PIPADOR REVISION

MRCOG Part 1 Revision Notes

Inflammation

PATHOLOGY • LECTURE 309

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | PATHOLOGY

309 Inflammation

Acute inflammation, mediators, outcomes, chronic and granulomatous inflammation and systemic effects

5 cardinal signs	Neutrophils	Macrophages	IL-1, IL-6, TNF-α
Rubor, calor, tumor, dolor, functio laesa	Acute (6–24 h)	Chronic; granulomas	Systemic effects

■ KEY FACTS: Learning objectives

- Describe the vascular and cellular events of acute inflammation and its mediators.
- Describe outcomes of acute inflammation, chronic and granulomatous inflammation.
- Describe systemic effects and O&G examples of inflammatory disease.

309.1 Acute inflammation

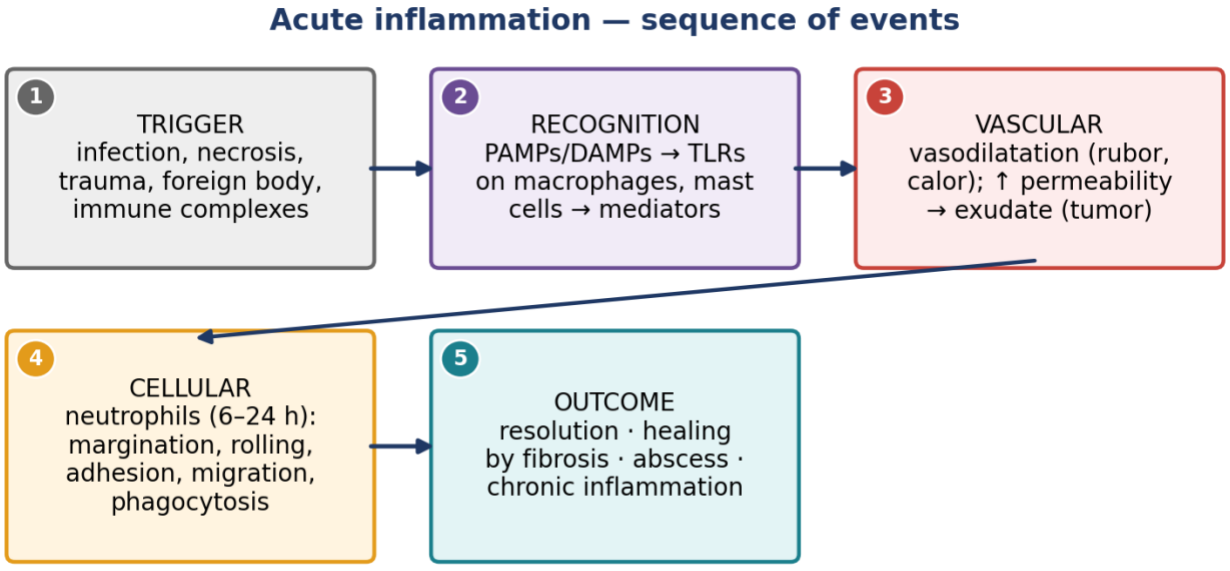


Figure 309.1 — Sequence of acute inflammation

Mediator	Source	Actions
Histamine, serotonin	Mast cells, platelets	Immediate vasodilatation and ↑ permeability
Prostaglandins (PGE_2, PGI_2)	COX pathway	Vasodilatation, pain, fever (NSAIDs block)
Leukotrienes (LTB_4, C_4–E_4)	5-LOX pathway	Neutrophil chemotaxis (LTB_4); permeability, bronchospasm
Cytokines (TNF-α, IL-1, IL-6), chemokines (IL-8)	Macrophages, endothelium	Endothelial activation, fever, acute-phase response, neutrophil recruitment
Complement (C3a, C5a)	Plasma	Chemotaxis (C5a), anaphylatoxins, opsonisation (C3b)
Bradykinin	Kinin system	Pain, permeability
Nitric oxide	Endothelium, macrophages	Vasodilatation, microbial killing

- Exudate (protein-rich, high specific gravity) differs from transudate (low protein — heart failure, hypoalbuminaemia).
- Purulent (suppurative) inflammation forms pus
- Fibrinous inflammation coats serosal surfaces (fibrinous pericarditis, peritoneal adhesions after surgery or PID).

309.2 Outcomes and chronic inflammation

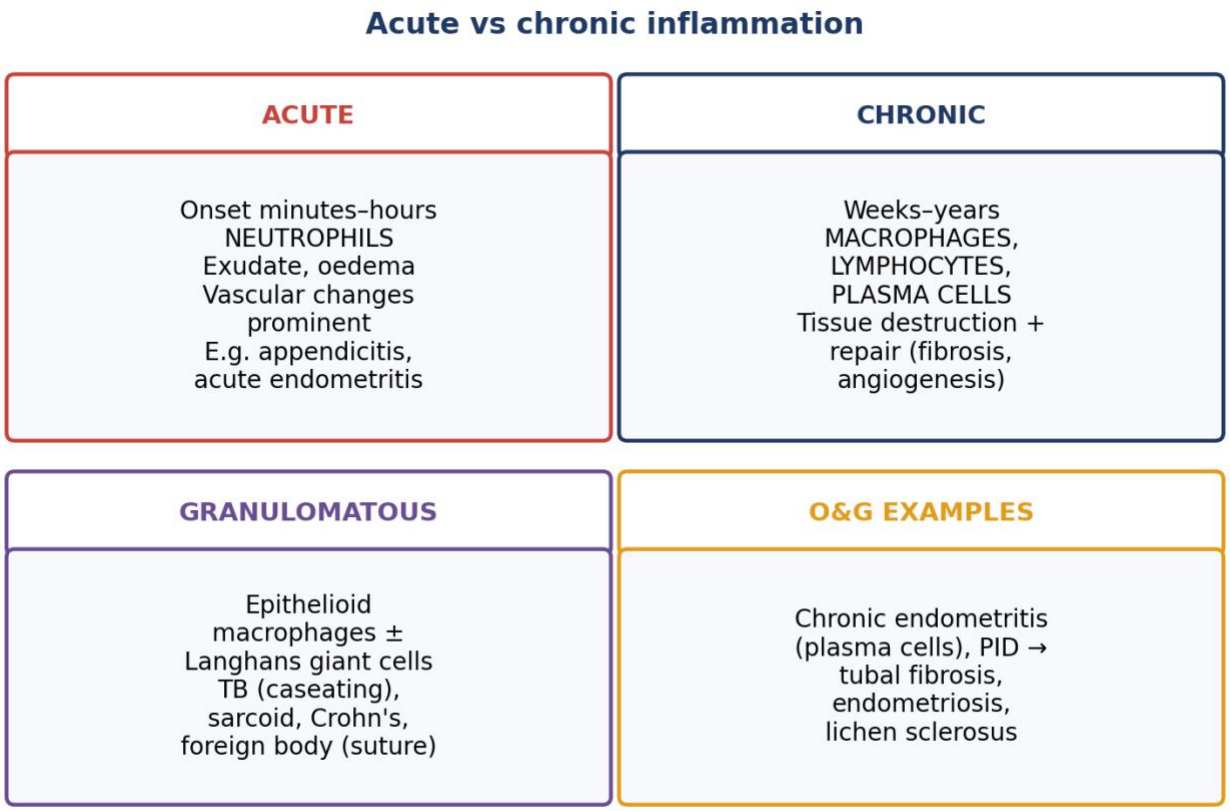


Figure 309.2 — Acute, chronic and granulomatous inflammation

- **Resolution** — clearance of debris, restoration of normal tissue (lobar pneumonia).
- **Repair by fibrosis** — tissue destruction or persistent injury (tubal adhesions after PID, Asherman's syndrome).
- **Abscess** — localised pus (tubo-ovarian, Bartholin's, breast).
- **Chronic inflammation** — persistent infection, autoimmunity, foreign material, endometriosis.

309.3 Systemic effects

- Fever (IL-1, TNF → hypothalamic PGE_2)
  - Leucocytosis (left shift)
  - Acute-phase proteins (IL-6 → CRP, fibrinogen, ferritin, hepcidin — anaemia of chronic disease), raised ESR.
- Sepsis and multi-organ dysfunction when the response is dysregulated (Lecture 307).
- Chronic inflammation → amyloidosis (AA), cachexia; inflammation also drives labour and preterm birth.

309.4 Must-know summary

■ EXAM TIP

- Acute: vascular (dilatation, permeability) + cellular (neutrophils: margination, rolling, adhesion, migration, phagocytosis).
- Mediators: histamine, PGs, leukotrienes, cytokines, complement, bradykinin, NO.
- Chronic: macrophages, lymphocytes, plasma cells, fibrosis; granulomatous: epithelioid macrophages, giant cells (TB, sarcoid, Crohn's, suture).
- Outcomes: resolution, fibrosis, abscess, chronicity.

■ MNEMONIC / MEMORY AID

Neutrophil recruitment — 'Marginate, Roll, Stick, Squeeze, Eat'.