OBJECTIVES

• REVIEW CLINICAL PRESENTATIONS OF SEPSIS
• DISCUSS EFFECTIVE MANAGEMENT STRATEGIES IN A PATIENT WITH SUSPECTED SEPSIS
• ANALYZE CLINICAL BIOMARKERS THAT MAY PREDICT OUTCOMES OF SEPSIS.
WHAT IS IT?

• THE SYSTEMIC RESPONSE TO INFECTION

• BACTERIA PREDATES HUMANS

• COMMON, EXPENSIVE, FATAL
SO WHAT IS SEPSIS?

• SEPSIS IS A CLINICAL SYNDROME RESULTING FROM A DYSREGULATED INFLAMMATORY HOST RESPONSE TO INFECTION

• SEPSIS RESULTS WHEN THE NORMAL HOST RESPONSE TO A LOCALIZED INFECTION BECOMES GENERALIZED AND INVOLVES NORMAL TISSUE/ORGAN SYSTEMS AWAY FROM THE SITE OF INJURY OR INFECTION

• CARRIES A HIGH MORTALITY RATE AND IS THE LEADING CAUSE OF MORTALITY DUE TO INFECTION IN HOSPITALIZED PATIENTS

• CAN BE DIFFICULT TO DISTINGUISH FROM A NORMAL INFLAMMATORY RESPONSE TO A NON-INFECTIOUS INSULT
OBLIGATORY STATISTICAL INFORMATION ABOUT SEPSIS

• LEADING CAUSE OF DEATH IN NON-CORONARY ICU PATIENTS
• CARRIES A MORTALITY RATE OF UP TO 50%
• UP TO 50% OF PATIENTS WHO SURVIVE SEPSIS SUFFER LONG-TERM COMPLICATIONS SUCH AS PERMANENT ORGAN DAMAGE, COGNITIVE IMPAIRMENT, AND PHYSICAL DISABILITY
• FOLLOWING HOSPITAL DISCHARGE, SEPSIS CARRIES AN INCREASED RISK OF DEATH, FURTHER SEPSIS, AND RECURRENT HOSPITAL ADMISSIONS WITHIN THE FIRST YEAR
• THE MOST EXPENSIVE CONDITION TREATED IN HOSPITALS, ACCOUNTING FOR OVER $20 BILLION ANNUALLY TO THE US HEALTHCARE SYSTEM
• RESPONSIBLE FOR A SIGNIFICANT INCREASE IN HOSPITAL “LENGTH OF STAYS”
The Sepsis Cascade

Activation of Coagulation and Complement System
Tissue Factor Release
Fibrinolytic Activity

Bacterial Products and Components

Macrophage

Neutrophil Activation, Aggregation, Degranulation; Release of O₂ Radicals and Proteases

Platelet Activation Aggregation

Metabolism of Arachidonic Acid, Release of Thromboxane A₂, PGS, LTS

T-Cell Release of IL-2, INF-γ, GM-CSF

Endothelial Damage

Tissue Injury

Organ Dysfunction

TNF-α
IL-1
IL-6
PAF
NO etc.
NORMAL HOST RESPONSE TO INFECTION

Immune Cells Activate
- Macrophages recognize and bind to microbial components
- Immune Cell binding to microbial components leads to

Inflammatory Response
- Polymorphonuclear leukocytes (PMNs) become active and migrate to source of infection
- PMNs release pro-inflammatory mediators to recruit more PMNs and macrophages

Anti-inflammatory Mediators Released
- Release of cytokines that inhibit release of pro-inflammatory cytokines
- Immune System suppressed as a result
Balance is the Key!

- The balance of pro-inflammatory and anti-inflammatory mediators regulate the host response to infection.

- The end result is overcoming infection, tissue repair/healing, and restoring homeostasis.
Alterations in Homeostasis leads to the clinical syndrome that is Sepsis.
TEXTBOOK DEFINITIONS/CATEGORIES

****NOT WELL DELINEATED CLINICALLY*****
CATEGORIES

• SIRS = SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
• SEPSIS
• SEVERE SEPSIS
• SEPTIC SHOCK
CRITERIA FOR DIAGNOSING SIRS

• 2 OF THE FOLLOWING:
  • TEMP >38 C OR <36 C
  • HEART RATE >90 BPM
  • RESPIRATORY RATE >20/MIN OR PACO2 <32 MM HG
  • WBC COUNT >12,000/MM3
OLD DEFINITION OF SEPSIS

• SIRS IN RESPONSE TO A CONFIRMED INFECTIOUS PROCESS
• 2-3% OF HOSPITALIZED PATIENTS
• 20% OF ICU ADMISSIONS
• CONTRIBUTES TO 30% OF DEATHS IN AMERICA
OLD DEFINITION OF SEVERE SEPSIS

• SEPSIS + ANY OF THE FOLLOWING:
  • ORGAN DYSFUNCTION
  • HYPOPERFUSION
  • HYPOTENSION
  • LACTIC ACIDOSIS
  • OLIGURIA
  • AMS
OLD DEFINITION OF SEPTIC SHOCK

- SEVERE SEPSIS +
  - SEPSIS-INDUCED HYPOTENSION AND LOW PERFUSION DESPITE ADEQUATE FLUID RESUSCITATION
**Systemic Inflammatory Response Syndrome**
A clinical response arising from a nonspecific insult, including ≥2 of the following:
- Temperature ≥38°C or ≤36°C
- Heart rate ≥90 beats/min
- Respirations ≥20/min
- WBC count ≥12,000/mm³ or ≤4,000/mm³ or >10% immature neutrophils

**SIRS with a presumed or confirmed infection**

**Sepsis with ≥1 sign of organ failure**
- Cardiovascular (refractory hypotension)
- Renal
- Respiratory
- Hepatic
- Hematologic
- CNS
- Unexplained metabolic acidosis

**Septic Shock**
NEW DEFINITION OF SEPSIS

• SUSPECTED OR CONFIRMED SOURCE OF INFECTION +

• EVIDENCE OF ORGAN DYSFUNCTION DEFINED BY 2 OR MORE POINTS IN THE SEQUENTIAL (SEPSIS RELATED) ORGAN FAILURE ASSESSMENT (SOFA) SCORE
• Clinical Suspicion based upon presentation and signs and symptoms of infection

• Detailed H&P is a MUST!

• qSOFA score with SIRS criteria to help support clinical suspicion

• BIOMARKERS are key in differentiating an infectious process from an inflammatory response

• Early intervention and aggressive treatment are key to improving outcomes/survival
QSOFA – PREDICTS ICU MORTALITY

• 2016 SOCIETY OF CRITICAL CARE MEDICINE
• 6 ORGAN SYSTEM CATEGORIES –
  • RESPIRATORY
  • CARDIOVASCULAR
  • HEPATIC
  • RENAL
  • COAGULATION
  • NEUROLOGIC

https://www.mdcalc.com/sequential-organ-failure-assessment-sofa-score
<table>
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<tr>
<th>Parameter</th>
<th>Normal Range</th>
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<tbody>
<tr>
<td>Partial pressure of oxygen</td>
<td>Norm: 75 - 100 mm Hg</td>
</tr>
<tr>
<td>Fraction of inhaled O2</td>
<td>%</td>
</tr>
<tr>
<td>Platelet count</td>
<td>Norm: 150 - 350 x10^3/μL</td>
</tr>
<tr>
<td>Glasgow Coma Scale</td>
<td>Norm: 3 - 15 points</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>Norm: 0.3 - 1.9 mg/dL</td>
</tr>
<tr>
<td>Level of hypotension (vasopressor Status For ≥ 1 Hr)</td>
<td></td>
</tr>
<tr>
<td>No hypotension</td>
<td>0</td>
</tr>
<tr>
<td>MAP &lt; 70</td>
<td>+1</td>
</tr>
<tr>
<td>On vasopressors, dopamine &lt; 5 μg/kg/min OR dobutamine (any dose)</td>
<td>+2</td>
</tr>
<tr>
<td>Dopamine &gt; 5 μg/kg/min OR Epi/Norepi &lt; 0.1 μg/kg/min</td>
<td>+3</td>
</tr>
<tr>
<td>Dopamine &gt; 15 μg/kg/min OR Epi/Norepi &gt; 0.1 μg/kg/min</td>
<td>+4</td>
</tr>
<tr>
<td>Creatinine (or urine output, use worst value)</td>
<td></td>
</tr>
<tr>
<td>Cr&lt; 1.2 mg/dL (&lt;106 μmol/L)</td>
<td>0</td>
</tr>
<tr>
<td>Cr 1.2-1.9 mg/dL (106-168 μmol/L)</td>
<td>+1</td>
</tr>
<tr>
<td>Cr 2.0-3.4 mg/dL (177-301 μmol/L)</td>
<td>+2</td>
</tr>
<tr>
<td>Cr 3.5-4.9 mg/dL (309-433 μmol/L) OR urine output &lt; 500ml/day</td>
<td>+3</td>
</tr>
<tr>
<td>Cr &gt; 5.0 mg/dL (&gt;442 μmol/L)</td>
<td>+4</td>
</tr>
</tbody>
</table>
So, what is a biomarker??

Although no one specific biomarker has been proven to be “the definitive test for sepsis”, several have shown value in early detection and help guide response to therapy. Multiple studies have shown the importance of various biomarkers for early detection of sepsis due to an infectious process. The biomarkers **Procalcitonin and Presepsin** show good sensitivity for an early bacterial invasion and host response. Procalcitonin is used routinely as part of sepsis workup and treatment.

Specific **Toll-like receptors** (proteins on cell surfaces of macrophages) have been shown to help differential gram + organisms from gram – organisms!

Decreasing **procalcitonin** levels during treatment indicate a good host response to treatment and allow for a stop date on antibiotic use which is not only a cost-savings benefit, but can assist with the inappropriate use of antibiotics which can result in drug resistant organisms.

Elevated serum **lactate levels** (a byproduct of anaerobic respiration) of > 2 are associated with poor prognosis. And are early indicators of tissue hypoperfusion

More research is needed, but the future of biomarkers appears promising!
WHY DO WE CARE?

• SEPSIS CAN RAPIDLY PROGRESS TO SEPTIC SHOCK IF NOT AGGRESSIVELY MANAGED!

AND

• SEPTIC SHOCK CAN RAPIDLY PROGRESS TO MULTI-ORGAN FAILURE AND DEATH IF NOT AGGRESSIVELY MANAGED!
SHOCK
WHAT IS SHOCK?

• IN MEDICINE, SHOCK (HYPOPERFUSION) IS A LIFE-THREATENING MEDICAL EMERGENCY CHARACTERIZED BY INABILITY OF THE BODY TO SUPPLY ENOUGH OXYGEN TO MEET TISSUE REQUIREMENTS.

• HYPOTENSION IS USUALLY, THOUGH NOT ALWAYS, PRESENT.

• WITHOUT PROMPT MEDICAL TREATMENT, SHOCK USUALLY CAUSES DEATH.
TYPES OF SHOCK

• NEUROGENIC SHOCK
• OBSTRUCTIVE SHOCK
• DISTRIBUTIVE SHOCK
• CARDIOGENIC SHOCK
• HYPOVOLEMIC SHOCK
DISTRIBUTIVE SHOCK

- SHOCK DUE TO VASODILATION
  - SEPTIC SHOCK
  - ANAPHYLACTIC SHOCK
  - ACUTE ADRENAL INSUFFICIENCY
    - DISCONTINUATION OF STEROIDS RAPIDLY
WHAT IS SEPTIC SHOCK?

- URGENT CONDITION WITH EMERGENCY INTERVENTION WARRANTED
- MORTALITY CAN BE UP TO 50%
MORE OUTSIDE CRITERIA FOR SEPTIC SHOCK

• HYPOTENSION (SYSTOLIC BP <90 MM/HG OR <40 MM HG DROP FROM BASELINE) DESPITE FLUIDS

• HYPOPROFUSION ABNORMALITIES
  • OLIGURIA
  • LACTIC ACIDOSIS
  • ACUTE CHANGE IN MENTAL STATUS

• BACTEREMIA

• MULTIPLE ORGAN DYSFUNCTION SYNDROME
EPIDEMIOLOGY

- 3 cases per 1,000 population
- 2 cases per 100 patients admitted to hospital
  - >100K deaths per year
  - AKA septicemia or “blood poisoning”
ETIOLOGY

- ANY POSSIBLE PATHOGEN – COMMON INCLUDE:
  - GRAM +
    - STAPH
    - STREP
    - ENTEROCOCCUS
  - GRAM –
    - E. COLI
    - KLEBSIELLA
    - PROTEUS
    - PSEUDOMONAS
  - FUNGI
    - CANDIDA
COMMUNITY ACQUIRED VS. NOSOCOMIAL

• COMMUNITY ACQUIRED
  • TYPICALLY DIAGNOSED <72 AFTER ADMISSION

• NOSOCOMIAL
  • RESIDENTS OF LONG-TERM CARE FACILITIES
  • PRIMARY
    • ABSENT IDENTIFIABLE SOURCE -EXCEPT- LINE SEPSIS
  • SECONDARY
    • SOURCE IDENTIFIABLE I.E. WOUND, ABSCESS, ETC.
ACT QUICKLY!

- RAPID INTERVENTION SAVES LIVES!
- LEARN TO RECOGNIZE MANIFESTATIONS EARLY!
- IF YOU DON’T IT MAY BE TOO LATE!
COMMON CAUSES

- LUNG INFECTIONS
- UTI
- ABSCESS
- PERITONITIS
- IV CATHS
- CELLULITIS
- BILIARY TREE
- UNKNOWN 20%
MENINGITIS

Brudzinski's neck sign

Kernig's sign
ENCEPHALITIS

- INFLAMMATION OF THE BRAIN
- TYPICALLY CAUSED BY A VIRUS
- PRIMARY
  - DIRECT INFECTION OF BRAIN AND SPINAL CORD
- SECONDARY
  - SPREAD OF VIRUS FROM ELSEWHERE IN THE BODY TO THE BRAIN
SYMPTOMS

• HEADACHE
• IRRITABILITY
• LETHARGY
• FEVER
• JOINT PAIN
• CONFUSION AND HALLUCINATIONS
• LOSS OF CONSCIOUSNESS
• BULGING IN THE SOFT SPOTS (FONTANELS) OF THE SKULL IN INFANTS
• PERSONALITY CHANGES
• DOUBLE VISION
• SEIZURES
• MUSCLE WEAKNESS
• LOSS OF SENSATION OR PARALYSIS IN CERTAIN AREAS
• TREMORS
• RASH
URINARY SOURCES OF SEPSIS

- PROSTATITIS
  - PROCEDURES
- UTI
- STONES
- CATHETERS
  - DM
LUNG INFECTIONS

- OLD MAN’S FRIEND
- ASPIRATION
- ATELECTASIS
ABSCESS

- TOOTH
- INTRAABDOMINAL
- MRSA
- POST-SURGICAL
PERITONITIS

• PERF. VISCUS – ACUTE ABDOMEN - RIGIDITY
• SBP
• DIALYSIS
• SIGNS/SYMPTOMS:
  • DISTENTION
  • REBOUND
  • GUARDING
  • NO FLATUS
  • N/V
LINES

- CENTRAL
  - JUGULAR
  - FEMORAL
  - SUBCLAVIAN
- PERIPHERAL
- PICC
- ARTERIAL
Figure 46-10 Placement of triple-lumen nontunneled percutaneous central venous catheter.
CELLULITIS

BILIARY TREE

- CHOLECYSTITIS
- ASCENDING CHOLANGITIS
  - CHARCOT’S TRIAD
  - LAB FINDINGS
  - BILIARY MANIPULATION OR OBSTRUCTION
  - HIGH MORTALITY
- PANCREATITIS
- CHOLEDODCHOLITHIASIS
  - MRCP, ERCP, T-TUBE
Figure 41-18 Placement of the T tube. Dotted lines indicate parts removed.

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SIGNS/SYMPOTOMS

- FEVER/CHILLS
- AMS
  - PULSE MAY BE WEAK OR THREADY
  - DROP IN SYSTOLIC BP >10-20 MM Hg AND AN INCREASE IN PULSE >15 BPM WITH POSITIONAL CHANGE
- TACHYCARDIA
- TACHYPNEA
- HYPOTENSION
- PETECHIA

**SIGNS RELATED TO PRIMARY SIGHT OF INFECTION**
SIGNS/SYMPTOMS RELATED TO PRIMARY INFECTION

- COUGH
- DYSURIA
- N/V/D
- ABDOMINAL PAIN
- STIFF NECK
- HEADACHE
- JAUNDICE
WHAT DOES THIS PATIENT LOOK LIKE IN MY ER OR HOSPITAL?

• RAPIDLY WORSENING!!!!!!

• THIS IS WHY WE CONTINUOUSLY REPEAT VITAL SIGNS!

• “SOMETHING IS NOT RIGHT”

• CAN PRESENT BENIGN THEN “CIRCLE THE DRAIN”
WHO IS MOST AT RISK

- ELDERLY
- RECENT SURGICAL PATIENTS
- PATIENTS WITH INDWELLING APPARATUS
- DIABETICS
- IMMUNOSUPPRESSED
- ALCOHOLICS
- TRAUMA/BURNS
- IV DRUG USERS
- MALIGNANCY
TREATMENT/MANAGEMENT
SURVIVING SEPSIS CAMPAIGN - DEFINED

• 2004 – 2010

• INTERVENTIONS OF EARLY GOAL-DIRECTED THERAPY DESCRIBED BY CRITICAL CARE AND ID EXPERTS FOR THE BEDSIDE CLINICIAN TO IMPROVE THE OUTCOME OF SEPSIS AND SEPTIC SHOCK.

• “WHAT WORKS” VS. “WHAT DOESN’T”
INTERVENTIONS THAT MATTER- I.E. WHAT AM I GOING TO DO!

• PRIOR TO THIS NO TRUE OUTLINED GUIDELINES FOR MANAGEMENT EXISTED.

• MULTIPLE LOOSELY -SET PROTOCOLS INDIVIDUALIZED TO EACH INSTITUTION OR EVEN PROVIDER
EVIDENCE BASED

• 135 REFERENCE ARTICLES USED
• 11 GROUPS
• RATED A-E
  • A. AT LEAST 2 LEVEL I INVESTIGATIONS
  • B. ONE LEVEL I INVESTIGATION
  • C. LEVEL II INVESTIGATIONS ONLY
  • D. SUPPORTED BY AT LEAST LEVEL III INVESTIGATION
  • E. SUPPORTED BY LEVEL IV OR V EVIDENCE
GRADING OF EVIDENCE

• I. LARGE, RANDOMIZED TRIALS WITH CLEAR-CUT RESULTS; LOW RISK OF FALSE-POSITIVE (ALPHA) ERROR OR FALSE-NEGATIVE (BETA) ERROR

• II. SMALL, RANDOMIZED TRIALS WITH UNCERTAIN RESULTS; MODERATE-TO-HIGH RISK OF FALSE +

• III. NONRANDOMIZED, CONTEMPORANEOUS CONTROLS

• IV. NONRANDOMIZED, HISTORICAL CONTROLS AND EXPERT OPINION

• V. CASE SERIES, UNCONTROLLED STUDIES, AND EXPERT OPINION
INTERVENTIONS INVESTIGATED

- Initial Resuscitation
- Diagnosis
- Antibiotics
- Source Control
- Fluid Therapy
- Vasopressors
- Inotropic Therapy
- Steroids
- Limitation of Support Considerations
- Activated Protein C
- Blood Products
- Mechanical Ventilation
- Sedation
- Glucose Control
- Renal Replacement
- DVT Prophylaxis
- Bicarb
- Stress Ulcer Prophylaxis
IV FLUIDS/RESUSCITATION

- TISSUE HYPOPERFUSION IS KEY ELEMENT OF SEPSIS
- PUSH, PUSH, PUSH = AGGRESSIVE FLUID RESUSCITATION!!!
- LIKE TRAUMA = 2 “LARGE BORE” IV LINES
  - AC OR HIGHER
- COLLOID VS. CRYSTALLOID DOES NOT MATTER
DIAGNOSIS/CULTURES

• BEFORE ANTIBIOTICS PLEASE
• 2 SETS MIN.
• AT LEAST ONE DRAW PERCUTANEOUSLY AND 1 DRAW THROUGH EACH VASCULAR ACCESS DEVICE
• URINE CULTURES
• CSF
• WOUND CX
• RESP. SECRETIONS
• OTHER – ASCITES, BODY FLUID, ETC.
IDENTIFIED BACTERIUM = SOURCE

- STREP. PNEUMO
  - PNEUMONIA, MENINGITIS
- KLEBSIELLA
  - BILIARY TRACT, UTI, LOWER RESPIRATORY TRACT
- ENTEROCOCCUS
  - URINARY TRACT
- E. COLI
  - UTI, BILIARY TRACT
- PROTEUS
  - URINARY TRACT
- PSEUDOMONAS
  - UTI
- STREP BOVIS OR VIRIDANS
  - ENDOCARDITIS
- STAPH AUREUS
  - LOWER RESPIRATORY TRACT, ENDOCARDITIS
LAB TESTS

- MULTIPLE PANELS
- CBC
- CMP
- PT/PTT
- PAN-CULTURES
- ABG
- LP
IDENTIFYING ACUTE ORGAN DYSFUNCTION AS A MARKER OF SEVERE SEPSIS

- Tachycardia
- Hypotension
- CVP
- PAOP
- Tachypnea
- PaO₂ < 70 mm Hg
- SaO₂ < 90%
- PaO₂/FiO₂ ≤ 300
- Jaundice
- ↑ Enzymes
- ↓ Albumin
- ↑ PT
- Oliguria
- Anuria
- ↑ Creatinine
- Platelets
- ↑ PT/APTT
- Protein C
- ↑ D-dimer

- Altered Consciousness
- Confusion
- Psychosis
- D-dimer
# CBC WITH DIFF

<table>
<thead>
<tr>
<th>Tests</th>
<th>Result</th>
<th>Flag</th>
<th>Units</th>
<th>Reference Interval</th>
<th>Lab</th>
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<tbody>
<tr>
<td>CBC With Differential/Platelet</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>WBC</td>
<td>5.7</td>
<td>x10E3/uL</td>
<td>4.0-10.5</td>
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<tr>
<td>RBC</td>
<td>5.27</td>
<td>x10E6/uL</td>
<td>4.10-5.60</td>
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<td>Hemoglobin</td>
<td>15.4</td>
<td>g/dL</td>
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<td>Hematocrit</td>
<td>44.1</td>
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<td>36.0-50.0</td>
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<td>MCV</td>
<td>84</td>
<td>fL</td>
<td>80-98</td>
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<td>%</td>
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<td>40-74</td>
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<td>46</td>
<td>%</td>
<td>14-46</td>
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<tr>
<td>Monocytes</td>
<td>6</td>
<td>%</td>
<td>4-13</td>
<td>01</td>
<td></td>
</tr>
<tr>
<td>Eos</td>
<td>1</td>
<td>%</td>
<td>0-7</td>
<td>01</td>
<td></td>
</tr>
<tr>
<td>Basos</td>
<td>0</td>
<td>%</td>
<td>0-3</td>
<td>01</td>
<td></td>
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<tr>
<td>Neutrophils (Absolute)</td>
<td>2.6</td>
<td>x10E3/uL</td>
<td>1.8-7.8</td>
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<td>Lymphs (Absolute)</td>
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<td>x10E3/uL</td>
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<td>Monocytes(Absolute)</td>
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<td>x10E3/uL</td>
<td>0.1-1.0</td>
<td>01</td>
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<td>x10E3/uL</td>
<td>0.0-0.4</td>
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<tr>
<td>Baso (Absolute)</td>
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<td>x10E3/uL</td>
<td>0.0-0.2</td>
<td>01</td>
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<tr>
<td>Immature Granulocytes</td>
<td>0</td>
<td>%</td>
<td>0-1</td>
<td>01</td>
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<tr>
<td>Immature Grans (Abs)</td>
<td>0.0</td>
<td>x10E3/uL</td>
<td>0.0-0.1</td>
<td>01</td>
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</table>

ANTIBIOTICS

• WITHIN THE FIRST HOUR
• BROAD SPECTRUM
• KNOW YOUR INFUSION RATES (RAPID VS. SLOW)
• THINK PATHOGENS
• COMBINATION RX FOR NEUTROPENIA
DISCOVER THE SOURCE!

- DRAIN ABSCESSES
- DEBRIDE NECROTIC TISSUE
- REMOVE INFECTED DEVICES
- SURGERY/REPAIR
Cerebrospinal fluid drawn from between two vertebrae
IMAGING

- ONLY IF THE PATIENT IS STABLE TO TRANSPORT!!!!!
- CT
- MRI
- X-RAY (MINIMUM CHEST)
EARLY GOAL-DIRECTED THERAPY (EGDT) TARGETS OF SEPSIS MANAGEMENT

- Mean arterial pressure (MAP) $\geq 65$
- Urine output $\geq 0.5$ ml/kg/hr
- Central Venous Pressure (CVP) 8-12 mm Hg
- Central Venous oxyhemoglobin saturation (SvO2) $\geq 65$ (if a pulmonary artery catheter is being used)
PRESSORS

- IF FLUID NOT DOING THE JOB!!
- GOAL IS TISSUE PERFUSION - BP
- DOPAMINE
- NOREPINEPHRINE
- ADMINISTER THROUGH CENTRAL LINE
- ASAP GET AN ARTERIAL LINE TO MEASURE BP (MORE ACCURATE)
- VASOPRESSION FOR REFRACTORY SHOCK IF OTHERS FAIL
INOTROPIC THERAPY

• DOBUTAMINE
  • FOR LOW CARDIAC OUTPUT DESPITE ADEQUATE FLUID RESUSCITATION
  • COMBINED WITH VASOPRESSOR THERAPY
Blood pressure and blood samples can be taken through catheter
STEROIDS?

- ONLY IF PATIENT IN SEPTIC SHOCK (WBC)
- IV HYDROCORTISONE 200-300 MG/DAY X 7D IN 3-4 DIVIDED DOSES FOR THOSE ON PRESSORS
- TO REVERSE RELATIVE ADRENAL INSUFFICIENCY
- START STEROIDS EMERGENTLY THEN CONSIDER ACTH STIMULATION TEST TO IDENTIFY RESPONDERS AND POSSIBLY DISCONTINUE THERAPY
- TAPER OFF WHEN NO LONGER NEEDED
MECHANICAL VENTILATION

• BEYOND SCOPE OF OUR MANAGEMENT LEVEL
• CALL A PULMONOLOGIST/CRITICAL CARE MD
• OVERALL VERY COMPLEX
• USUAL GOAL TO MAINTAIN END-INSPIRATORY PLATEAU PRESSURES <30 CM H2O
• SEMIRECUMBENT UNLESS CONTRAINDICATED WITH HEAD OF BED AT 45%
• NEED AN NG TUBE
SEDATION

- INTERMITTENT BOLUS VS. CONT. INFUSION
- SEDATIVE NAMES
- NEUROMUSCULAR BLOCKERS TO BE AVOIDED
  - RISK OF PROLONGED SKELETAL MUSCLE WEAKNESS
BLOOD PRODUCTS

• PRBCS
• ONLY WHEN HGB <7.0 OR WITH ACTIVE BLOOD LOSS
• ERYTHROPOIETIN NOT RECOMMENDED
• FFP ONLY IF PLANNED INTERVENTION OR ACTIVE HEMORRHAGE
• ANTITHROMBIN NOT RECOMMENDED
• PLATELET TRANSFUSION ONLY IF <5K UNLESS INTERVENTION PLANNED – THEN ONLY IF <30K
**GLUCOSE CONTROL**

- **<150 MG/DL GOAL ONCE STABLE**
- **SLIDING SCALE**

---

**Figure 10-2**

**Insulin Algorithm Sheet**

<table>
<thead>
<tr>
<th>Name</th>
<th>Provider</th>
<th>Date</th>
<th>Phone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time between injection and meal (minutes)</td>
<td>Blood glucose value (mg/dL)</td>
<td>Breakfast</td>
<td>Lunch</td>
</tr>
<tr>
<td>Humalog</td>
<td>Regular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>5 to 15</td>
<td>&lt; 80</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>81 to 150</td>
<td></td>
</tr>
<tr>
<td>5 to 15</td>
<td>30 to 45</td>
<td>151 to 200</td>
<td></td>
</tr>
<tr>
<td>15 to 30</td>
<td>45 to 60</td>
<td>201 to 250</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>60</td>
<td>251 to 300</td>
<td></td>
</tr>
<tr>
<td>30+</td>
<td>60+</td>
<td>301 to 350</td>
<td></td>
</tr>
<tr>
<td>30+</td>
<td>60+</td>
<td>351 to 400</td>
<td></td>
</tr>
<tr>
<td>30+</td>
<td>60+</td>
<td>401 to 450</td>
<td></td>
</tr>
<tr>
<td>30+</td>
<td>60+</td>
<td>451+</td>
<td></td>
</tr>
</tbody>
</table>

AM long-acting insulin dose
PM long-acting insulin dose

☐ Take before dinner
☐ Take at bedtime
RENAL REPLACEMENT

• CONTINUOUS HEMOFILTRATION AN INITIAL OPTION
• SHORT TERM HEMODIALYSIS IF NEEDED
DIET

- NPO INITIALLY
- POSSIBLE TPN
- POSSIBLE ENTERAL FEEDS
STRESS ULCER PREVENTION

• WHY NEEDED?

• PPI BOLUS FOLLOWED BY CONTINUOUS INFUSION
  • EX: “PROTONIX 80 MG BOLUS IV FOLLOWED BY 8 MG/HR CONTINUOUS INFUSION. D/C AFTER 72 H AND GIVE 40 MG DAILY IV OR PO IF ABLE.”
DVT PROPHYLAXIS

• ONLY IF NO COAG/BLEEDING DISORDER!!!
• LMW HEPARIN
• IF CONTRAINDICATED (IF BLEED OR RECENT BLEED)
  • ICD (INTERMITTENT COMPRESSION DEVICE)
  • GRADUATED COMPRESSION STOCKINGS
DIC

- CONSUMPTION OF PLATELETS
- PROLONGED CLOTTING TIMES
- BLOOD CLOTS CLOG VESSELS DUE TO ALTERED HEMOSTASIS
- MULTIFACTORIAL
- ALREADY LECTURED ON EARLIER THIS YEAR...
LIMITATION OF SUPPORT

• TALK TO YOUR PATIENTS!!!
• TALK TO THEIR FAMILIES!!!
• BE HONEST ABOUT ODDS/OUTCOMES!!!
• ADVANCE PLANNING IS KEY TO AVOIDING NIGHTMARES
BIOMARKERS MAY HOLD THE KEY TO EARLY DETECTION AND DISEASE MANAGEMENT

Because early recognition of sepsis is key to improving outcomes, several potential biomarkers are being investigated that may alert clinicians to the initial inflammatory response.

These biomarkers could provide a faster, more accurate diagnosis of sepsis.

Early diagnosis and intervention could mean improved outcomes and survival for septic patients.

Biomarkers being investigated include:
- Procalcitonin (seems to be effective in distinguishing bacterial infections vs nonbacterial)
- C-reactive Protein
- Lipopolysaccharide binding protein
- Toll-like receptor-2
VRE

• TYPICALLY AFFECTS URINARY SYSTEM
• CAN BE RESISTANT TO MORE THAN ONE DRUG
• COLONIZES BOWEL
• MODIFIED CONTACT PRECAUTIONS
REASSESS

• REASSESS AFTER 48-72 HOURS WITH DATA WHEN CULTURES COME BACK

• TO PREVENT SUPERINFECTIONS:
  • C. DIFF
  • FUNGI
  • VRE
C. DIFF COLITIS

• NATURAL PROTECTIVE GUT BACTERIA KILLED BY ABX
• PATHOGENIC BACTERIA FLOURISH
• WATERY STOOL /DIARRHEA 10X QD OR MORE
• “CORN TORTILLA”
• COMPLICATION TOXIC MEGALOCOLON
• CONTACT PRECAUTIONS
• RX: FLAGYL, RIFAXAMIN, QUESTRAN, VANCOMYCIN
  • NOT LEVAQUIN
ARDS

• MAIN PATHOLOGICAL FEATURE IS DIFFUSE ALVEOLAR DAMAGE

• INCREASED VASCULAR PERMEABILITY LEADS TO INTERSTITIAL AND ALVEOLAR PULMONARY EDEMA, ALVEOLAR COLLAPSE, HYPOXEMIA

• PRESENTS AS ACUTE ONSET RESPIRATORY FAILURE

• SYNDROME, NOT A DISEASE

• VARIETY OF CAUSES

• USUAL ONSET IS 12-48 HOURS AFTER ORIGINAL INSULT
ARDS CONT.

- BILATERAL INFLTRATES ON CXR
- NORMAL PCWP (<18 MM HG)
- SEVERE HYPOXEMIA
  - ALI PAO2/FIO2 <300
  - ARDS PAO2/FIO2 <200
- ESTIMATED THAT ARDS IS DUE TO SEPSIS 40% OF TIME
FUNGEMIA

- CANDIDA – FLUCONAZOLE
- EVERYTHING ELSE = AMPHO B
PREVENTION - WHAT CAN BE DONE?

- RAPID CYCLE SEPSIS TEAM
  - SCREEN 90% OF ADULT NON-TRAUMA ED PTS.
- SYNOPSIS VIEW ON EPIC FOR TRENDS
- SEPSIS MANAGEMENT FLOW-CHART FOR ED
VIDEOS – SEPSIS ALLIANCE.ORG

- HTTP://YOUTU.BE/2ELPYOMIMZG
- HTTP://YOUTU.BE/ELJTHKDK6U4
- SEPSIS EMERGENCY
REFERENCES


WWW.SEPSISALLIANCE.ORG
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