ALI/ARDS

Terrence Shenfield BS, RRT, RPFT, NPS, AE-C
Clinical Educator
A & T Lectures
Acute Respiratory Distress Syndrome

- An acute inflammatory condition in the lungs due to an underlying disease process or trauma
- 150,000 cases each year
- 40% mortality
- Common causes are sepsis or trauma
  - Acute onset of respiratory failure
  - Bilateral infiltrates on frontal chest radiograph
  - Absence of left ventricular failure (rule out cardiogenic pulmonary edema)
  - Hypoxemia with a ratio of PaO2/FiO2<200
ALI

- Same criteria as ARDS
- $\text{PaO}_2/\text{FiO}_2 < 300\text{mmHg}$
ALI/ARDS

- Two types “primary” and “secondary”
- Direct (Primary) Injury
  - 70-80% cases
  - Pneumonia the most common 40-50%
  - Aspiration
  - Pulmonary contusion (chest trauma)
  - Inhalation of toxic gases
  - Near drowning
ALI/ARDS

- Indirect (Secondary) Injury (20%-30% of cases)
- Typically caused by systemic inflammation with generalized activation of mediators
  - Infection (sepsis, peritonitis)
  - Tissue Ischemia (necrosis, pancreatitis)
  - Tissue damage (trauma, CABG, post-op, some intoxications)
ALI/ARDS

- Independent Risk Factors
  - Old age
  - Infection
  - Neurological disease
Clinical Picture

- Cyanosis
- Tachypnea
- Dyspnea
- Crackles
- Extra-pulmonary manifestations of underlying disease
  - Inflammatory cascade
ALI/ARDS 3 Phases

- Initial acute phase (days)
  - Noncardiogenic pulmonary edema secondary to capillary leakage
- Second phase (1-2 weeks)
  - Inflammatory
  - Organization of edema into hyaline membranes
- Third phase
  - Fibrosis of pulmonary tissue
ALI/ARDS

- With time can progress to fibrosing alveolitis
- Lung compliance is reduced and hypoxaemia persists
- Pulmonary hypertension can progress to right heart failure
- Resolution can occur over 6-12 months
- Lung function can return to normal
- Overall mortality is approximately 50%
ALI/ARDS
Pulmonary Insult

- Interstitial and alveolar edema
- Increase extravascular lung water
- Increased stiffness and weight of lungs
- Compression atelectasis
- Reduction of compliance
- Decrease of functional residual capacity
- Increase intrapulmonary shunting
Diagnostic Workup

- **Medical History**
  - Acute onset of respiratory failure in combination with an underlying insult that initiates the pulmonary inflammation

- **CXR**
  - Bilateral infiltrates

- **Echocardiography or PA catheterization**

- Documented PCWP <18mmHg
  - Exclusion of significant LV dysfunction (this rules out cardiogenic pulmonary edema)
Diagnostic Workup

- ABG and FiO2

- PaO2/FiO2 <200 (ARDS)
- <300 ALI
- Hypoxemia
- Hypocapneic
  - Anxiety and increased ventilatory drive
  - Respiratory acidosis typically follows
Early Management

- Ventilatory support
- ?? NIV
  - Fully conscious
  - Cooperative
  - Hemodynamically stable
  - Tolerant of short periods off vent support
  - Able to take large breaths
  - Terminate if no marked improvement in 1 hour
Management

- Vast Majority of patients will require mechanical ventilation
  - Inadequate gas exchange due to flooded alveolar
- Potential Complications from intubation
  - Prolonged hypoxemia
  - Aspiration
  - Misplacement of tube
  - Hemodynamic compromise
Case Study Scenario

- 40 yo HIV+ patient with bilateral interstitial infiltrates on CXR transferred to the ICU on face mask of 10L/min
- PCP suspected ----on Bactrim and prednisone
- BP 112/70  HR 110  O2 Sat 94% RR 28
- 2 hours later patient noted to be lethargic with O2 Sat 77%
Case Study Scenario

- The fellow successfully intubates the patient
- Vent Settings
  - A/C 500/14/5 FiO2 100%
- ABG
  - 7.24/42/60 on 100%
  - PaO2/FiO2 = 60
- What do we have? ALI or ARDS? Why?
Management

“Open up the lung and keep the lung open”
- Lachmann and Johnson Intensive Care Med 1992

During the early phase of ALI/ARDS opening up lung compressed by the weight of inflammatory exudate improves arterial oxygenation

Attempting this during later phases of ARDS when collapsed lung has becomes fibrotic is difficult
Recruitment Maneuvers

- An administration of high ventilation pressure to a patient for a brief period of time
- Aim is to reinflate collapsed lung tissue, thus “recruiting” that tissue
- Aim is also to prevent “de-recruitment” by applying PEEP after the maneuvers
- The desired outcome is improved oxygenation
- There are specific indications
Contraindications for RM

- Hemodynamic compromise:
  - Recruitment maneuvers cause a transient loss of venous return, compromising cardiac output.
- Existing barotrauma
- Increased intracranial pressure
- Predisposition to barotrauma:
  - Apical bullous lung disease
  - Focal lung pathology eg. lobar pneumonia
Recruitment Maneuver

- Manual Hyperinflation
  - Clamp the ETT at end inspiration
  - Attach a bag valve with a PEEP valve
  - Bag patient at a PEEP of 15-20 cm H2O for 20-30 sec
  - This is performed 2-3 times then patient is placed back on the vent with a PEEP of 15-20
Recruitment Maneuver

- Prolonged Inspiratory pause hold
- Total inspiratory pressure increased to 40cm H2O after which an inspiratory hold of 40 sec is applied
- This is repeated 3 times
Recruitment Maneuver

- Continuous Positive Airway Pressure
  - Increase pressure support to 40cm H2O for 40 seconds
  - Allow the patient to be ventilated with PEEP 18-20cm H2O
  - This should be performed twice then place patient back on normal vent mode with PEEP 15-20
Pre and post recruitment maneuvers
Ventilator Mode

- No proven mode conclusively proven superior in ARDS
- ARDSnet protocol
- APRV
- Adult HFOV
- Nitric oxide
- Prone positioning
APRV Vs. ARDSnet Protocol Ventilation For ARDS/ALI

- Open lung ventilation strategy
- Minimize VILI
- Keep plateau pressures < 30cmH2O

Findings
- Mortality was the same
- Fewer inflammatory mediators (IL-1, TNF, cytokines), were present with APRV
Adult HFOV- Not good news!


- Both trails showed increased mortality associated with the use of HFOV

- Reason? Maybe better adherence to low tidal volume strategy
### Lower PEEP/higher FiO2

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### Higher PEEP/lower FiO₂

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Tidal Volume

- No proven mode conclusively proven superior in ARDS
- Tidal Volume 6ml/kg shown to decrease mortality (9%)
- Low tidal volume ventilation might reduce CO2 elimination – permissive hypercapnia
PEEP

- Positive End Expiratory Pressure
- Prevents collapse of open and perfused lung regions and thus maintain arterial oxygenation
- Indirectly causes high end inspiratory pressure which may open up collapsed lung improving arterial oxygenation
- Prevents cyclical opening and closing of airway
- May cause overdistension/barotrauma
Ventilator Induced Lung Injury

- VILI caused by over distension with tidal opening and closing of lung units
- Small lesions in the alveoli and airway trigger a inflammatory response
- Inflammation generalized can lead to multiple organ dysfunction, the most significant cause of death in ARDS patients
Inspiratory : Expiratory Ratio

- I:E ratio reversal
- Longer inspiratory time increases intra-alveolar mixing
  - Improves CO2 removal
- Shorter expiratory time prevents time-dependent alveolar collapse
- Inverse I:E ratio with PCV
Fluid Balance

- Positive fluid balance is common
- Fluid removal is resistant and takes long to resolve
- The use of high PEEP can cause circulatory compromise in hypovolemic patients
  - Consider fluid bolus to correct hypotension
Positioning

- Prone positioning
  - Pleural pressure more evenly distributed to dorsal regions of lung that are typically dependent
  - More homogeneous distribution of pressure
  - Improved ventilation perfusion ratio

- Oxygenation improves in 70% patients
  - Typically within minutes

- Standing
  - Improves success of gastric feeding
  - Suggested decrease in nosocomial pneumonia
Pulmonary Vasoactive Drugs

- Inhaled NO
  - Pulmonary vasodilator
  - Improves VQ ratio
  - Improves oxygenation in 60%
  - Lasts 2-3 days – no effect on outcome
Infection

- Nosocomial pneumonia is greater in ARDS patients
- VAP risk increases 3%/day in the 1st week
- VAP associated with increase mortality of 20-30%
- Prescribe appropriate antibiotic therapy if indicated
References

