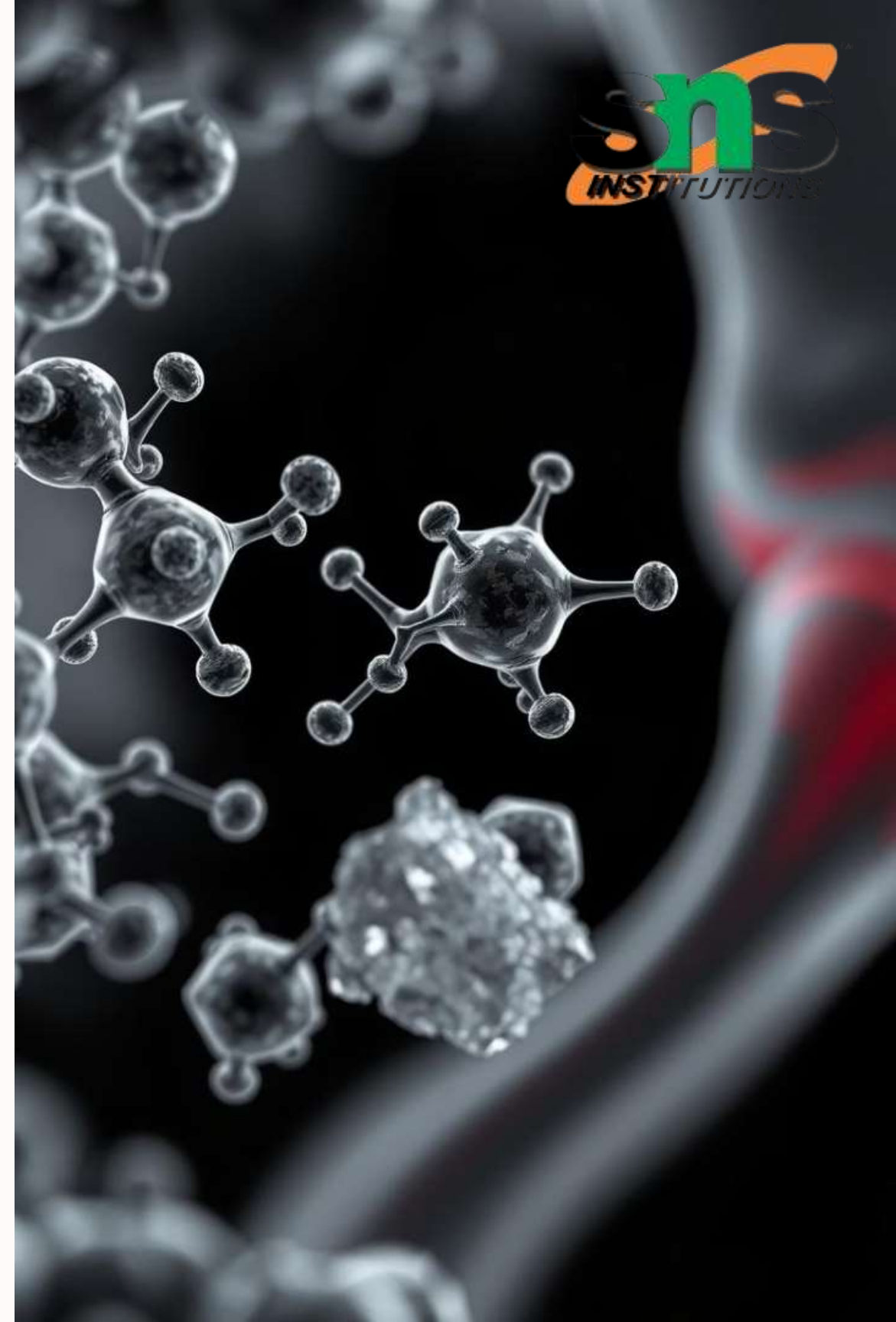




Catabolism of Purine Nucleotides, Hyperuricemia, and Gout

This presentation explores purine nucleotide breakdown and its link to diseases. We will cover hyperuricemia, a condition defined by high uric acid levels, and gout, which affects millions of adults.

S by Sri Vikram S



Purine Nucleotide Sources and Importance



Dietary Intake

Purines enter the body through food.



De Novo Synthesis

The body also creates purines internally.



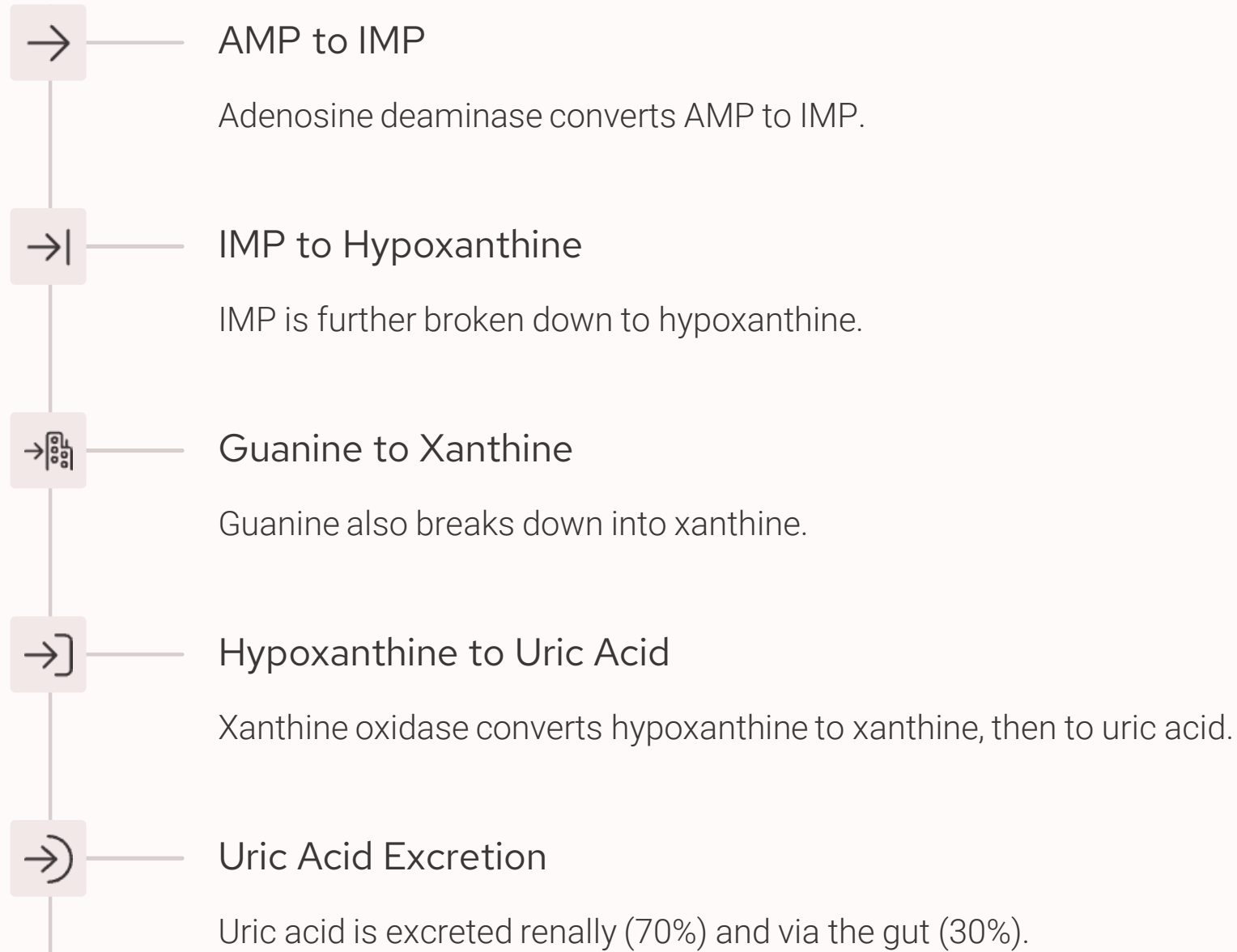
Salvage Pathways

Existing purines are recycled for use.

Purines are crucial for DNA/RNA synthesis. They also play vital roles in energy transfer and cell signaling within the body. Daily intake and endogenous production contribute to overall purine levels.



Catabolic Pathway of Purine Nucleotides



This pathway shows how purine nucleotides are broken down step-by-step. Uric acid is the final product, primarily eliminated by the kidneys.

Hyperuricemia: Causes and Consequences

Causes

- Uric acid overproduction
- Uric acid underexcretion
- Genetic mutations (SLC22A12, ABCG2)
- High purine diet, fructose intake
- Renal disease, myeloproliferative disorders
- Certain medications

Consequences

- Gout attacks
- Kidney stones
- Chronic kidney disease

Hyperuricemia often results from underexcretion of uric acid. Various factors, including genetics, diet, and medical conditions, contribute to its development. Untreated, it can lead to severe health issues.

Gout: Pathophysiology and Clinical Presentation



MSU Crystal Deposition

Urate crystals accumulate in joints.



Inflammatory Response

Inflammasome activation causes severe pain.



Acute Gouty Arthritis

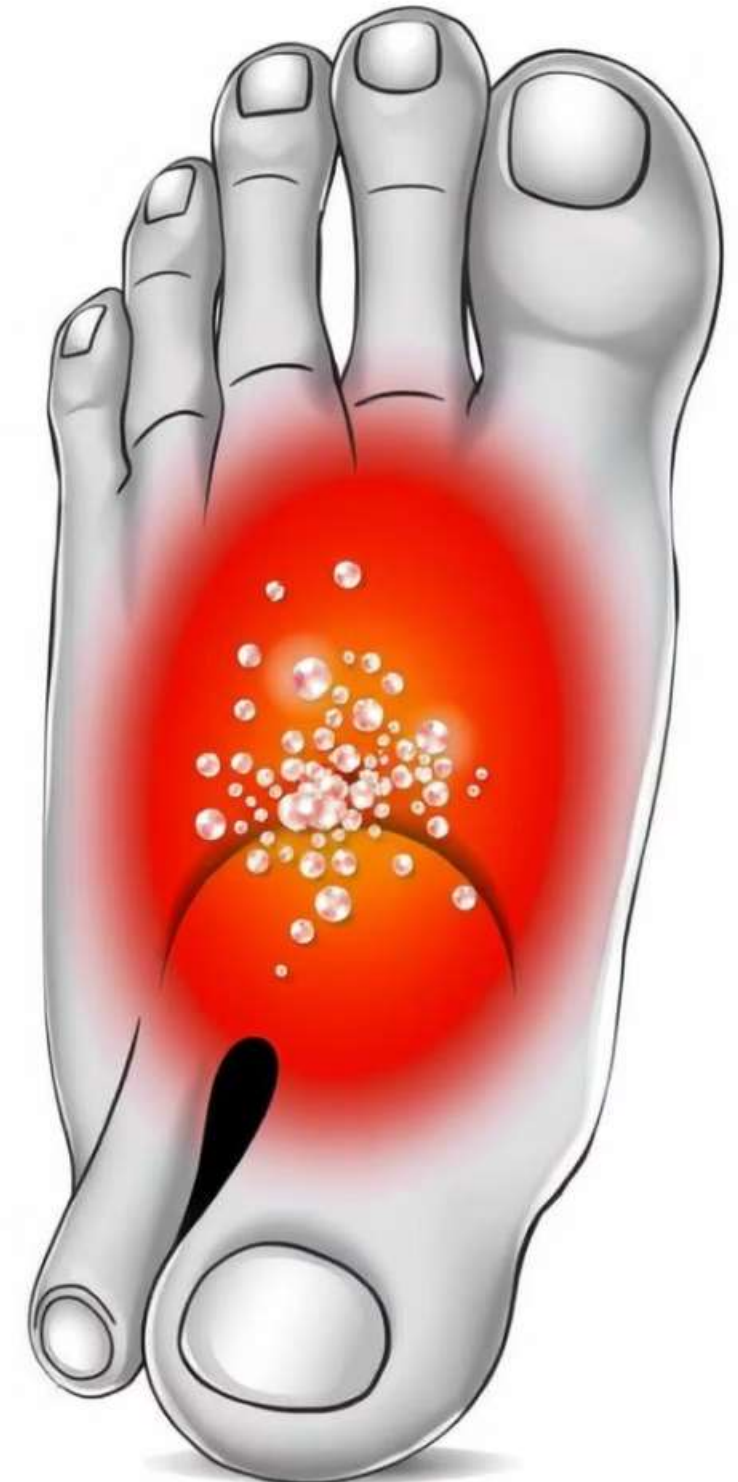
Sudden, intense joint pain, redness, swelling occurs.



Podagra

The big toe is frequently affected.

Gout is characterized by monosodium urate crystal deposition. This triggers an inflammatory response. The severe pain, especially in the big toe, is a hallmark. Chronic gout can lead to tophi formation and increased cardiovascular risk.



Diagnosis of Gout

Joint Aspiration

Fluid is extracted from the affected joint.

Synovial Fluid Analysis

Examining the fluid for crystal presence.

Polarized Light Microscopy

Identifies characteristic MSU crystals.

Serum Uric Acid Levels

Blood tests help assess uric acid concentration.

ESR/CRP Measurement

Inflammatory markers are checked during flares.

X-rays

Evaluates long-term joint damage.

Accurate diagnosis of gout involves several steps. Joint aspiration is key to directly observing MSU crystals. Blood tests and X-rays also provide important diagnostic information.

Treatment Strategies for Hyperuricemia and Gout

Lifestyle Modifications

Diet adjustments, weight management, and proper hydration are crucial.

Acute Flare Management

NSAIDs, colchicine, and corticosteroids reduce inflammation and pain.

Urate-Lowering Therapy (ULT)

Allopurinol and febuxostat lower uric acid production.

Enhanced Excretion

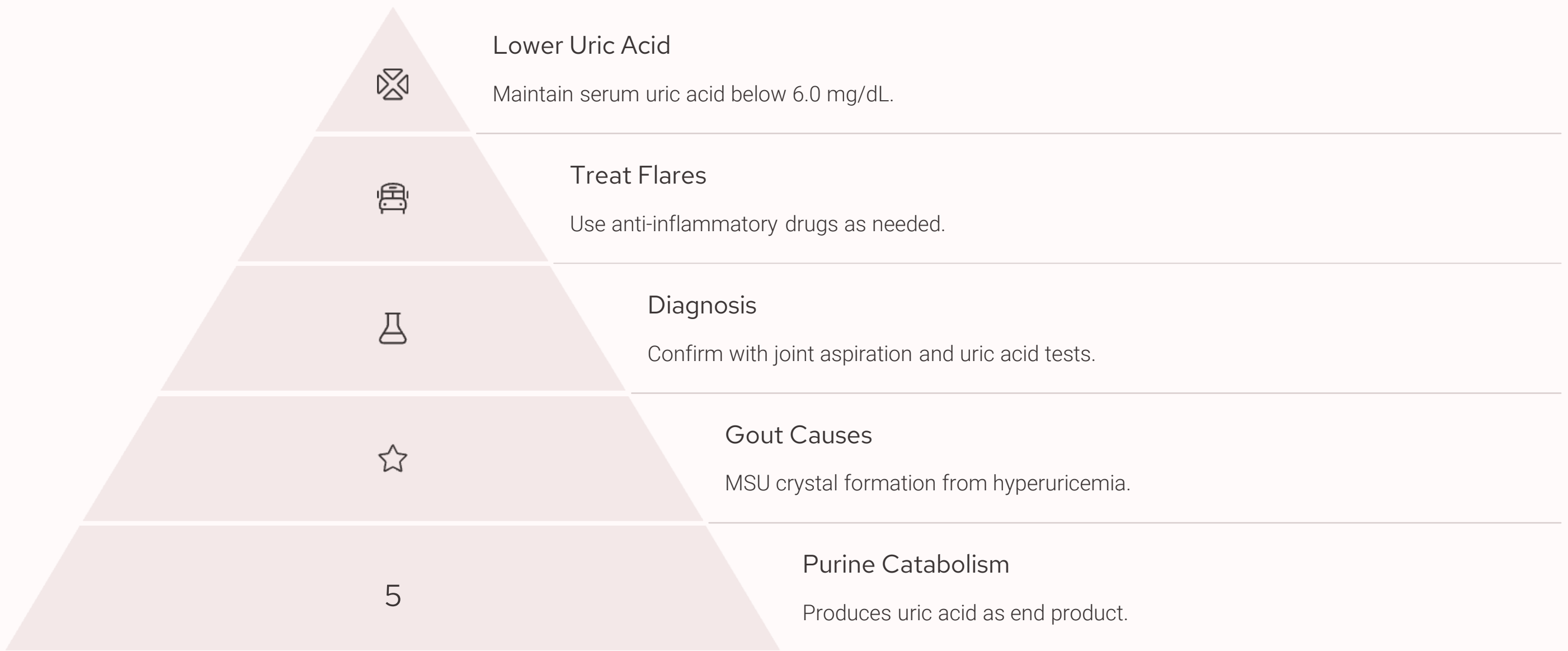
Probenecid helps kidneys remove more uric acid.

Refractory Gout

Pegloticase is used for severe, resistant cases.

Treatment combines lifestyle changes with medication. The goal is to reduce uric acid levels. This prevents future gout attacks and associated complications.

Summary: Purine Metabolism and Gout Management



Uric acid is the final product of purine catabolism. Hyperuricemia leads to MSU crystal formation and gout. Diagnosis involves joint aspiration and uric acid measurement. Treatment includes lifestyle changes, anti-inflammatory drugs, and ULT. The goal is to prevent flares and maintain low uric acid levels.