# SHOCK

- Presented by Dr.Pravin Verma

# SHOCK

DEF : Circulation fails to meet nutrtional needs of cells & remove metabolic waste products resulting from discrepency in size of vascular bed & volume of intravascular fluid. It is low blood flow state in vital organs.

TYPES :

# HAEMATOGENIC/HYPOVOLEMIC SHOCK

- HAEMORHAGE
  * CONCEALED-Chest, abdomen, pelvic bld
  * REVEALED-External bld= Head, chest, abd, pelvis, #s, crush injury
- PLASMA LOSS-Burns
- WATER & ELECTROLYTES LOSS-DEHYDRATION= Vomitting, diarrhoea, sever UTI, fever

# SHOCK TYPES ( CONT.. )

- TRAUMATIC SHOCK- Similar to haematogenic shock along with associated problems i. e.
- Head injury
- Chest injury-Pericardial effusion,S heart contusion, cardiac temponade ( ass with cardiogenic shock ). – Lung contusion, haemothorax ( ass with hypoxia )
- GIT- Liver, splenic, mesentric vein tear (as with toxin release)

# NEUROGENIC SHOCK

NEUROGENIC SHOCK

- Causes- paraplegia, qudruplegia, spinal injury, spinal anaesthesia→ Blockade of SNS < arterial & venous tone→ pooling of blood in venous system

VS HV SHOCK-PR (n), BP<, Warm & dry skin ( cold & pale in HVS )

Rx- Trendelenberg'gs position, IV Fluids & Vasopressures

- VASOVAGAL/ VASOGENIC SHOCK

  Sudden pain, fear→ vagal stimulation→ <PR, BP & Dilatation of capillary bed & periferal pooling in skin, splanchnic bed→ <blood flow to CNS→Unconciousness

- PSYCHOGENIC SHOCK

  Fear, bad news, accident

# CARDIGENIC SHOCK ( PUMP FAILURE )

- IHD, Myocarditis, cardiomyopathuy,MI, CCF, cardiac arrythmia, heart injury

RVF-Periferal oedema, ascites, hepato/ spleno megali

LVF- Pulm oedema, hypoxia, crepts

- MASSIVE PULM EMBOLISM-50 % PA occlusion→RVF→ < Lt ventricular filling

- CARDIAC COMPRESSIVE SHOCK- Pericardial effusion, tension pneumothorax, large diaphragmatic hernia

# SEPTIC SHOCK

Infective foci-- i. e. meningitis, cholangitis, peritonitis, septicemia by gram –ve bact.→endotoxin release→SIRS→SEPTICEMIC SHOCK

EARLY( WARM STAGE )-Dilatation of skin A-V shunts→< periferal vascular resistance, but N Cardiac Output.

LATE ( COLD STAGE )- Vascular leakage→hypovolemia→Hypovolemic Shock

# ANAPHYLACTIC SHOCK

Penicillin/ Serum administration→Type-I hypersensitivity→Mast cells & Basophils ativation→Histamine,prostaglandins & SRS( slow release substance) of anaphylaxis------→-→

- Laryngeal oedema, bronchospasm--- resp. distress
- Massive periferal vasodilation---hypovolemia

# HYPOVOLEMIC SHOCK

- DEF
- CAUSES
- PATHOPHYSIOLOGY--- < INTRAVASCULAR VOLUME→ < RA FILLING→ < RV FILLING→ < LA FILLING→ < LV FILLING→ < STROKE VOLUME & C.O.→ SHOCK→ MODS→MOSF

# COMPENSATORY MECHANISMS

- ADRENERGIC DISCHRGE-1 min

Stimulation of SNS→ adrenergic discharge→ vasoconstriction of veins & venules in skin, kidney & splanchnic bed→> VR to RA→> SV & CO

- HYPERVENTILATION

Shock→anaerobic respiration→ metabolic acidosis→ hyperventilation

# COMPENSATORY MECHANISMS ( CONT…. )

- RELEASE OF VASOACTIVE AMINES- 1-2 MIN
  - RENIN-ANGIOTENTIN→Low perfusion of kidney→renin from kidney→AT-I from liver→AT-II conversion in lungs→ potent selective vasoconstricter
  - EPINEPHRIN→release from adrenal medulla due to adrenergic discharge
  - VASOPRESSIN→< CO→ Hypotension→ stimulation of baroreceptors ( aortic arch & carotid body→ADH from posterior pituitary
- COLLAPSE—Recumbent position-> VR

# COMPENSATORY MECHANISMS ( CONT…. )

- RESORPTION OF FLUID FROM INTERSTITIAL TISSUE TO INTRAVASCULAR COMPARTMENT

> adrenergic discharge→ constriction of arterioles & pre & post capillary sphincters, venules & veins in splanchnic bed, skin, muscles→ < capillary intravascular hydrostatic pressure→

# COMPENSATORY MECHANISMS ( CONT…. )

- RESORPTION OF FLUID FROM INTRACELLULAR TO EXTRACELLULAR SPACE

  anaerobic respiration & < insulin, > glucagon, epinephrin & cortisol→ > extracellular glucose & > metabolic products of an. Resp→> osmolarity of blood→

# COMPENSATORY MECHANISMS ( CONT…. )

- RENAL CONSERVATION OF WATER & Na+

- stress→ > ACTH→> aldosterone
- > AT-II→
- Baroreceptor activation→ ADH release

→ all will lead to→ reabs of water from renal tubules

# STAGES OF SHOCK

| Charecter | I | II | III | IV |
|---|---|---|---|---|
| Blood loss | < 15% 750 ml | 15-30% 0.8-1 lit | 30-40% 1.5-2 lit | > 40% > 2 lit |
| Syst. BP | N | N | < | < |
| PR/min | Slight > | 100-120 | >120 weak | >120 thready |
| UOP ml/hr | 30 | 20-30 | < 20 | Ngligible |
| Capillary Refill sec | N-2 | Delayed | Delayed | Very Delayed |

# STAGES OF SHOCK ( Cont.. )

| Character | I | II | III | IV |
|---|---|---|---|---|
| Color | N | Pale & Cold | Pale & Cold | Grey & Ashen |
| Mental Status | Alert | Anxious | Anxious & Agressive | Drowsy/ Confused/ Unconcio us |

# MONITORING OF HYPOVOLEMIC SHOCK

- PR
- BP→ < systolic, ^ diastolic (vasoconstriction) & , PP
- RR→ ^ rate & depth ( if not – check for resp or CNS abn
- UOP→ <, & to access fluid resuscitation
- CVP→ <
- SWAN-GANZ CATHETER→ to measure

(a) Cardiac output—by thermodilution method, greater the temp fall slower is the circulation

# MONITORING OF HYPOVOLEMIC SHOCK

(b) Mixed venous O2 levels—Pco2 of pulm artery blood is 4 mm of Hg higher than systemic arterial blood

(c) Vascular pressures– Rt atrial pressure & pulm artery systolic (25 mm Hg) & diastolic pressure (10)

(d) PCWP (N-8-12 mm Hg or 11-16 Cm of water)-- > in LVF, < in pulm embolism, guide to therapy with fluids

- ECG- Cardiac ischemia, Hb & HCT
- RFT, Blood gases & electrolytes, BG & CM

# MANAGEMENT OF HV SHOCK

- AIRWAY→ maintain patency—remove foreign body from mouth, nose, pharynx. Extend neck. Give O2 by ventury mask 6 lit/min
- BREATHING→ IPPV or Endotracheal intubation or tracheostomy if needed

Adv- expansion of alveoli- pushes blood from pulm vasculature to lt atrium- ^ cardiac output

- CIRCULATION→

A. Immediate control of bleeding- compression bandage or ligation of blood vessels

# MANAGEMENT OF HV SHOCK cont….

- Improve circulation-- Head low & leg rise position. It- ^ venous return from leg muscles, ^ cerebral circulation, < periferal oedema
- Extracellular Fluid Replaement→

<u>Kind of fluid</u>

crystalloids preferred= non-protein, non sugar (osmotic diuresis) With Na concentration near to plasma—NS OR RL

If blood loss—Fresh BT (*stored blood--^ K+--< cardiac fn) is better than colloids (*problem with CM & ^ blding with colloids)

<u>Dose—</u>1-2 lit within 45 mins

# MANAGEMENT OF HV SHOCK cont….

Route—wide bored IV cath or needle via accesible vein or do venesection if needed

NaHCo3 1 amp iv to combat acidosis

- If no improvement with 3 lit fluid or transient effect→ search for blding & Rx it.
- DRUGS→ IF NEEDED

A. Sedatives

for pain due to ass inj= #, head inj, crush inj etc.

Adult- morphin or pethidine(* causes vasodilatation) children- phenobarbitone, head inj-phenytoin (* less resp depression)

# MANAGEMENT OF HV SHOCK cont….

B. Chronotropic agents--^ses HR (used in bradycardia pts), Atropine, Isoproternol ( adv-Vasodilatation of systemic arterial & capillary sphincters)

C. Ionotropic agents—In low dose causes ^ myocardial contractility but in hgh dose—vasocontriction--< perfusion. Dopamine & Dobutamine(* better renal perfusion)

D. Vasoconstricters—Phenylephrine & metaraminol(* ^ PR, BP, Cardiac contractiity), seldom used

# TRAUMATIC SHOCK

- Similar to haematogenic shock along with associated problems i. e.
- Head injury
- Chest injury-Pericardial effusion,S heart contusion, cardiac temponade ( ass with cardiogenic shock ). –Lung contusion, haemothorax ( ass with hypoxia )
- GIT- Liver, splenic, mesentric vein tear (as with toxin release)

# TRAUMATIC SHOCK cont…

- Pathophysiology—similar to HVS ass with

1. Traumatized tisue→microthrombi→pulm vasculature blockae→^ Rt vent diastolic pressure→ ^ Rt atrial pressure→ < venous return
2. Humeral  prducts of micro-thrombi→generalizes ^ cap perm→^ transudation→Periferal oedema & < IV volume

- S/S---similar to HVS + periferal & pulm oedema & more fluid required for resuscitation

# TRAUMATIC SHOCK cont…

- Mx—

1. Resuscitation
2. Local Rx of trauma & control of bld—Immobilize #s & surgical debridement
3. < microthrobi—heparin 10000 units sc

# CRUSH SYNDROME

*Earthquake, mine etc.

- Pathophysiology→ Muscle crush→

1. Necrosis→ SIRS→ HVS
2. Extravasation of blood→ compartment syndome →HVS
3. Myoglobinuria→ ATN→Renal failure

- Mx

1. Resuscitation
2. Tourniquet proximal to crush
3. Debridement & parallel incisions to relieve compartment
4. Diuretics—Mannitol 20% 1gm/kg
5. Haemodialysis
6. Urinary cath & Monitor UOP

# SEPTIC SHOCK

- ETIOLOGY

Gram +ve cocci—staph, strepto, pneumo(skin, subcu, lung inf)

Gram –ve bacilli---E. coli, klebsiella, proteus, pseudomonas, bacteriodes (RS-lung abscess, pneumonia. GIT-cholecystitis, cholangitis, peritonitis, intra-abdominal abcess. GUT-uti, pyelonephritis, perinephric abscess, prostatitis, septic abortion

Viruses, Fungi & parasites

# SEPTIC SHOCK cont..

- PATHOGENESIS
- EARLY WARM STAGE

Inf focus→toxin production localy→^ body temp→dilat of skin vasculature→fluid loss→< periferal resistance & adrenergic discharge→ ^ CO & ^ PR but low BP

- LATE COLD STAGE

Liberation of toxic products in circulation→^ vascular permeability→HVS

# SEPTIC SHOCK cont..

- S/S
- Early warm stage-- fever > 100*F with chills, PR ^, < BP, Warm & pink skin, Cutaneous veins full
- Late cold stage—all s/s of HVS except presence of inf focus
- Ix
- CBC with ESR & HCT, Urine R & M, X-ray chest, USG Abd, Lapaoscopy, Cystoscopy, Pus/urine/blood C/S

# SEPTIC SHOCK cont..

- D/D & MONITORING-with HVS same as HVS=PR, BP, RR, UOP, CVP etc.
- Mx

1. Rx of infective foci—Drainage, debridement, Antibiotics—empirical before c/s report available (broad spectrum with against anaerobic inf=3$^{rd}$ generation cephalosporins-cefotaxime,ceftriaxone, ceftazidime with gentamycin or amikacin with metronidazole. Chloromycetin & clindamycin if bacteroid inf suspected
2. Rx of shock-ABC

# SEPTIC SHOCK cont..

3. DRUGS

- Vasoactive—Chronotropic, Ionotropic, Both alfa & beta adrenergic stim=metaraminol, vasodilator=phenoxybenzamine
- Steroids—
  - High dose short term-methyle prednisolon 15-30 mg/kg or dexamethasone repeat after 4 hrs if needed
  - Rational-little immunosupression but better benificial effects ie=1. ionotropic effect 2. protects body cells against efeects of endotoxins 3. improves cardiac, pulm & renal fns

# CARDIGENIC SHOCK ( PUMP FAILURE )

- ETIOLOGY
- IHD, Myocarditis, cardiomyopathuy,MI, CCF, cardiac arrythmia, heart injury
- MASSIVE PULM EMBOLISM-50 % PA occlusion→RVF→ < Lt ventricular filling
- CARDIAC COMPRESSIVE SHOCK- Pericardial effusion, tension pneumothorax, large diaphragmatic hernia

# CARDIOGENIC SHOCK cont..

- S/S→

RVF- < BP, ^ PR, NVE, Periferal oedema, ascites, hepato/ spleno megali

LVF- < BP, ^ PR, crepts, dyspnoea, S3

- DIAGNOSIS & D/D
- Mx

** Pulm embolism-Heparin

- < Preload—Diuretics=furosemide
- Cardiotonics--Ionotropics & chronotropics, Beta- blockers
- < Afterload-vasodilators= nitroprusside, nitroglyserine