THE MYTHS IN TREATING THE SHOCKY PATIENT

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OBJECTIVES

➢ To discuss an evidence based approach to management of the shocky patient
➢ To describe some myths and controversies in the treatment of septic, obstructive, anaphylactic and toxic shock

DISCLOSURES

Participant in several advisory boards and have received unrestricted education and research grants at UHN:
➢ Leo, BI, Roche, Sanofi Aventis, Bayer, Janssen, Pfizer, Purdue, Octapharma, CSL Behring

WHAT IS SHOCK?

Inadequate Perfusion to meet Demands
Cellular Hypoxia
Systemic Effects
The Road to Death

ED Doc to the Rescue

TYPES OF SHOCK

➢ Hypovolemic: intravascular volume loss
➢ Cardiogenic: pump failure (decreased CO, incr SVR)
➢ Distributive: - Septic
- Anaphylactic
- Neurogenic
- Toxic
- Other

BEDSIDE MYTHS & PEARLS …

➢ An adequate bp does not exclude shock
➢ Vasopressors: raise bp (MAP 65-90), increase vital organ perfusion, but reduce peripheral perfusion
➢ Clinical signs of hypoperfusion despite adequate bp:
  ➢ Cool, mottled, vasoconstricted skin (blood shunted centrally)
  ➢ Oliguria, anuria
  ➢ Depressed sensorium
BEDSIDE MYTHS & PEARLS …
- wbc in urine + hypotension ≠ septic shock
  (corollary: PE and MI don’t respond to cipro!)
- 250 cc bolus increases urine output by 0.3 cc/hr
  (corollary: CHF and CRF patients may die of intravascular volume depletion)

- Shocky patients don’t die of central line deficiency, but rather under-resuscitation.
  - central lines can aid in volume resuscitation, serial bloods
    (grade D recommendation, CAEP guidelines 2008)
  - CVP and SVO2 monitoring: no independent mortality benefit
  - Arterial lines are not immediately required when vasopressors are used in the ED
    - “when resources allow” (grade D recommendations, CAEP guidelines 2008)

- bedside ultrasound helps distinguish between the types of shock: IVC collapse, LV function, RV size, pericardial fluid (tamponade), AAA, free abd fluid

SURVIVING SHOCK
- Early recognition
- Early, aggressive management
- Mortality: septic shock: > 35%
  cardiogenic shock > 60%
CASE 1
- 64 y.o. lady brought by EMS after collapse at home
- fever, cough, poor oral intake, weakness x 5 days
- P 130, rr 28, bp 60/40, T 39°C, O2 Sats 94% R/A
- iv N/S bolus x 3 L: bp 70/50
- ECG: sinus tachycardia

SEPTIC SHOCK
- Septic shock = inflammatory response to infection with hypotension refractory to fluid resuscitation
- Surviving Sepsis Campaign 2008
- CAEP Guidelines 2008
- Rivers study NEJM 2001: EDGT

SEPTIC SHOCK
- Early goal-directed resuscitation of the septic patient within 6 hrs of recognition
- Blood cultures before antibiotics
- Prompt imaging to confirm source of infection
- Broad-spectrum antibiotic therapy within 1 hr of diagnosis
- Blood transfusion if Hb < 70 g/L (hematocrit < 30)

RIVERS STUDY
EDGT Group: therapy in first 6 hours:
- How much fluid: 5-8 L vs. 3-6 L
- How many intubated: 1/2 (same as control)
- Blood products: 2/3 vs. 1/5
- Vasopressors: 1/3 (same as control)
- Ionotropes: 14% vs. 1%

*Biggest difference between groups was amount of fluid, blood and ionotrope use

CONTROVERSIES …
Which fluid: crystalloid or colloid?
- N/S or R/L effective
- No benefit to using albumin (increased cost)
- Pentastarch associated with higher mortality
- Judicious use of PRBCs beneficial

NEJM 2001:345:1368
NEJM 2008:358:125
CONTROVERSIES ...
Which antibiotics?
- Higher mortality if delayed or inappropriate choice of antibiotic
- In most cases, origin of infection is predictable:
  - 25%: lower respiratory tract
  - 25%: urinary
  - 15%: skin, soft-tissue
  - 15%: GI

CHOICE OF ANTIBIOTIC
Respiratory origin:
- Streptococcus pneumoniae
- Klebsiella pneumoniae
- Staphylococcus aureus

Urinary Origin
- E coli
- Proteus species
- Klebsiella species

GI Origin
- E coli
- Streptococcus faecalis
- Bacteroides fragilis

Skin Origin
- S aureus
- Staphylococcus epidermidis
- Streptococci

Empiric Treatment of Septic Shock Without Known Infection Source:
- Vancomycin plus Imipenem or Piperacillin-tazobactam or Cefepime
CONTROVERSIES ...

What about steroids?
- Rationale: relative adrenal insufficiency, anti-inflammatory effect, upregulation of catecholamine receptors
- High dose (30 mg/kg): higher mortality
- Low dose (50 mg iv hydrocortisone): no benefit
- Risk of secondary infections, superinfections and new sepsis/shock

NEJM 2008;358(2):111

CONTROVERSIES ...

What about steroids?
- No role in ED unless known (Addison’s) or expected (chronic steroid use) adrenal insufficiency
- ICU likely to do ACTH stimulation test

CONTROVERSIES ...

Which vasopressor
- Dopamine more arrhythmogenic (AF) and greater tachycardia than norepinephrine
- Norepinephrine more potent alpha agonist
- No mortality benefit of one over the other
- Use the agent you’re most familiar with and available

NEJM 2010;362(9):779

CONTROVERSIES ...

Refractory hypotension
- Epinephrine: 1-10 mcg/min
- Phenylephrine: 3 mcg/kg iv bolus (0.1 – 0.5 mg) q10-15 minutes; infusion 100-180 mcg/min
- Pure alpha agent: useful with tachycardia/arrhythmias

NEJM 2010;362(9):779

IONOTROPIC SUPPORT
- Add dobutamine if low cardiac output and high SVR
  - eg. reasonable bp after fluids/vasopressor but cool, clammy, poor cap refill, low urine output
  - eg. Svo2 < 70 after fluids, vasopressor (PRBC)
  - Increases HR, contractility and CO with beta stimulation, but may drop bp by peripheral vasodilation (use with vasopressor)

NO CONTROVERSIES HERE...
- Elevated lactate a good indication of tissue hypoperfusion (anaerobic metabolism): decreasing lactate a surrogate marker of improving perfusion
- Activated protein C:
  - Anticoagulant and anti-inflammatory effects
  - Prowess-Shock trial (2011): no benefit in shock
  - Xigris withdrawn from market (Eli Lilly)
CASE 1
- More iv N/S given: urine output increased
- Dopamine 15 mcg/kg/min: bp 96/65
- Hb 145, cultures sent
- CXR: no clear infiltrate
- PipTazo 4.5 g iv + Levofloxacin 750 mg iv
- No blood products, ionotrope, steroids or protein C

CASE 1
- Intensivist paged STAT to ED to place fancy central line, but senior emerg resident insists on placing line in ED under U/S guidance
- ED nurse informs intensivist of ED policy of “no art lines in the ED”

CASE 2
- 44 y.o. previously healthy male with recent ORIF for ankle fracture
- Presents with sudden CP + SOB
- p 120, bp 70/40, rr 30, O2 sats 92% R/A
- Persistent hypotension despite fluids

CASE 2
- Bedside echo: dilated RV/RA
- CTA ordered
- Patient decompensates with no measurable bp and weak pulses bilaterally
- What to do?
CONTROVERSIES
- Fluid resuscitation + pressors + heparin + call ICU AND...
- Hope for the best!
  OR
- Look up dose of thrombolytic and give it iv
  OR
- Transfer for interventional intra-arterial procedure
  OR
- Transfer for surgical thrombectomy

CASE 2
Obstructive Shock 2° to PE
- Excessive fluids worsen RV dysfunction
- Usually insufficient time to consider catheter-directed lytic or surgery
- Limited data from case series
- Traditional recommendation: 100 mg tPA over 2 hrs
- Rapid infusion: 0.6 mg/kg over 2 min (50 mg iv STAT)
- TNK also effective in small case series

CASE 3
- 32 y.o. lady eating Chinese food at restaurant
- Suddenly gets red, itchy, stomach cramps and swelling of face
- Known "bad" reactions to nuts
- Left her Epi-Pen at home
- Driven by friend to ED: p 110, rr 28, bp 80/-, 02 sats 99%

CASE 3
- Rx: 02, iv fluid bolus, Epi 0.5 mg im, iv Benadryl
- Airway: ok for now (no stridor, airway cart at bedside)
- Breathing: few wheezes both bases
- Circulation: bp now 70/-

CONTROVERSIES
Anaphylactic Shock
- Epinephrine:
  - sc useless, im may be ineffective due to poor muscle perfusion
  - iv fastest, most effective choice
  - bolus: dosing errors and side effects
  - 1:10000: 25-50 mcg iv bolus
  - infusion: start 2-10 mcg/min
CONTROVERSIES

Antihistamines
- Relieve itch and hives: nothing else!
- No evidence for benefit of H2 blockers after H1 blocker given

CONTROVERSIES

Steroids
- No proven benefit
- No initial benefit for anaphylaxis
- Routinely given to prevent late phase response (23% chance of biphasic reaction in 8 – 10 hrs)

CONTROVERSIES

Observation Period
- Older literature: must admit to hospital
- Newer guidelines: observation period of 8-12 hours
- ED culture: watch for a couple of hours
- State of the art: when bed needed for next patient (document: patient demanding to leave)

CASE 4
- 18 y.o. female
- 4 day Hx of fatigue, back pain
- Developed a faint rash and fever two days previous and placed on Z-pak at WIC
- O/E: NAD
  - p 130
  - bp 95/60
  - T 36 C

CASE 4
- placed in ambulatory area of ED
- progressively worsens over following few hours with pallor, increasing HR and decreasing bp
- asked about tampon use: menstruating
- develops dyspnoea and confusion

TOXINS

CONTROVERSIES

Toxic Shock Syndrome
- Etiology: Group A streptococcus
  - Staphylococcus aureus
- Management: Early Goal Directed Therapy
- ?which antibiotic
- ?IVIG
TOXIC SHOCK SYNDROME

- Antibiotics: Clindamycin 900 mg iv
  plus
  Cefazolin 2 g iv
  (or Vancomycin 2g iv if MRSA suspected)
- IVIG 400 mg/kg single dose over several hours