🫁 ACUTE LUNG INJURY (ALI) & ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

🔹 Definition

Acute Lung Injury (ALI):

Sudden onset of hypoxemia and bilateral pulmonary edema not caused by cardiac failure (noncardiogenic pulmonary edema).

Acute Respiratory Distress Syndrome (ARDS):

Severe form of ALI, defined as:

- Respiratory failure within 1 week of a known clinical insult
- Bilateral opacities on chest imaging
- Not fully explained by pleural effusions, atelectasis, cardiac failure, or fluid overload
- Graded by severity of hypoxemia

🧠 *Exam Pearl:* Do not confuse with neonatal respiratory distress syndrome (surfactant deficiency in preterm infants).

🔹 Causes / Triggers of ARDS

Cause	Approximate %
Pneumonia	35–45%
Sepsis	30–35%
Aspiration	—
Trauma (brain injury, abdominal surgery, multiple fractures)	—
Pancreatitis	—
Transfusion reactions	—

COVID-19 pneumonia	Subset progresses to ARDS

🎯 *Key Concept:* All triggers share extensive alveolar injury via inflammation.

🔹 Pathogenesis

Initial insult (infection, sepsis, trauma, aspiration) → Release of proinflammatory cytokines (IL-1, TNF-α) → Endothelial activation → Sequestration & activation of neutrophils in pulmonary capillaries → Neutrophils release reactive oxygen species (ROS), proteases, and inflammatory mediators → Damage to alveolar epithelium & endothelium

- Increased vascular permeability → pulmonary edema
- Loss of surfactant → alveolar collapse & stiff lungs
- Impaired gas exchange → hypoxemia

💡 *Exam Insight:* Severity depends on balance between destructive factors (neutrophils, ROS) and protective factors (antiproteases, antioxidants).

🔹 Morphology

Gross Appearance

- Dark red, firm, airless, heavy lungs
- Bilateral involvement

Microscopic Features

- Capillary congestion
- Necrotic alveolar epithelial cells
- Interstitial & intra-alveolar edema and hemorrhage
- Neutrophilic collections (especially in sepsis)
- Hyaline membranes lining alveolar ducts (fibrin-rich edema + necrotic epithelial remnants)

Organizing Stage

- Type II pneumocyte proliferation → alveolar lining regeneration
- Often incomplete resolution → fibrosis & septal thickening

🧠 *Exam Tip:* Hyaline membranes are hallmark of ARDS, resembling neonatal RDS.

🔹 Clinical Features

- Develops in ~85% of cases within 72 hours of insult
- Presents as severe hypoxemia and dyspnea
- Imaging: bilateral ground glass opacities on CT / X-ray
- High-risk patients: elderly, sepsis, multiorgan failure

Prognosis

- Mortality: ~40% (mostly from underlying condition or infection)
- Respiratory failure rarely sole cause of death

- Survivors → often reduced physical endurance due to persistent lung function abnormalities

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🔹 Summary Table: ALI vs ARDS

Feature	ALI	ARDS
Onset	Abrupt	Abrupt (severe ALI)
Pulmonary edema	Noncardiogenic	Noncardiogenic
Hypoxemia	Mild	Moderate to severe
Chest imaging	Bilateral infiltrates	Bilateral infiltrates
Mortality	Lower	~40%
Histology	Diffuse alveolar damage	Diffuse alveolar damage with hyaline membranes

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🔹 Exam Pearls 🎯

- Hyaline membranes = hallmark
- Most common triggers: pneumonia & sepsis
- ARDS occurs within 1 week of insult
- Noncardiogenic pulmonary edema
- Neutrophils play central role in pathogenesis
- Survivors may have persistent lung stiffness & reduced exercise tolerance

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🔹 Flowchart for ARDS Pathogenesis

Clinical insult (sepsis / pneumonia / trauma / aspiration)
→ ↑ Proinflammatory cytokines (IL-1, TNF-α) →
Neutrophil recruitment & activation → ROS & protease release → Alveolar epithelial & endothelial injury

- ↑ Vascular permeability → Pulmonary edema
- Surfactant loss → Alveolar collapse
- Hypoxemia & stiff lungs

-> The End <-