Uncover New Insights in Gout Flare Management

Gout should be viewed as an autoinflammatory disease instead of a purely metabolic disease, and the interplay of inflammatory pathways should be accounted for while approaching gout flare management.[1]

Management

Treatment goal for gout flares[2-4]

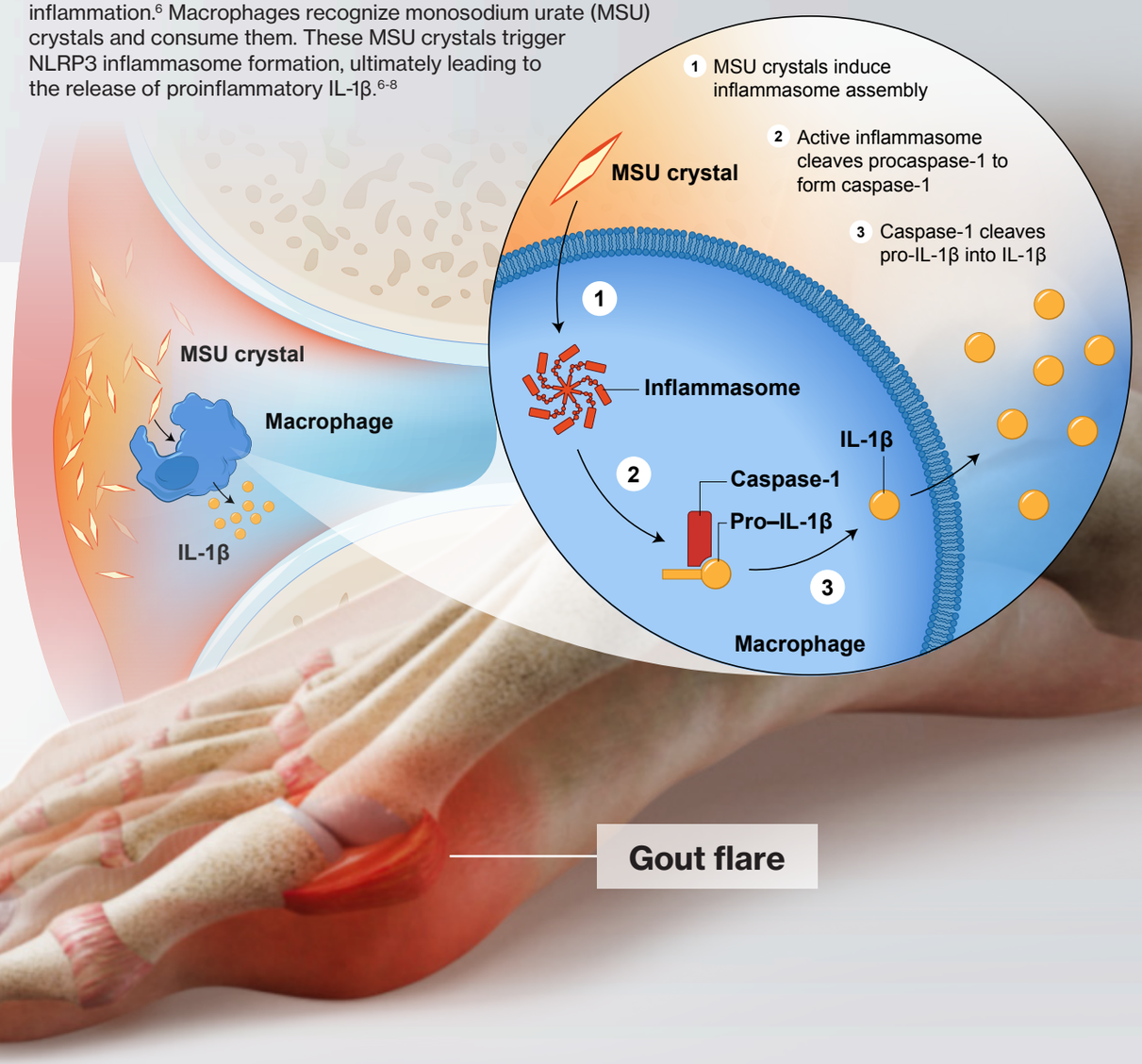
Decrease acute inflammation induced by MSU crystals during gout flares as quickly as possible.

The American College of Rheumatology[5]

- Strongly recommends using colchicine, nonsteroidal anti-inflammatories, or glucocorticoids as appropriate first-line therapy over interleukin (IL)-1 inhibitors or adrenocorticotropic hormone
- Conditionally recommends using an IL-1 inhibitor over no therapy (beyond supportive/analgesic treatment) for whom antiinflammatory therapies are either ineffective, poorly tolerated, or contraindicated

Inflammatory response in gout flares mediated by IL-1

IL-1 is a crucial mediator of gout flare–associated pain and inflammation.[6] Macrophages recognize monosodium urate (MSU) crystals and consume them. These MSU crystals trigger NLRP3 inflammasome formation, ultimately leading to the release of proinflammatory IL-1β.[6-8]



NLRP3, NLR family pyrin domain containing 3.

1. Bodofsky S, et al. *Semin Arthritis Rheum.* 2020;50(5):1089-1100. **2.** Schlesinger N. *Drugs.* 2004;64(21):2399-2416. **3.** Pascual E, Sivera F. *Curr Opin Rheumatol.* 2007;19(2):122-127. **4.** Fels E, Sundy JS. *Curr Opin Rheumatol.* 2008;20(2):198-202. **5.** FitzGerald JD, et al. *Arthritis Rheumatol.* 2020;72(6):879-895. **6.** Klück V, et al. *Joint Bone Spine.* 2021;88(2):105092. **7.** Schlesinger N. *Drugs.* 2011;71(11):1413-1439. **8.** Schett G, et al. *RMD Open.* 2015;1(suppl 1):e000046.

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