Clinico-Pathological aspects of Shock

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Abstract

Shock can be defined as "a common grave medical emergency characterised basically by reduction in the effective circulating blood volume and blood pressure". (Robbins) or as "Disparity between the volume of blood and the volume capacity of the vascular system" which cause inability of body tissue to metabolise nutrients due to inadequate oxygen supply. Shock can be classified into: Primary and Secondary.Cardiogenic, Vasogenic, Hematogenic, Neurogenic and Electrocution. The therapy includes Blood, Plasma, Saline transfusion, antibiotic, antihistaminic, hyperimmune serum, vasoconstrictor according to the cause of the shock.

Keywords: Clinical, Pathological, Shock, Blood pressure, Blood Volume,Trauma.

Primary shock (Traumatic shock): It develops immediately after injury, trauma or extensive surgical wounds during operation and massive handling of internal abdominal organs. It is of nervous origin, transient in nature causing widespread capillary paralysis. In case of human beings, psychic status such as fear, excitement, anxiety, frightening, intense pain and apprehension may lead to primary shock. In case of wild or timid animals, due to restraining or compulsory exercise during circus, the condition may develop. Primary shock is similar to syncope or fainting.

Secondary shock: It terminates fatally due to more severity. The essential feature of this shock is that there is disproportion between the volume of blood and volume of blood vascular space. Sufficient blood is not present to fill the blood vessels and to maintain the blood pressure leading to non availability of blood for pumping out through heart.

Causative factors

A. Hypovolemic shock due to reduction in the blood volume:

1. It is caused due to injuries, loss of fluid, plasma, blood, erythrocyte and plasma proteins loss due to hemorrhage within and outside the body. e.g. severe burns, crushing injuries, persistent vomiting and diarrhea.
2. Loss of fluid, electrolytes, water loss, dehydration and deprivation occurs in pyloric stenosis leading to sodium deficiency, Addisson's disease and diabetic coma.
3. Intense edema occurs in poisoning by war gases (phosgene, mustard gas, lewisite) and poisons like ANTU.

B. Capillary bed dilatation: Normally some capillaries are patent while others are closed but if all the capillaries get dilated, blood will be pooled in them and so diminished blood is available for the heart to pump. Determinative factors may be neurogenic stimuli, bacterial toxins, toxic metabolic products and anorexia.

C. Acute circulatory failure: If the heart suddenly fails in conditions like infarction of myocardium, paroxysmal tachycardia, cardiac tamponade, massive pulmonary embolism, the circulation cannot be maintained, blood pressure is reduced due to reduced blood volume, cardiac output is low resulting into shock and coma.

Pathogenesis of shock

1.Ischemia: Extensive hemorrhage, burns in which there is reduced blood volume, blood circulation is inadequate, reflex contraction of some arterioles cause resultant ischemia, degeneration, fibrosis and necrosis, loss of function of the tissues and organs like liver, kidney and heart.

2.Toxic or Septic shock: Normally muscles and liver harbour certain bacteria, but if greater number of virulent and toxin producing bacteria are present in

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intestine which produce potent toxins gain access into circulation resulting vasomotor collapse leading to shock. In response to shock detoxifying mechanism of the body is activated in the initial stage later on get exhausted, condition gets aggravated resulting into irreversible shock. Potent endotoxins enter into circulation. e.g. Gram negative sepsis, acute diffused peritonitis, acute gangrenous mastitis, acute metritis, per acute coliform mastitis, acute intestinal accidents and infarction of large segment of intestine.

3.Vasotropic principles: Whenever there is anorexia, ischemia the cortex of the kidney produces vaso excitatory material (VEM) and liver, skeletal muscles and spleen produces vaso - depressor materials (VDM) probably ferritin under anaerobic conditions. In the initial stage of shock, there is decreased blood volume, VEM is produced to effect vaso constriction as a compensatory mechanism but if the shock continues there is tissue hypoxia, fall in blood pressure. Decreased anaerobic respiration cause tissue ischemia and hypoxia leading to anaerobic glycolysis, loss of energy and reduced protein synthesis, since ATP is not formed. Accumulation of pyruvic acid, lactic acid leading to acidosis and release of lysosomal enzymes which injure the cells leading to irreversible shock. To combat the shock the defence mechanism of the body gets activated and VDM is produced causing capillary dilatation.

1. Hematogenic shock or traumatic shock: It occurs when there is reduction in the circulating blood volume due to blood or fluid loss or hemorrhages more than 35 percent of the total blood volume, fluid loss due to neonatal calf diarrhea or colitis-X, acute intestinal obstruction and dehydration. e.g. trauma, extensive burn, rough handling of visceral organs during surgery.

2. Vasogenic shock: It occurs when there is peripheral vasodilatation and pooling of blood in the vessels, leakage into the tissues resulting into reduction of the effective circulating volume e.g. Severe burns, extensive surgical wounds, prolapse of uterus, rapid withdrawal of ascetic fluid, severe colic pain in horses and trauma to blood sequestration i.e. epithelium or endothelium of blood vessel.

3. Nervous shock: It is caused due to an acute lesion in the central nervous system, damage to the nerve cell or spinal cord. There is temporary cessation of function. e.g. stunning, irritation of nerves due to bacteria or virus, hypoglycemia. The nervous shock leads to lesions like nerve paralysis.

4. Electrocution/ Lightning shock: Exposure to high voltage electric current or flashes of linear lightning during thunderstorm, broken overhead electrical transmission wire with high voltage, faulty wiring in cowshed or byre leads to destruction of nervous tissue and death due to paralysis of vital medullary centre, ventricular fibrillation.

Clinical Symptoms

1. Generalized clinical symptoms: Dehydration, pink but dry mucous membrane, sunken eyes, loss of elasticity of the skin, anorexia, extremities are cold, increased heart beat (120-140/minute) having small amplitude in horse and cattle, tachycardia, weak, rapid imperceptible and thready pulse, cardiac arrhythmia, gastro intestinal stasis, increased thirst, respiratory rates are increased and shallow, sobbing type of respiration is observed in horse and dog, cheyne or stoke´s respiration is recorded, conjunctiva is brick red, congested, clonic convulsions, obtusia. Superficial veins are collapsed, yawing in horses and camels.

2. Clinical symptoms in neurogenic shock: Facial paralysis, paraplegia, muscular tremors, disappearance of cutaneous sensitivity, extremities are cold, animal is unable to rise, sternal or lateral decumbency, interference with the function of respiratory muscles, anesthesia due to caudal nerve lesion, paralysis of the sacral nerve causes loss of function of the bladder and rectum.

3. Clinical symptoms in electrocution shock: Burns may be localized to muzzle and feet in the form of radial deposits of carbon with blackening of the tissues and organs. The clinical symptoms observed are unconsciousness, struggling, depression, blindness, unilateral posterior paralysis, monoplegia, cutaneous hyperesthesia and nystagmus.

Post complications of Shock

1. Renal insufficiency: Renal parenchyma, cortex, glomeluri become ischemic due to renal vaso-constriction and vascular collapse , inflammatory edema develops due to blockage of renal tubules due to pigment cast. Therefore renal functions get suppressed with resultant oliguria, anuria and uremia.

2. Cardiac failure: fatty degeneration of myocardium may cause cardiac failure and death

3. Cerebral ischemia: Decreased blood pressure leads to insufficient supply of blood to brain leading to anoxia, neuronal degeneration, cerebral edema, encephalomalacia, meningitis, encephalitis and death.

4. Pulmonary infection: Edematous fluid is a good medium for growth of bacteria, fungi, rickettsia, and viruses. In case of pulmonary edema there is fatal respiratory affection due to super imposed infection of the lung.

Treatment

1. Replacement therapy :-

a) Direct method: Injection of whole blood or blood transfusion @ 5.5 cc/ kg body weight.

b) Indirect method: Transfusion of plasma @ 10 cc/ kg body weight.
c) Transfusion of normal saline or dextrose normal saline or Ringer's solution or plasma volume expanders. Eg. 25-50 gms of albumin/lit of fluid, large volume of isotonic solutions, crystalloid solution, electrolytes, Ringer's lactate, solution which maintains the acid base balance, small volume of hypertonic solution, dextran, gelatin, polymers, hexa starch.
d) Antibiotic therapy: To combat infectious or septic shock.
e) Use of anti histaminic drugs. e.g. Chlorphenarimine maleate (CPM)
f) Use of anti allergic or life saving drugs like Dexamethasone.
g) Use of cyclooygenase inhibitor. Eg. Flunixin mgelumine(0.25/kg body weight) or ketoprofen @ 0.5-2.2 mg/kg body weight, Tirilazid mesylate
h) Use of vaso constrictors: i) Alfa adrenergic blockers
ii) Selected catechol amines
iii) Immuno therapy
i) Use of hyper immune serum.
j) Use of drugs acting on cardio vascular system, adrenaline, epinephrine, digitalis, methoxamine, nor-epinephrine, metaraminol, dopamine, iso protenol, phenoxy benzamine
k) Treatment for counter acting acidosis: Sodium bicarbonate along with distilled water or dextrose.
l) Proper ventilation is provided by administering artificial oxygen or placing the animal in oxygen chamber or performing an emergency tracheotomy operation.
m) Treatment for reducing ammonia content of blood. Ammonia content increases during shock which is detoxified by increasing basal metabolic by drug acting on liver.
n) Use of Vitamin C: it is used for the stimulation of adrenal cortex and reducing the blood clotting activity.
o) Blood lactate concentration in horses above 8.3 mmol / L indicates increased risk for mortality and level above 11.2 mmol / L indicates fatal outcome, hence the condition may be treated by fluid replacement therapy.
p) Monitoring and maintaining of systolic blood pressure: Central Venous Pressure (CVP) of the standing horse is recorded as 11.5 to 5.6 Cm H2O, which is markedly influenced by head position and excitement. Therefore efferts should be made to keep CVP below 5 Cm H2O to combet shock.

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