inflammation
- Development of maximal inflammation within one day
- Hyperuricemia
- Joint redness
- More than one attack of acute arthritis
- Pain or redness in the first metatarsophalangeal joint
- Subcortical cyst without erosions on radiography
- Suspected tophus
- Unilateral attack involving first metatarsophalangeal joint
- Unilateral attack involving tarsal joint

Adapted with permission from Wallace SL, Robinson H, Masi AT, Decker JL, McCarty DJ, Yü TF. Preliminary criteria for the classification of the acute arthritis of primary gout. Arthritis Rheum. 1977;20(3):896.

Pathophysiology of hyperuricemia

Purine is converted to hypoxanthine, which is then oxidized by XO (xanthine-oxidase) to form xanthine. Uric acid is formed when xanthine is oxidized again by xanthine oxidase. Fig. 5 shows the enzymatic pathway of purine to the uric acid end product.[3,7,9]

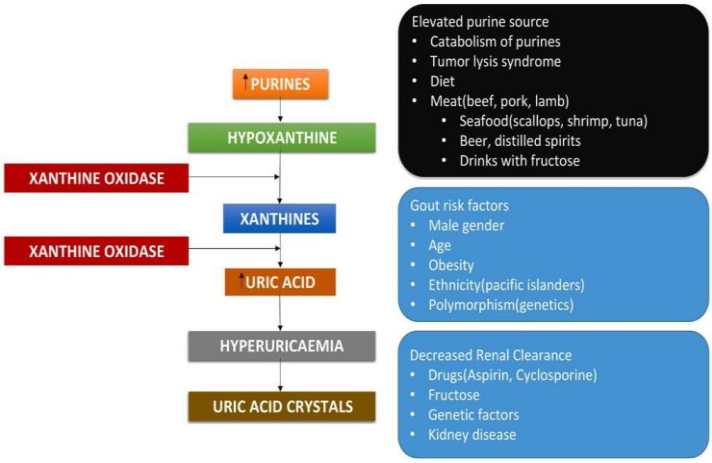


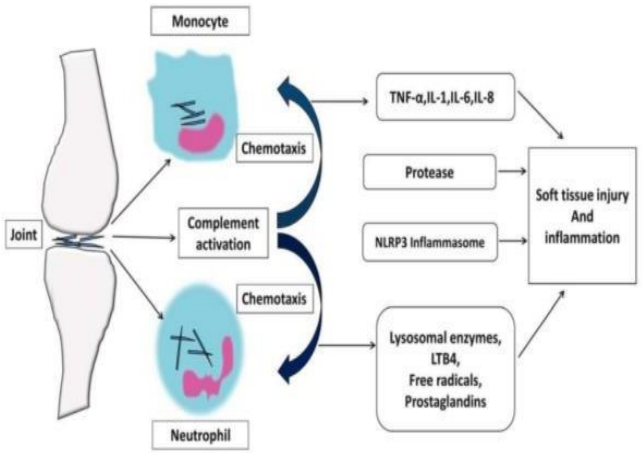
Fig. 5. Enzymatic pathway of purine to uric acid end product and risk factors of elevated uric acid level.[3]

Pathophysiology gout arthtritis

When these crystals are engulfed by synovial cells, they allow lysosomal enzymes to be released and activates inflammatory chemokines, which start the inflammatory process. The release of mast cells and monocytes, as well as the activation of neutrophils, is linked to gouty arthritis. These crystals can be contained by well-differentiated macrophages without triggering an inflammatory response. Mast cells also play an important role in triggering acute gouty attacks by releasing histamine and IL-1 (Interleukin), as a result of this vascular permeability and vasodilation increase. Chemotactic factors generated by monocytes and mast cells, as well as local vasodilation, contribute to neutrophil chemotaxis. The recruitment of endothelial cells also exacerbates the inflammatory reaction and neutrophil migration. This causes an excess of neutrophils in the region. The 90% of neutrophil activation and acute inflammation exacerbation is caused by an excess of chemotactic factors such as leukotrienes, platelet-activating agent, and interleukins, especially IL-8, within the synovium. As a result, focusing on IL-8 could be a successful strategy for preventing acute gout attacks. The acute gout attack is normally self-limiting. It goes away within a few hours or days after it appears. By clearing cellular apoptotic remnants, macrophages also help in the slowing of the inflammatory cascade. TNF -β (tumor necrosis factor-beta) is also secreted by macrophages, which inhibits IL-1, a major player in the inflammatory process.[3]

Chronic gouty arthritis is caused by systemic inflammation that occurs as a result of repeated gout attacks. Chronic gout is marked by severe synovitis, bony erosions, cartilage destruction, and tophi growth. This may be due to a variety of factors. Chondrocytes are activated to release inflammatory cytokines, matrix metalloproteases, and nitric oxide when urate crystals are found in the synovium, causing cartilage damage and the initiation of bone erosions. Anti-inflammatory cytokines are essential regulators of the inflammatory response. Proteolysis of proinflammatory cytokines, as well as decreased expression of TNF-α (tumor necrosis factor-alpha) and interleukin receptors on the surface of leukocytes, are both involved in resolving the acute attack. Vasodilation and increased vascular permeability are required for macrophage extravasations into the synovial fluid to clear the inflammatory regions (Fig.6).[3]

Fig. 6. Pathophysiology of Gouty Arthritis inflammation.[3]



NLRP3 (Nod-like receptor protein) inflammasome plays an important role in the onset of acute inflammation of gout. ASC (adaptor molecule apoptosis- associated speck-like protein containing a CARD) is an important linker between the NLRP3 protein and pro-caspase-1 protease in joint inflammation of acute gout. Martinon et al. When monocytes ingest MSU crystals, Toll-like receptors TLR2 and TLR4 activate the NLRP3 inflammasome. ASC adaptor protein promotes the hydrolysis of pro-caspase-1 protease and converts pro-caspase-1 protease to active caspase-1 protease. Resulting in the development of pro-inflammatory IL-1 β and mature IL-1 β after cleavage by active caspase-1, eventually leading to a strong inflammatory response in gout patients (see Fig. 7).[3,11,16]

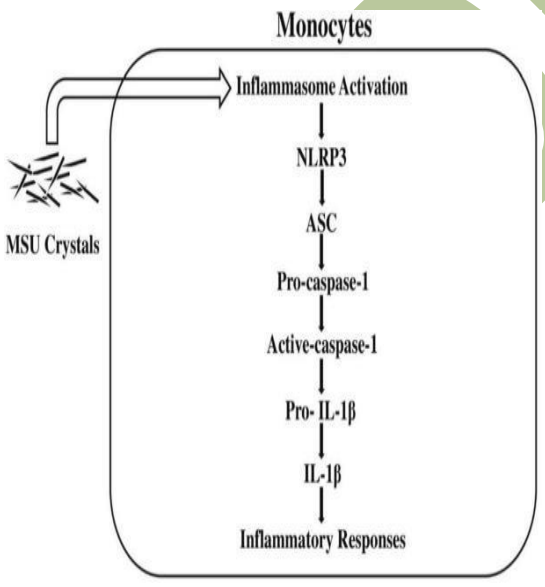


Figure 7. NLRP3 Inflammasome activation and Pathway of inflammatory response in monocyte.[3]

Microscopic confirmation of monosodium urate crystals in synovial fluid or tophi is considered the gold standard for gout diagnosis. Monosodium urate crystals appear needle-shaped and negatively birefringent under polarising light microscopy (figure 8). In synovial fluid, crystals range in length from 1 µm to 20 µm, but can measure up to 40 µm in tophi.[5,7,8]

Gout can also be diagnosed with a high level of certainty and without recourse to joint aspiration in the presence of typical signs and symptoms (eg, podagra). A diagnostic compound score for gout has been developed and validated for patients presenting with monoarthritis in primary care settings. This score comprises the following items: male sex, previous patient-reported arthritis attack, onset within 1 day, joint redness, first metatarsophalangeal joint involvement, hypertension or cardiovascular disease, and high concentration of serum urate. A score of less than 4 ruled out gout in more than 97% of patients, and more than 80% of patients with a score of 8 or higher had gout.[5]

The ultrasonographic findings of gout include a double contour sign (reflecting monosodium urate crystal deposition on the surface of hyaline articular cartilage), intra-articular or intrabursal tophi, and a snowstorm appearance (Figure 9A, B).[6] *Plain radiographs are usually normal at the time of initial diagnosis. In late-stage disease, characteristic features include defined, juxta-articular punched out erosions with sclerotic margins and over-hanging edges (figure 9C). Joint space width is generally preserved except in advanced tophaceous gout, at which*

time joint space widening or narrowing can occur (figure 9C).[5]

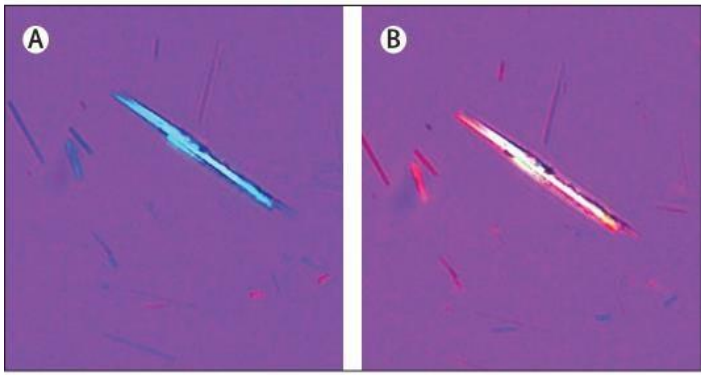


Figure 8. Polarising light microscopy of synovial fluid demonstrating negatively birefringent monosodium urate crystals.[5]

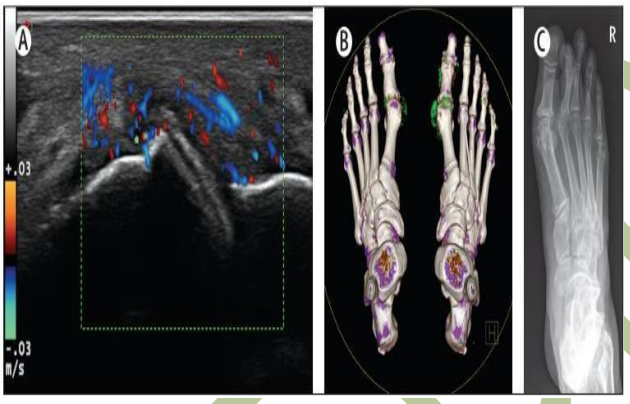


Figure 9. Radiology of gout (A) Ultrasonography scan of the dorsal aspect of the first metatarsophalangeal joint showing the double contour sign, which reflects monosodium urate crystal deposition on the surface of the articular hyaline cartilage; image acquired during a flare, showing intense colour doppler signal. Image courtesy of Philip Courtney. (B) Dual-energy CT of a patient with gout, showing deposition of monosodium urate crystals (shown in green), particularly at the first metatarsophalangeal joints. (C) Plain radiograph of the right foot demonstrating erosive gout in a patient with extensive subcutaneous tophi. Note the involvement of the first, second, and fifth metatarsophalangeal joints, and toes.[5]

Macroscopic subcutaneous tophi present as "draining or chalk-like subcutaneous nodule under transparent skin, often with overlying vascularity, located in typical locations: joints, ears, olecranon bursae, finger pads, tendons (eg, Achilles)". Subcutaneous tophi can vary greatly in size, from barely palpable to large confluent masses (figure 10).[5,4] *According to the macroscopic picture literature in the case report shows mass with yellowish grey colour, fragilly consistency with dimension of 5 x 4 x 3 cm was received.*

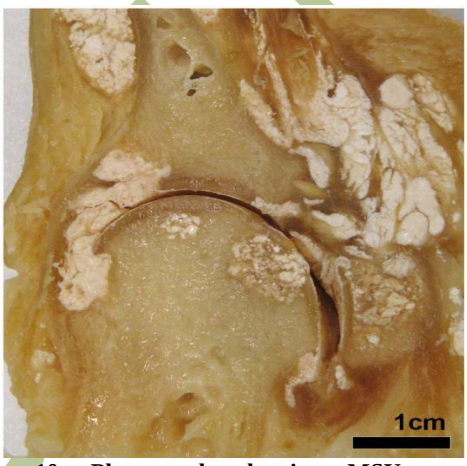


Figure 10. Photograph showing MSU crystal deposition, tophus and joint damage at the macroscopic level (sagittal plane) in the left first metatarsophalangeal joint from a cadaveric donor with tophaceous gout. Bone erosion and cartilage damage adjacent to MSU crystal deposition and tophus can be seen. Fibrous septae are also evident between deposits of MSU crystals within the tophus.[1]

Microscopic features consist of tophi (advanced), reactive granulomatous inflammation, surrounds fluffy (cotton candy-like) material, fibrotic synovium, aggregates of urate crystals - considered gold standard (figure 11).[7] *According to the microscopic picture in the literature, in this case report shows that it consists of partly muscle tissue, in some places a gouty deposit is seen surrounded by fibrous connective tissue and a mononuclear inflammatory cell infiltrate. Locally found histiocytes and giant cells.*

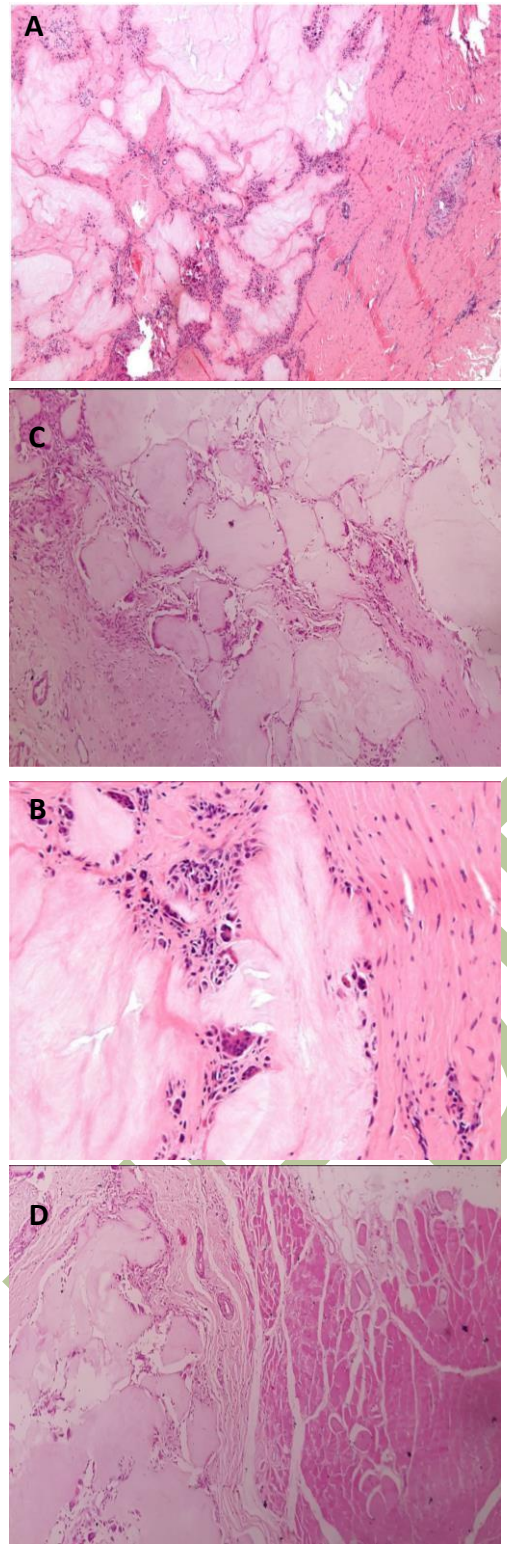


Figure 11. Based on literature. (A,B). Gouty tophus (low and high magnification).[7] Based in case report, (C,D). Gouty thopus is surrounded by a stroma fibrous connective tissue and inflammatory cell of lymphocytes, and muscle cell are visible in between.

In the treatment of gout, a distinction must be made between an acute gout attack and the chronic phase. The acute attack must be treated first. Rapid pain relief by diminishing the inflammatory reaction is crucial for achieving a rapid improvement of mobility. Further joint damage, especially involving the cartilage, is reduced by terminating the inflammatory reaction and joint function is rapidly restored. Sustained lowering of the elevated uric acid is the priority subsequently for secondary prevention of complications of chronic gout, which can involve both joints (deforming them with secondary osteoarthritis) and the kidneys (chronic uric acid renal disease). Besides the non-pharmacological dietary treatment methods, drugs are available to reduce the uric acid, but their use must be carefully considered when treating elderly patients.[4,16]

If the disease has not yet led to joint or organ damage, this clinical manifestation can be prevented by consistent uric acid-lowering treatment. According to the recommendations of the EULAR the serum urate level should be lowered constantly below the solubility threshold of 360 µmol/l (6.0mg/dl). Non-pharmacological treatment in the form of dietary changes and lifestyle modification should take place, regardless of the uric acid level. Success with conservative measures can be expected up to a serum urate level of approximately 7mg/dl. Pharmacological treatment is indicated at the latest after the second attack or if organ complications, such as joint destruction or renal calculi, are present. provides an overview of possible treatment approaches.[4]

The differential diagnosis for acute monoarticular joint may also form at the joint space swelling includes pseudogout, infection, and trauma. Pseudogout, or calcium pyrophosphate deposition disease, can mimic gout in clinical appearance and may respond to non-steroidal anti-inflammatory drugs (NSAIDs). Findings of calcium pyrophosphate crystals and normal serum uric acid levels on

joint fluid analysis can differentiated pseudogout from gout.[4,17]

CONCLUSION

A 49-year-old man came for treatment at a private hospital in Medan complaining of a bump, hemorrhage and pain in the big toe sinistra experienced for 1 month. Patient had history of hypertension, gout arthtritis and cardiac arteriosclerotic diseases (CAD). Then, the patient was referred to the orthopaedic department, where he was diagnosed with gout arthritis and recommended for hospitalization. Based on clinical data manifestation, laboratory results, macroscopic and microscopic examinations, this patient was diagnosed as gouty arthritis.

REFERENCE

1. Towiwat P, Chhana A, Dalbeth N. The anatomical pathology of gout: A systematic literature review. BMC Musculoskelet Disord. 2019;20(1):1–14.
2. Dehlin M, Jacobsson L, Roddy E. Global epidemiology of gout: prevalence, incidence, treatment patterns and risk factors. Nat Rev Rheumatol. 2020 Jul 15;16(7):380–90.
3. Patil T, Soni A, Acharya S. A brief review on in vivo models for Gouty Arthritis. Metab Open. 2021 Sep;11:100100.
4. Hainer BL, Matheson E, Wilkes RT. Diagnosis, treatment, and prevention of gout. Am Fam Physician. 2014 Dec 15;90(12):831–6.
5. Dalbeth N, Gosling AL, Gaffo A, Abhishek A. Gout. Lancet (London, England). 2021 May 15;397(10287):1843–55.
6. Presentation C. Volume 21 Number 1. Etlik Vet Mikrobiyoloji Dergisi; Cilt 21 Sayı 1. 2010;21(1).
7. Vijay Shankar, MD. Arthritis, Gout, and gouty arthritis [Internet]. PathologyOutlines.com, Inc. 2020. Available from: http://pathologyoutlines.com/joints.html#gout.
8. Dalbeth N, Merriman TR, Stamp LK. Gout. *Lancet*. 2016;388(10055):2039–2052.
9. Richette P, Bardin T. Gout. *Lancet*. 2010;375(9711):318–328.
10. Neogi T. Gout. *N Engl J Med*. 2011;364(5):443–452.
11. Choi HK, Mount DB, Reginato AM. Pathogenesis of gout. *Ann Intern Med*. 2005;143(7):499–516.
12. Toprover M, Krasnokutsky S, Pillinger MH. Gout in the hand and wrist. *Bull Hosp Jt Dis*. 2014;72(1):71–78.
13. Pascual E, Sivera F, Andrés M. Synovial fluid analysis for crystals. *Curr Opin Rheumatol*. 2011;23(2):161–169.
14. Ragab G, Elshahaly M, Bardin T. Gout: An old disease in new perspective. *J Adv Res*. 2017;8(5):495–511.
15. Thiele RG, Schlesinger N. Diagnosis of gout by ultrasound. *Rheumatology (Oxford)*. 2007;46(7):1116–1121.
16. Kuo CF, Grainge MJ, Zhang W, Doherty M. Global epidemiology of gout. *Nat Rev Rheumatol*. 2015;11(11):649–662.
17. McCarty DJ. Gout. In: Firestein GS, Budd RC, Gabriel SE, McInnes IB, O'Dell JR, editors. *Kelley and Firestein's Textbook of Rheumatology*. 10th ed. Philadelphia: Elsevier; 2017. p. 1527–1553.