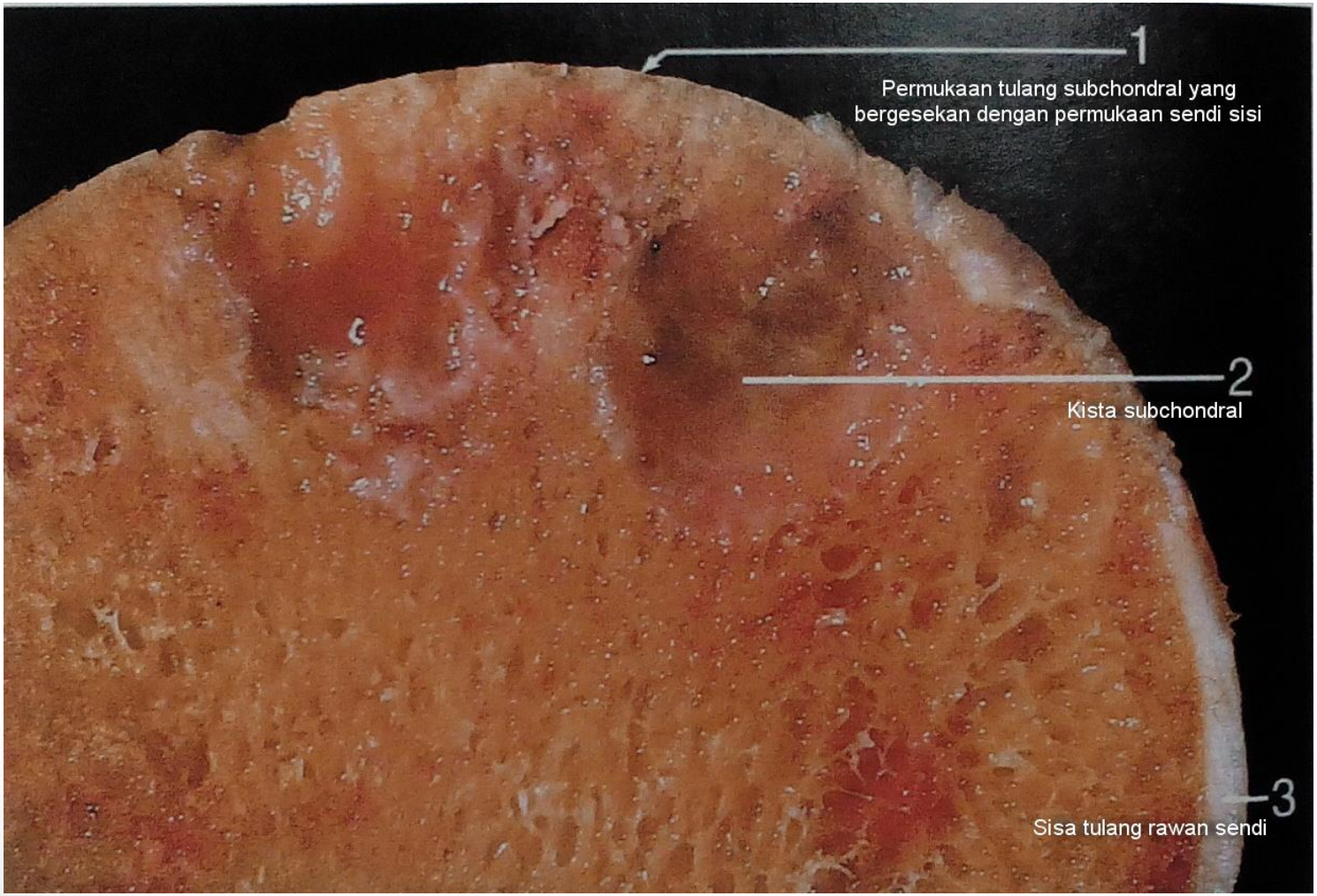
Patologi Muskuloskeletal

Osteoarthritis

SINONIM : “DEGENERATIVE JOINT DISEASE”.

BENTUK KELAINANNYA ADALAH:
TERJADI EROSI PROGRESIF PADA TULANG RAWAN SENDI.

Osteoarthritis



PROSES KELAINAN BERPUSAT PADA CHONDROSIT DALAM TIGA FASE YAITU :

1. CHONDROSIT MENGALAMI JEJAS TERKAIT DENGAN USIA, FAKTOR GENETIK DANFAKTOR BIOKEMIS.

2. PADA AWAL **OA**, CHONDROSIT MENGALAMI PROLIFERASI DAN MENGHASILKAN MEDIATOR, KOLAGEN, PROTEOGLIKAN DAN PROTEASE YANG MEMBENTUK MATRIKS TULANG RAWAN DAN MENGINISIASI PERUBAHAN PROSES RADANG SEKUNDER DI SYNOVIUM DAN TULANG SUBCHONDRAL.

3. SELANJUTNYA PADA FASE LANJUT, DENGAN BERULANGNYA PROSES RADANG KRONIK MENYEBABKAN CHONDROSIT "LEPAS", TULANG RAWAN "HILANG" DAN SECARA EKSTENSIF DIGANTI TULANG SUBCHONDRAL.

PROSES TERJADINYA KISTA SUBCHONDRAL :

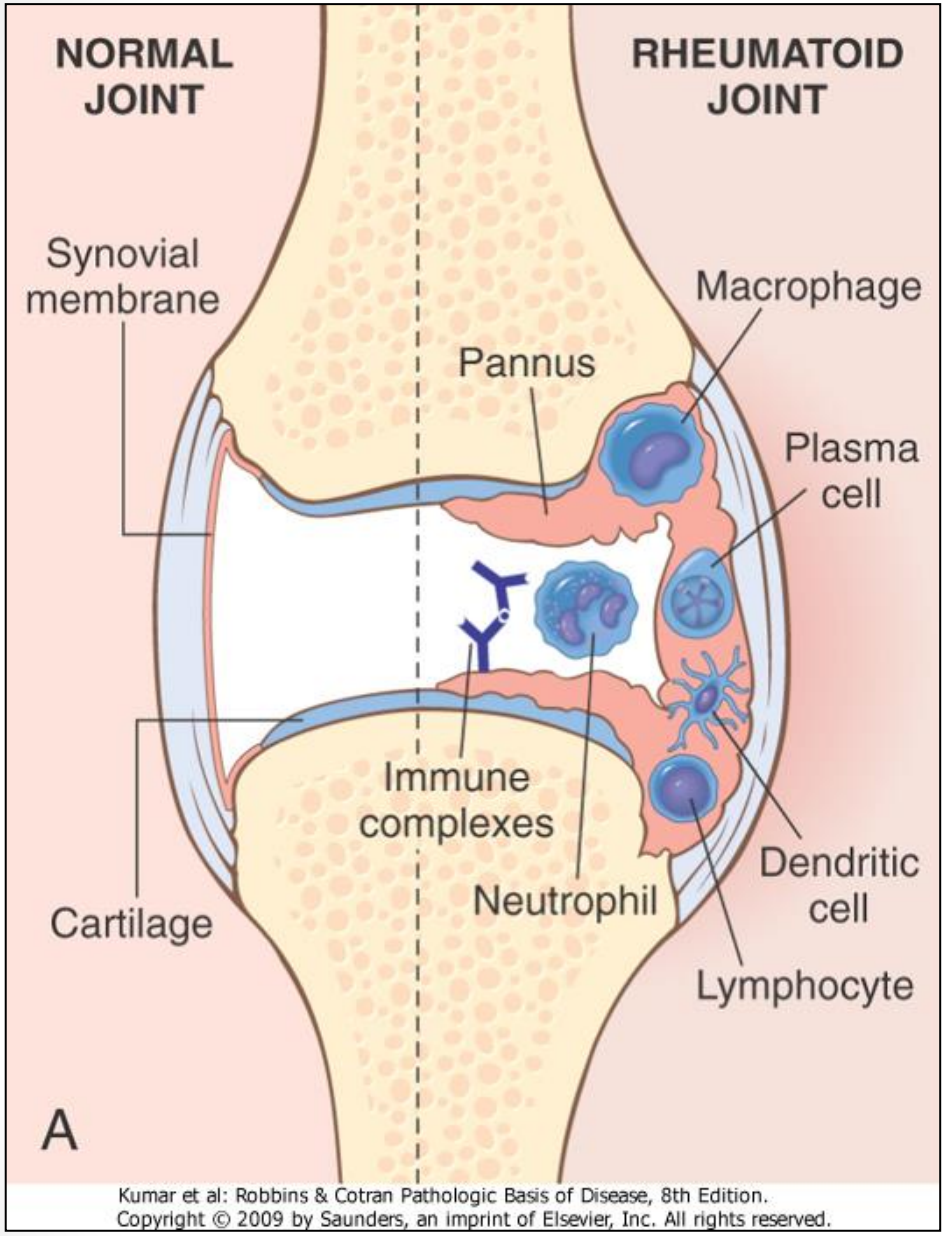
PADA **OA** YANG BERAT,
DIJUMPAI KISTA YAITU RONGGA DI TULANG SUBCHONDRAL.
BERISI CAIRAN SINOVIAL, DENGAN DINDING JARINGAN FIBROUS.

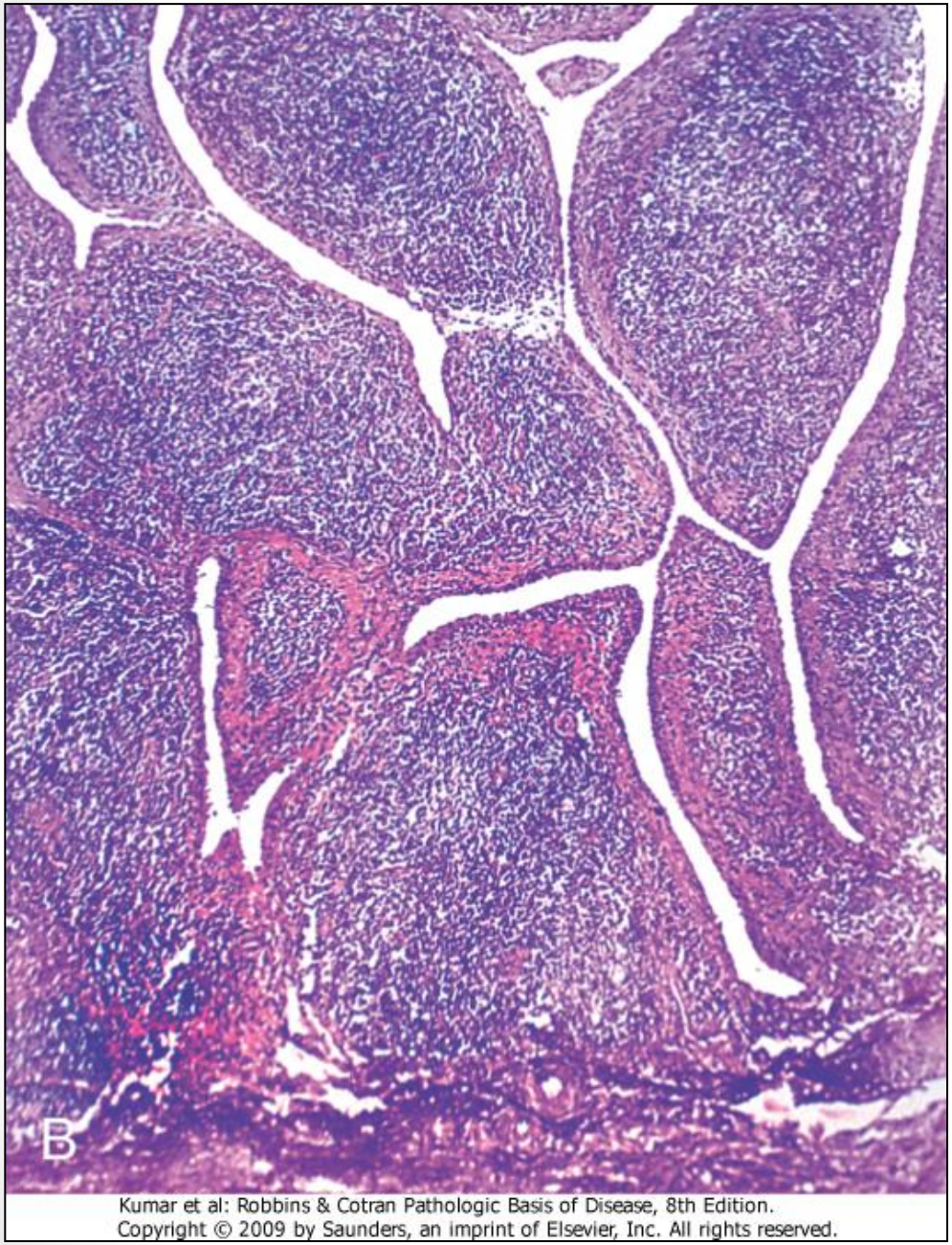
KISTA TERBENTUK KARENA TERJADI FRAKTUR KECIL-KECIL PADA TULANG SENDI, DAN “CELAH” DI ANTARA TULANG YANG MENGALAMI FRAKTUR, MENYEBABKAN CAIRAN SINOVIAL MASUK KE DALAM REGIO SUBCHONDRAL. SEDIKIT DEMI SEDIKIT AKHIRNYA BERTAMBAH BANYAK MEMBENTUK DINDING FIBROUS.

RHEUMATOID arthritis

ADALAH KELAINAN RADANG SISTEMIK KRONIS, PADA BANYAK JARINGAN DAN ORGAN - KULIT, PEMBULUH DARAH, JANTUNG,PARU DAN OTOT.

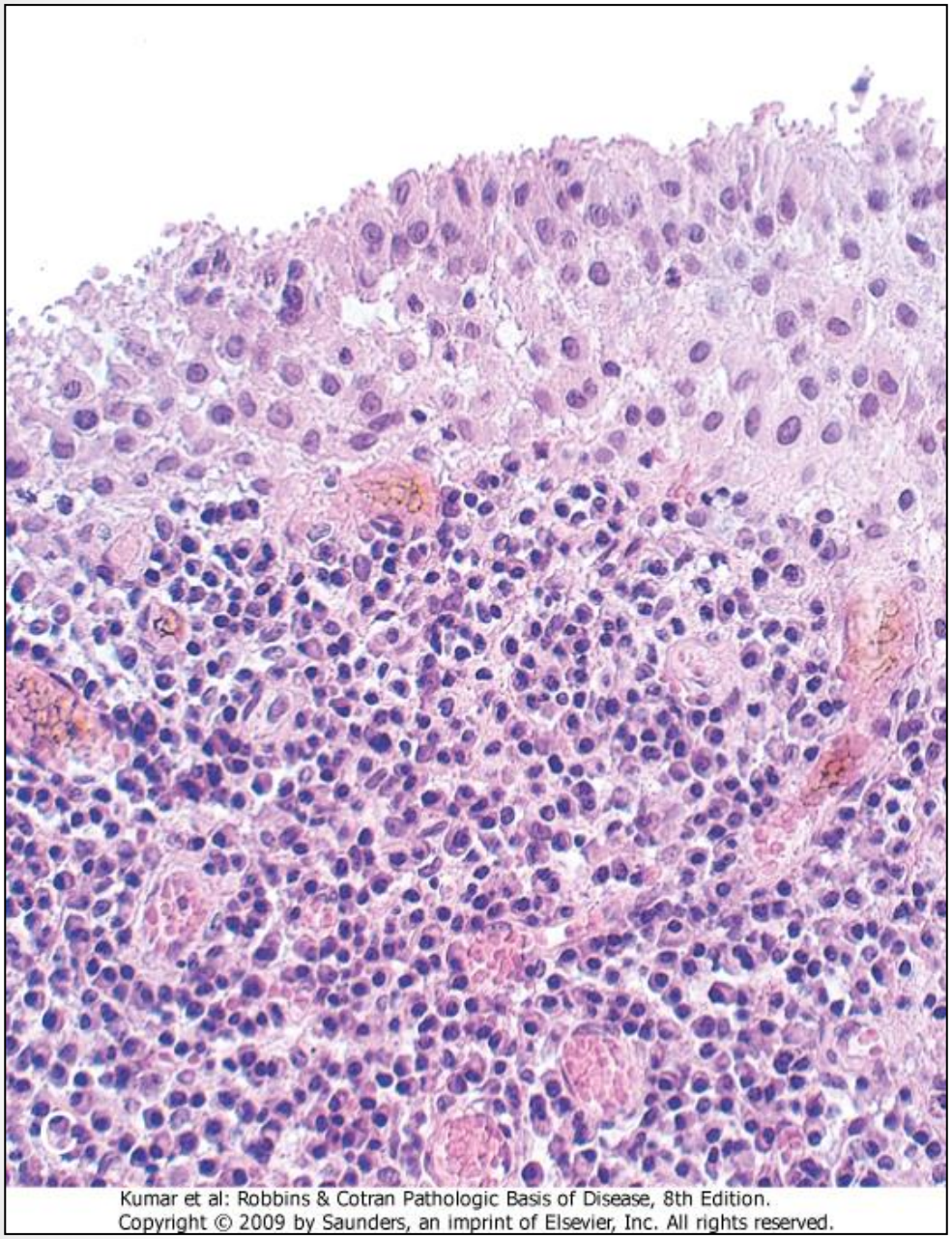
TETAPI .SECARA PRINSIP MENGENAI SENDI YAITU SEBAGAI RADANG SINOVITIS NON SUPPURATIF, PROLIFERATIF YANG MENYEBABKAN KERUSAKAN TULANG RAWAN DAN TERJADI ANKILOSIS SENDI.





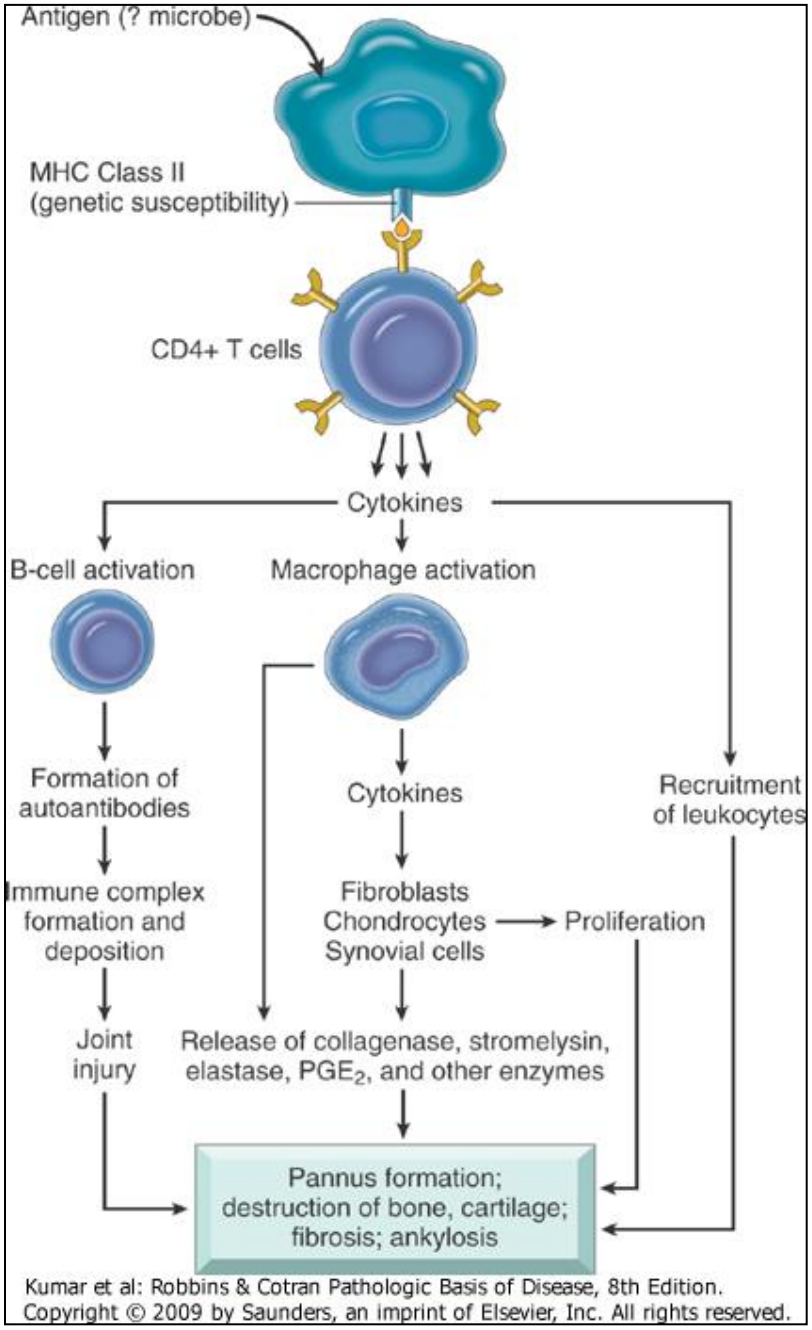
Rheumatoid arthritis.

DENGAN PEMBESARAN KECIL, TAMPAK HIPERTROFI SINOVIAL DENGAN PEMBENTUKAN VILLI.

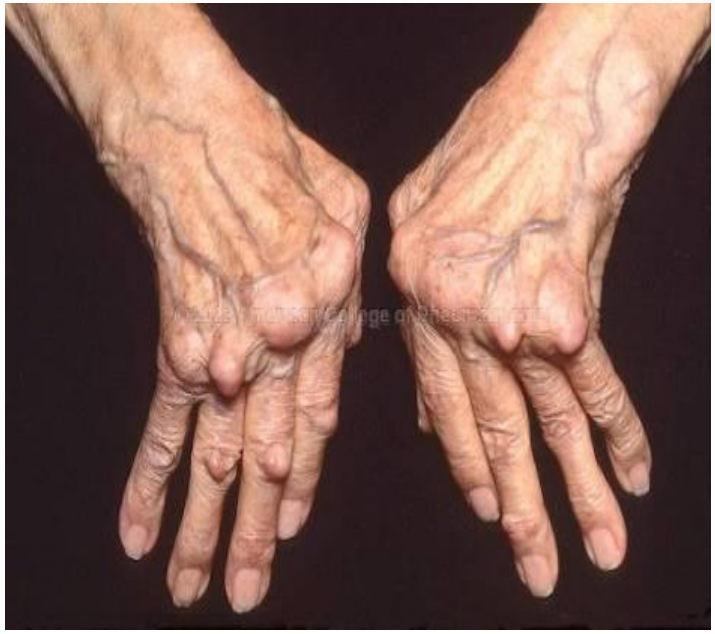


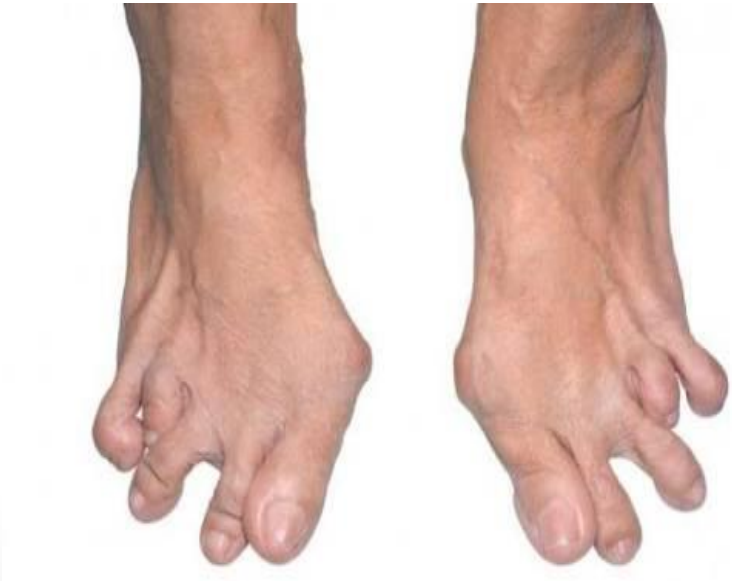
Rheumatoid arthritis.

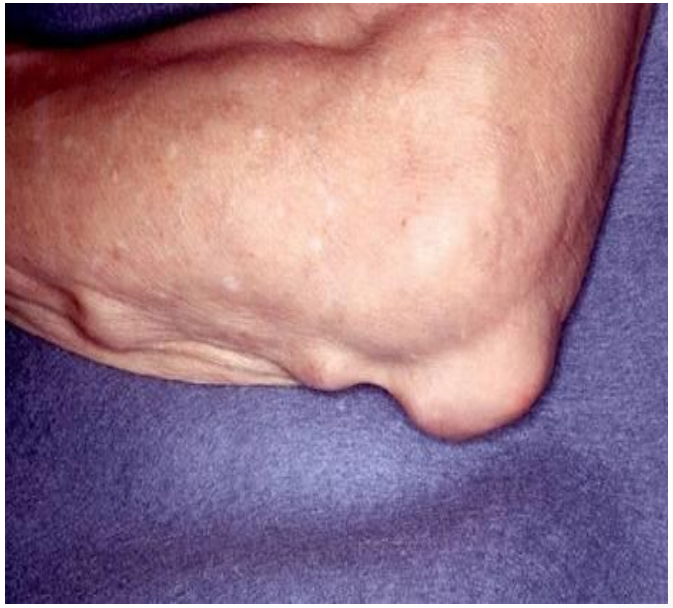
DENGAN PEMBESARAN BESAR, JARINGAN SUBSINOVIAL TERDIRI ATAS AGREGASI SEL RADANG KRONIK



Contoh Sendi yang mengalami artritis reumatoid







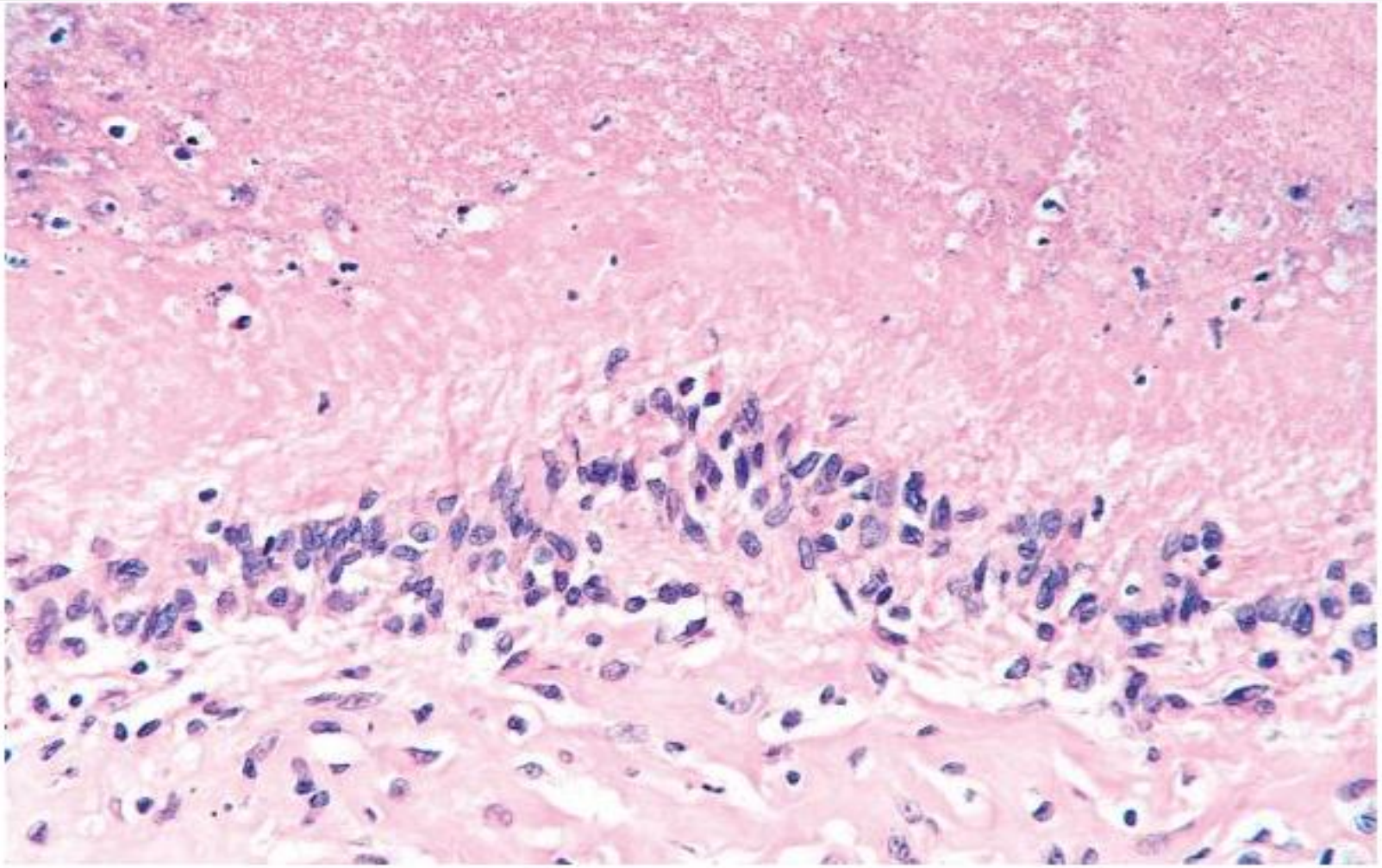


FIGURE 26-43 Subcutaneous rheumatoid nodule with an area of necrosis *(top)* surrounded by a palisade of macrophages and scattered chronic inflammatory cells.

GOUT arthritis

GOUT ADALAH KELAINAN YANG DITANDAI ADANYA SERANGAN YANG SIFATNYA "TRANSIENT"(TIDAK MENETAP) DALAM BENTUK ARTHRITIS AKUT YANG DIPICU KRISTALISASI ASAM URAT DI DALAM SENDI.

Hyperuricemia (plasma urate level > 6.8 mg/dL)

PERJALANAN PENYAKIT SELANJUTNYA DAPAT MENJADI ARTHRITIS GOUT KRONIS YANG DITANDAI ADANYA BENTUKAN "TOPHI"

PRIMARY GOUT (90% OF CASES)

- Overproduction of uric acid
  - Diet
  - Unknown enzyme defects (80% to 90%)
  - Known enzyme defects (e.g., partial HGPRT deficiency, rare)
- Reduced excretion of uric acid with normal production

SECONDARY GOUT (10% OF CASES)

- Overproduction of uric acid with increased urinary excretion
  - Increased nucleic acid turnover (e.g., leukemias and other aggressive neoplasms)
  - Inborn errors of metabolism (e.g., complete HGPRT deficiency)
- Reduced excretion of uric acid with normal production
  - Chronic renal disease

Many factors contribute to the conversion of asymptomatic

hyperuricemia into primary gout:

1. *Age* of the individual and *duration of the hyperuricemia*. Gout rarely appears before 20 to 30 years of hyperuricemia
2. *Genetic predisposition*. In addition to the well-defined X-linked abnormalities of HGPRT, primary gout follows multifactorial inheritance & runs in families
3. Heavy *alcohol* consumption: gouty arthritis
4. *Obesity* increases the risk of asymptomatic gout
5. Certain *drugs* (e.g., thiazides) reduce excretion of urate → gout
6. *Lead toxicity* increases the tendency to develop saturnine gout

Pathogenesis:

- Uric acid is the end product of purine metabolism.
- Two pathways are involved in purine synthesis:

  (1) a de novo pathway in which purines are synthesized from non-purine precursors

  (2) a salvage pathway in which free purine bases derived from the breakdown of nucleic acids of endogenous or exogenous origin are recaptured (salvaged)

- The enzyme hypoxanthine guanine phosphoribosyl transferase (HGPRT) is involved in the salvage pathway
- A deficiency of this enzyme → increased synthesis of purine nucleotides through the de novo pathway and hence increased production of uric acid
- A complete lack of HGPRT occurs in the uncommon X-linked *Lesch-Nyhan syndrome*

Metabolisme purin

ASAM URAT ADALAH PRODUK AKHIR PADA METABOLISME PURIN

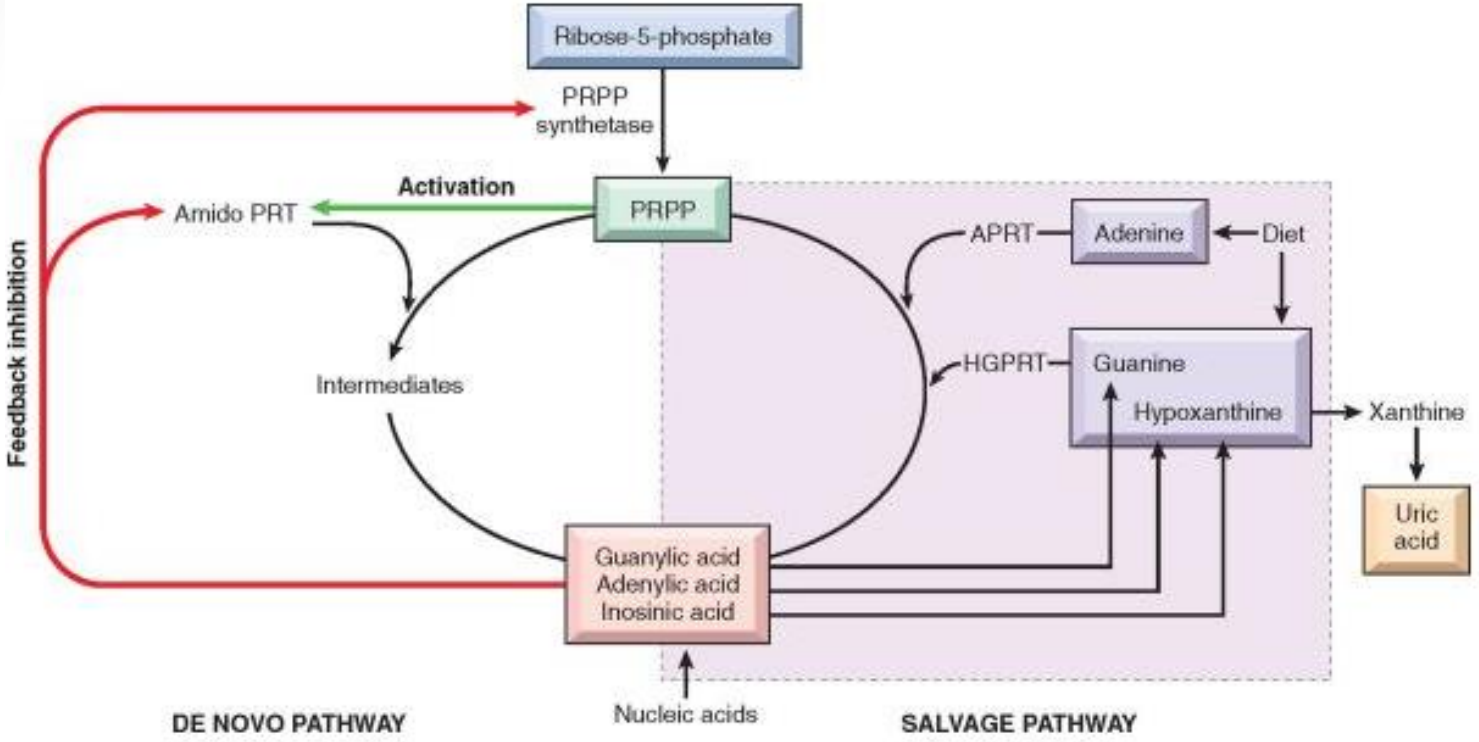


FIGURE 26-46 Purine metabolism. The conversion of PRPP to purine nucleotides is catalyzed by amido-PRT in the de novo pathway and by APRT and HGPRT in the salvage pathway. APRT, adenosine phosphoribosyltransferase; HGPRT, hypoxanthine-guanine phosporibosyltransferase; PRPP, phosphoribosyl pyrophosphate; PRT, phosphoribosyltransferase.

Clinical Course

The natural history of gout is said to have four stages:

(1) *asymptomatic hyperuricemia*, (2) *acute gouty arthritis*,

(3*) intercritical gout*, (4) *chronic tophaceous gout*

- *Asymptomatic hyperuricemia:* puberty in males & after menopause in females
- *Acute arthritis:* sudden onset of excruciating joint pain → localized hyperemia, warmth, & exquisite tenderness
- Most first attacks are monoarticular; 50% occur in the first metatarsophalangeal joint
- About 90% of affected: insteps, ankles, heels, knees, wrists, fingers, & elbows
- About 20% of those with chronic gout die of renal failure

Patogenesis Gout Arthritis Akut

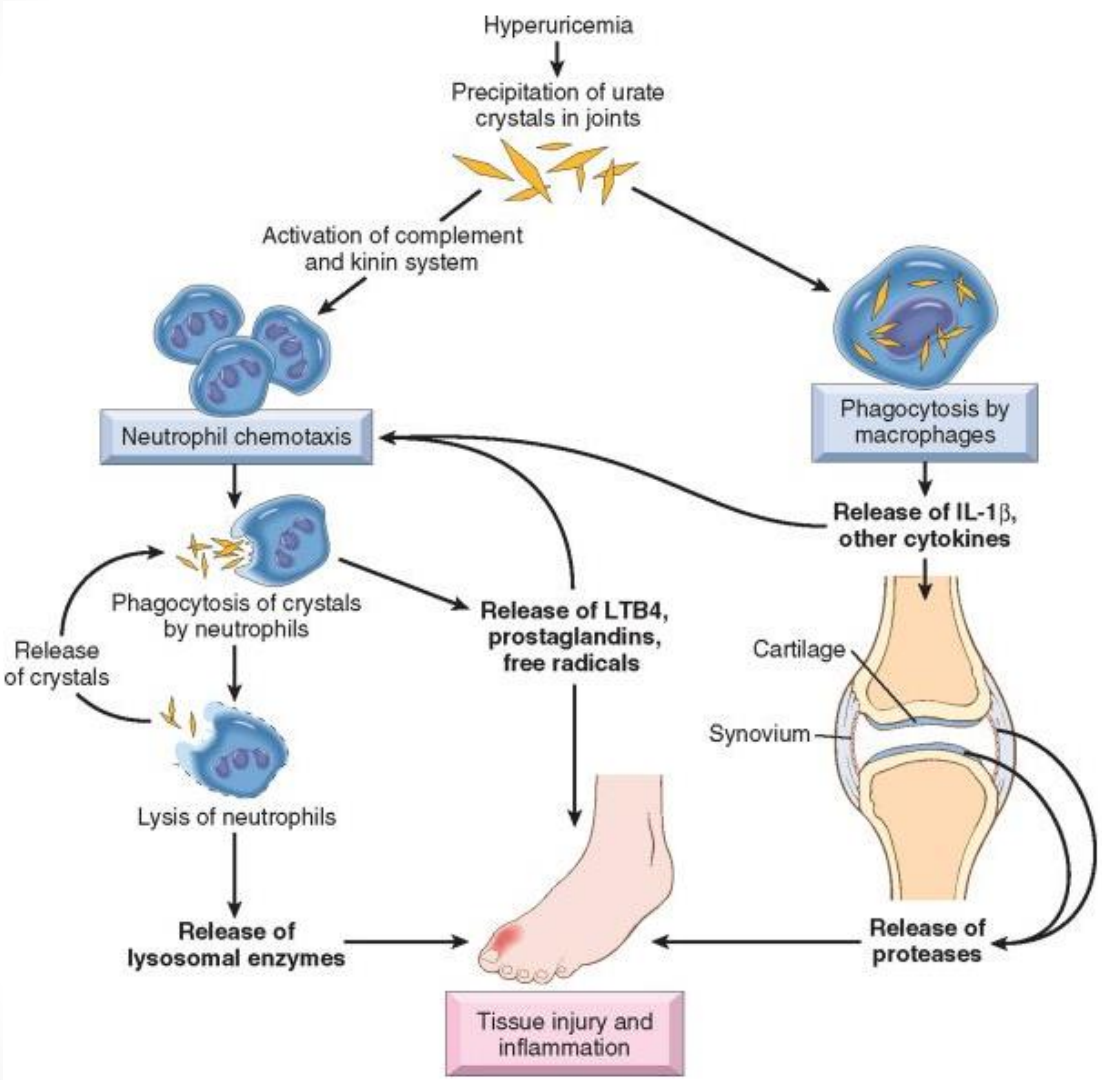
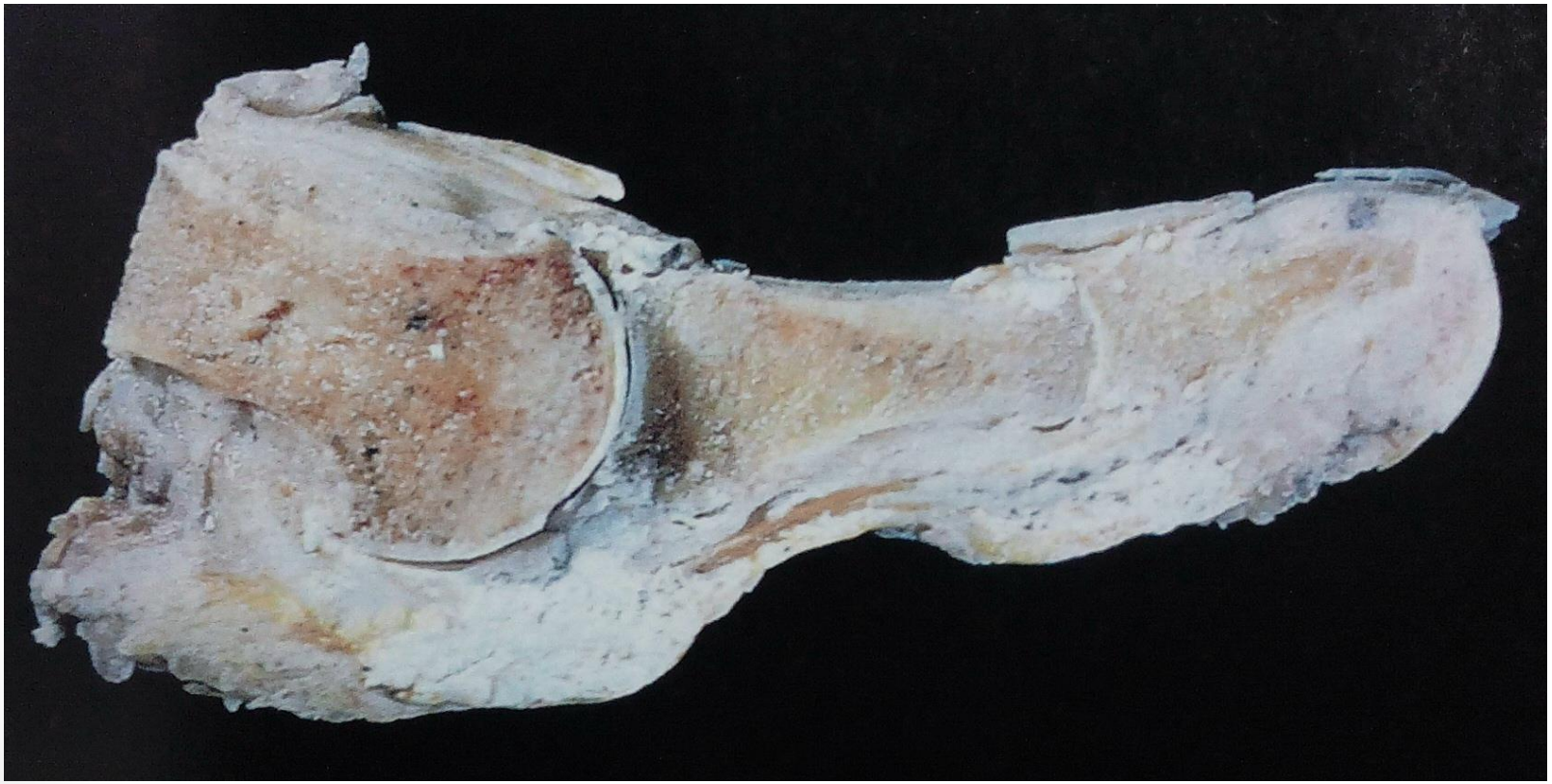


FIGURE 26-47 Pathogenesis of acute gouty arthritis. LTB4, leukotriene B4.



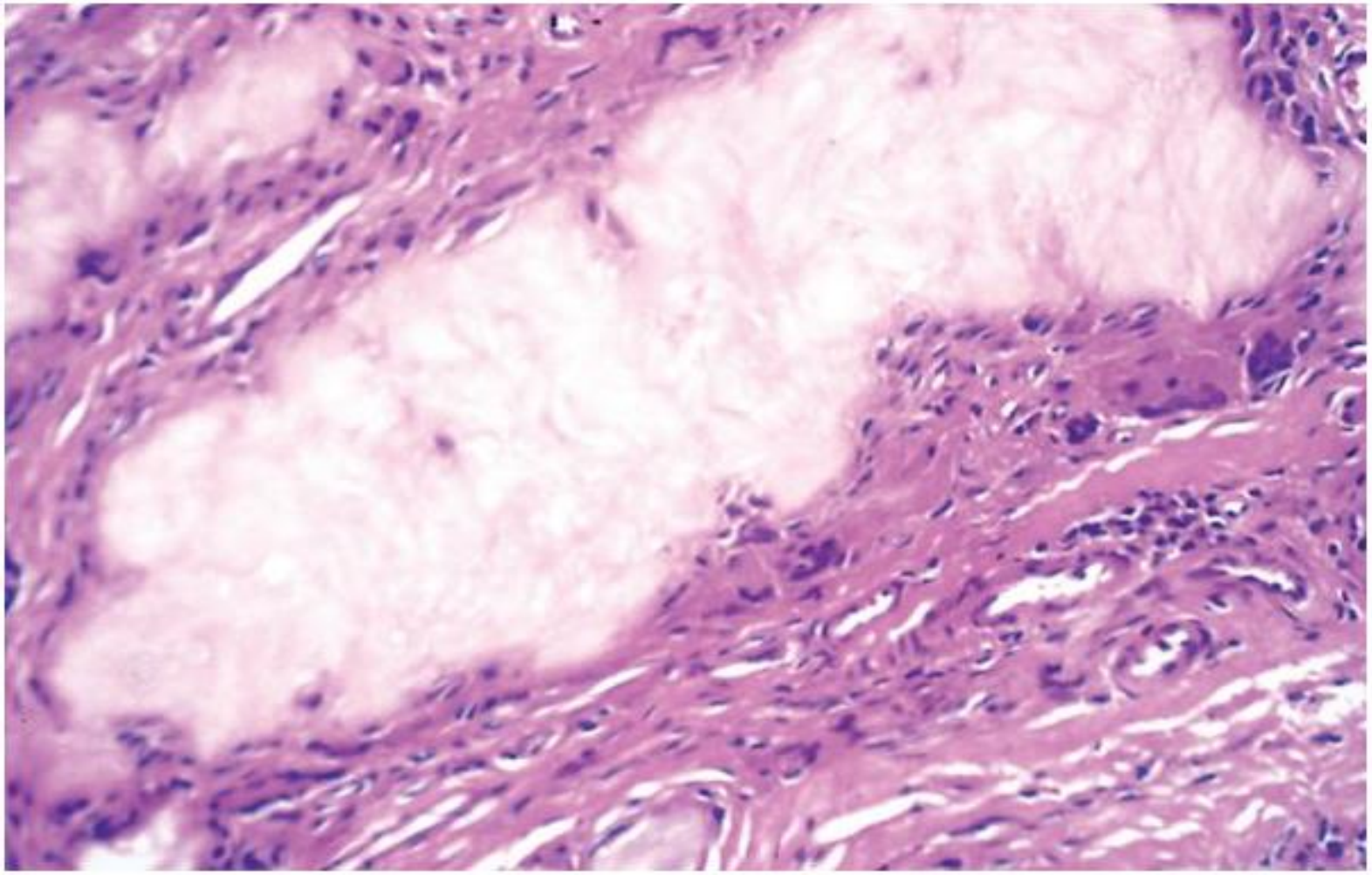


FIGURE 26-49 Photomicrograph of a gouty tophus. An aggregate of dissolved urate crystals is surrounded by reactive fibroblasts, mononuclear inflammatory cells, and giant cells.