Anaphylactic shock

It is a medical emergency that requires immediate recognition and intervention. Acute hypersensitivity reaction in response to allergic substance (Drug, Food or venom) → degranulation of mast cells → massive histamine release → systemic vasodilation and increased capillary leakage.

Clinical features: rapid onset of symptoms (minutes to hours): tachycardia, tachypnea. hypotension, flushed, itchy skin. bronchospasm, laryngeal edema → wheeze, stridor, dyspnea, cyanosis. swelling of conjunctiva, lips, tongue and/or uvula, angioedema.

Treatment

- Epinephrine 0.5 ml i.m. It is the drug of choice and potentially lifesaving. i.m. injection in the thigh (vastus lateralis) gives more rapid effect. Repeat after 5 min if no response.
- Antihistamine (e.g. chlorpheniramine 10 mg i.v.).
- Hydrocortisone 200 mg i.v to ↓ antigen/antibody reaction.
- Intravenous fluids (0.5-1 L) and monitor BP.

Complications: Airway obstruction and cardiovascular collapse.

Neurogenic shock

Neurogenic shock is caused by loss of sympathetic tone of blood vessels resulting in the massive dilatation of arterioles and venules. It can be caused by spinal anesthesia, spinal cord injury, cerebral hemorrhage and traumatic brain injury.

Pathophysiology: damage of autonomic pathways → loss of sympathetic vascular tone → unopposed vagal tone → peripheral vasodilation → pooling of peripheral blood

Clinical features: Bradycardia, hypotension and flushed, warm skin. Other clinical features related to the underlying disease: neurological deficits (e.g., flaccid paralysis in spinal trauma)

Treatment: Fluid resuscitation, atropine to treat bradycardia. vasopressors: if fluid resuscitation fails to increase MAP beyond 90 mmHg

- Normal heart rate: phenylephrine
- If heart rate < 60/min: epinephrine

Cardiogenic shock

Etiology: The most common cause of cardiogenic shock is LV dysfunction and necrosis as a result of acute myocardial infarction (AMI). Acute valvular insufficiency or stenosis prevents the normal ejection of blood. Besides ventricular septal or free wall rupture.

Pathophysiology: Compensatory mechanism to decrease in COP, include an increase in sympathetic tone (seen clinically as increased HR and peripheral vasoconstriction) which initially serve to increase CO and maintain central arterial pressure. Decrease in CO result in a decreased perfusion to vital tissues and organs, also the decrease in CO leads to a reduction in the flow of blood through the coronary arteries, which can lead to infarct extension and a further worsening of cardiac performance.

Clinical manifestation of cardiogenic shock

Sign and symptoms similar to that of hypovolemic shock (hypotension, tachypnea and tachycardia, oliguria and cold extremities) but patients frequently have signs of volume overload because the heart cannot move blood through the circulation. Peripheral edema can be seen in the extremities; lung sounds are diminished, and rales may be present as pulmonary edema develops. These findings are particularly evident in patients with severe HF. Hypovolemia occurs in up to 20% of patients in cardiogenic shock. There is Reduced CO, BP, but elevated PCWP and SVR (systemic vascular resistance).

Useful diagnostic measures: chest x ray to check pulmonary edema, ECG and ECHO to detect cardiac abnormalities.

Treatment options of cardiogenic shock: Fluids, vasodilators and inotropes.

A. Fluids

Augmentation of preload with a fluid to improve CO is the first option. However, if there is pulmonary edema or if PCWP is more than 18 mmHg, or If the PCWP rises but the CO does not improve, fluid should be discontinued since there is no benefit from fluid therapy instead, they may worsen pulmonary edema. Additionally, elevating the preload without improving CO can increase LV wall tension, which is a major determinant of myocardial oxygen consumption; consequently, myocardial ischemia could develop.

B. Vasodilators

A peripheral vasodilator acts to decrease pulmonary venous congestion by reducing preload. It will improve CO by decreasing the resistance to ventricular ejection (afterload) as well. In patients with myocardial ischemia, vasodilators improve subendocardial blood flow, reduce the myocardial wall tension, and reduce the LV radius. The resultant decrease in myocardial oxygen consumption will help prevent further depression of cardiac function. Vasodilators in patients with LV failure (HF), lower elevated SVR, that occur due to reflex increase in sympathetic tone in response to a fall in systemic arterial pressure, so vasodilators improve ventricular function. One of major limitation of using vasodilators is blood pressure reduction, so Vasodilators should be reserved for situations in which hemodynamic monitoring shows the patient to have LV failure with elevations in PCWP and SVR, and a SBP greater than 90 mm Hg.

C. Inotropic support

A rapid-acting inotropic agent (e.g., dopamine, dobutamine, epinephrine) also can be used to increase myocardial contractility and CO. The disadvantage of this intervention is that improved CO is accompanied by an increased myocardial oxygen demand. So, if PCWP elevated, while BP is low the inotropes are the best choice to improve COP and BP. If the BP stabilized vasodilators can be tried. If there is concomitant pulmonary edema, diuretics can be started when BP elevated to normal values.