TRAUMATIC SHOCK

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DEFINITION

- **Shock** it is a severe disturbance of hemodynamic in which the circulatory system fails to maintain adequate perfusion of vital organ.
- **Traumatic shock** is characterized by severe tissue. damage, such as multiple fractures, severe contusions, or burns.

SHOCK IN TRAUMA

- Classification

1. Hypovolemic
2. Distributive
3. Cardiogenic

HYPOVOLEMIC SHOCK

- MOST COMMON CAUSE OF SHOCK IN THE TRAUMA PATIENT

1. DUE TO HEMORRHAGE (LOSS OF RBCS IMPAIRS OXYGEN TRANSPORTATION
2. IN ANY TRAUMA PATIENT WITH SHOCK, ASSUME HAEMORRHAGE IS CAUSE UNTIL PROVEN OTHERWISE

DISTRIBUTIVE SHOCK

- NEUROGENIC SHOCK

1. DECREASED SYSTEMIC VASCULAR RESISTANCE DUE TO VASODILATION
2. MOST COMMON CAUSE IN SPINAL CORD INJURY

CARDIOGENIC SHOCK

- INTRINSIC

1. BLUNT CARDIAC TRAUMA LEADING TO MUSCLE DAMAGE AND DYSRHYTHMIA
2. VALVULAR DISCRUPTION

- EXTRINSIC

1. PERICARDIAL TAMPONADE
2. TENSION PNEUMOTHORAX

TRAUMATIC SHOCK MECHANISM

- Microcirculation –Systemic vascular resistance rises to maintain a level of systemic pressure that is adequate for perfusion of the heart and brain at the expense of other tissue. Arteriolar vascular smooth cells has both α- and β–adrenergic receptors. Norepinephrine release - acting on α 1 -receptors as vasoconstrictor - is the fundamental compensatory response in shock. –Reduced filtration because of decreased capillary surface area across which filtration occurs.
- Consequence: increased interstitial and intravascular volume at the expense of intracellular volume.

TRAUMATIC SHOCK MECHANISM

- Cellular response –Decline of intracellular high energy phosphate stores (decreased amount of ATP) because of the mitochondrial dysfunction.
- Consequences: Accumulation of hydrogen ions, lactate (products of anaerobic metabolism) As shock progresses, these vasodilatation metabolites cause further hypotension and hypo perfusion

TRAUMATIC SHOCK MECHANISM

- Cardiovascular response –Decreased ventricular filling (decreased preload). The increased heart rate is a useful but limited compensatory mechanism to maintain the adequate stroke volume –Impaired myocardial contractility which reduces the stroke volume –Elevated systemic vascular resistance (except of hyper dynamic stage of septic shock) increases the afterload

TRAUMATIC SHOCK MECHANISM

- Pulmonary response –Increased pulmonary vascular resistance (particularly in septic shock) –Tachypnoe, but restricted ventilation, reduced functional residual capacity – atelectasis. –Acute respiratory distress syndrome characterized by noncardiogenic pulmonary oedema secondary to pulmonary capillary endothelial and alveolar epithelial injury

TRAUMATIC SHOCK MECHANISM

- Renal response –Consequences of hypo perfusion: reduced renal blood flow, increased afferent arteriolar resistance – reduced glomerular filtration rate together with the increased aldosterone and vasopressin production will cause reduced urine volume –Acute tubular necrosis as a result of interaction of shock, sepsis and administration of nephrotoxic agents

TRAUMATIC SHOCK MECHANISM

- Inflammatory responses –Activation of an extensive network of proinflammatory mediator systems plays a significant role in the progression of shock and contributes to the development of organ injury Activation of classic and alternative pathways of complement cascade causing cell damage Activation of coagulation cascade causes microvascular thrombosis Tumour necrosis factor-α, produced by activated macrophages contributes to hypotension, lactic acidosis, and respiratory failure IL-8 upregulate adhesion molecules on the neutrophil to enhance aggregation, and damage to the vascular endothelium Increased Thromboxane A 2 levels is potent vasoconstrictor that contributes to the pulmonary hypertension

CLINICAL MANIFESTATION

1. PRESENCE OF PERIPHERAL AND PULMONARY EDEMA
2. INFUSION OF LARGE VOLUME FLUID WHICH MAY BE ADEQUATE FOR PURE HYPOVOLEMIC SHOCK IS USUALLY INADEQUATE FOR TRAUMATIC SHOCK
3. TACHYCARDIA AND TACHYPNEA
4. WEAK, THREAD PULSES
5. HYPOTENSION
6. COOL AND CLAMMY SKIN
7. MENTAL STATUS CHANGES
8. DECREASES URINE OUTPUT (DARK AND CONCENTRATED)

TREATMENT AND MANAGEMENT

PRE-HOSPITAL CARE:

1. EXTERNAL BLEEDING SHOULD BE CONTROLLED BY DIRECT PRESSURE
2. IMMOBILIZATION PATIENT
3. SECURING ADEQUATE AIRWAYS
4. ENSURING VENTILATION
5. MEDICATION TO INCREASE THE HEART PUMPING ABILITIES (DOBUTAMINE,EPINEPHRINE,NOREPINEPHRINE)

THANK YOU