

CARDIOGENIC SHOCK: CLINIC, EMERGENCY CARE.

G.T. Mamedova

assistant at the Department of Anatomy and Clinical Anatomy at the Bukhara State Medical Institute

ANNOTATION

Cardiogenic shock is a life-threatening condition of critical organ hypoperfusion (lack of blood supply), due to a decrease in cardiac output, which is characterized by: 1. A decrease in SBP <90 mmHg . for more than 30 minutes, mean blood pressure less than 65 mm Hg . for more than 30 minutes, or the need to use vasopressors to maintain SBP≥90 mmHg .

KEYWORDS

cardiogenic shock, heart failure, congestive heart failure, acute heart failure, cardiac failure

Cardiogenic shock is a life-threatening condition of critical organ hypoperfusion (lack of blood supply), due to a decrease in cardiac output, which is characterized by: 1. **A decrease in SBP <90 mmHg** . for more than 30 minutes, mean blood pressure less than 65 mm Hg . for more than 30 minutes, or the need to use vasopressors to maintain SBP≥90 mmHg ; 2. **Signs of pulmonary congestion or increased** filling pressure of the left ventricle; 3. Signs of **organ hypoperfusion** , at least the presence of one of the following criteria: impaired consciousness; •cold, damp skin;• oliguria ; •increase in plasma serum lactate > 2 mmol/l.

There are 3 main clinical variants of cardiogenic shock :

- **arrhythmic shock** due to a drop in minute blood volume during tachycardia/ tachyarrhythmia or bradycardia/ bradyarrhythmia . After stopping the arrhythmia, hemodynamics are restored quite quickly;
- **reflex shock** (pain collapse), which develops as a reaction to pain; characterized by a rapid response to analgesic therapy;
- **true cardiogenic shock** is characterized by a detailed picture of shock that is resistant to therapy.

Family doctor strategy: effective pain relief, preparation for urgent evacuation by intensive care unit to a specialized hospital.

CLINIC : symptoms of ACS are possible (detailed in the relevant protocols) or signs of non-ischemic heart damage , along with the appearance of signs of acute hemodynamic failure and hypoperfusion : severe general weakness, dizziness, “fog before the eyes”, palpitations, a feeling of interruptions in the heart, suffocation . *gray cyanosis or pale cyanotic, “marbled”, moist skin* ; acrocyanosis ; collapsed veins; cold hands and feet; nail bed test for more than 2 s. (decreased peripheral blood flow velocity). Impaired consciousness: lethargy, confusion, less often - agitation. Oliguria (decrease in urine output less than <0.5 ml/kg/h). Decrease in systolic blood pressure less than 90 mmHg ; decrease in pulse arterial pressure up to 20 mm Hg . and below., decrease in average blood pressure less than 65 mm Hg (formula for calculating average blood pressure = (2DBP + SBP) / 3).

Family doctor tactics:

- the patient must be given a horizontal position with slightly elevated lower limbs (in the absence of signs of left ventricular failure);
- oxygen therapy with a mask or through nasal catheters;

- effective pain relief as in myocardial infarction, relief of rhythm disturbances;
- in case of reflex shock, when the effect of pain relief is insufficient, *a mezatone solution can be administered slowly intravenously* (0.5 ml of the drug in 20 ml of isotonic sodium chloride solution or 5% glucose solution);
- if it is not possible to immediately hospitalize the patient (and if the necessary medications are available), a test bolus injection of 100 ml of rheopolyglucin is carried out with repeated administration of 50 ml every 5 minutes until a systolic blood pressure of 100 mm Hg is achieved. Art. (a single dose of rheopolyglucin should not exceed 400 ml), and/or dopamine (200 mg of the drug is dissolved in 125 ml of 5% glucose solution and administered intravenously, starting with a dose of 3-5 mcg/(kg•min), gradually increasing until the effect is achieved, or the maximum dose – 25 mcg/(kg•min) – if complications develop). Instead of dopamine, dobutrex or norepinephrine can be used ;
- in the absence of contraindications, in order to correct microcirculatory disorders, heparin is prescribed at a dose of 10,000-15,000 IU intravenously, with continued therapy in the hospital.
- infusion (NaCl or Ringer's solution >200ml/15–30min) recommended as first-line therapy in the absence of signs hypervolemia .
- For inotropic purposes (to increase cardiac output) they are used dobutamine and levosimendan (the use of levosimendan is especially indicated for development of CABG in patients with CHF taking β - blockers). Infusion dobutamine is administered at a dose of 2–20 mg/kg/min. Levosimendan can be administered in a dose 12 mcg/kg over 10 minutes, then infusion of 0.1 mg/kg/min, reducing the dose to 0.05 or increasing if ineffective to 0.2 mg/kg/min. At the same time, it is important so that the heart rate does not exceed 100 beats/min. If tachycardia or disturbances develop heart rate, the dose of inotropes should be reduced if possible.
- Vasopressors should be used only when it is impossible to achieve target SBP values and elimination of hypoperfusion symptoms during therapy infusion solutions and dobutamine / levosimendan . The vasopressor of choice should be norepinephrine. Norepinephrine is administered at a dose of 0.2–1.0 mg/kg/min.
- Loop diuretics - used cautiously when combined clinically cardiogenic shock with acute left ventricular failure, only on background of normalization of blood pressure numbers.

Literature

1. Rogers WJ, Frederick PD, Stoehr E. et al. Trends in presenting characteristics and hospital mortality among patients with ST elevation and non-ST elevation myocardial infarction in the national registry of myocardial infarction from 1990 to 2006 // Am. Heart J. 2008. Vol. 156, N 6. P. 1026–1034.
2. Harker M., Carville S., Henderson R. et al. Key recommendations and evidence from the NICE guideline for the acute management of ST-segment-elevation myocardial infarction // Heart. 2014. Vol. 100, N 7. P. 536–543.
3. Jernberg T., Johanson P., Held C. et al. Association between adoption of evidence-based treatment and survival for patients with ST-elevation myocardial infarction // JAMA. 2011. Vol. 35, N 16. P. 1677–1684.
4. O'Flaherty M., Buchan I., Capewell S. Contributions of treatment and lifestyle to declining CVD mortality: why have CVD mortality rates declined so much since the 1960s? //Heart. 2013. Vol. 99. P. 159–162.



5. Reynolds HR, Hochman JS Cardiogenic shock: current concepts and improving outcomes // *Circulation*. 2008. Vol. 117. P. 686–697.
6. Pirrachio R., Parenica J., Rigon MR et al. The effectiveness of inodilators in reducing short-term mortality among patients with severe cardiogenic shock: a propensity-based analysis // *PLoS One*. 2013. Vol. 8, N 8. Article ID e71659.
7. Axler O. Low diastolic blood pressure as the best predictor of mortality in cardiogenic shock // *Crit. Care Med*. 2013. Vol. 41, N 11. P. 2644–2647.
8. Stout KK, Verrier ED Acute valvular regurgitation // *Circulation*. 2009. Vol. 119, N 25. P. 3232–3241.