Heart failure (neonatology)

This article has been translated from WikiSkripta; ready for the **editor's review**.

Heart failure occurs when cardiac output is unable to meet the body's metabolic needs. A decline in cardiac output triggers a cascade of compensatory mechanisms to maintain organ and tissue perfusion.[1] In newborns, it develops most often on the basis of myocardial dysfunction, left-sided obstructive lesions, tachyarrhythmias and large arteriovenous malformations. The consequence is tachycardia, tachypnea with retraction, hepatomegaly, cardiomegaly. In the later period, the most common cause of heart failure is a large left-right shunt (large ventricular septal defect (VSD) or large persistent Botall's aneurysm (PDA)), which manifests after a decline in pulmonary vascular resistance.[2]

Causes

1. heart malformations:
   - shunts: ventricular septal defect (VSD), patent ductus arteriosus of Botall (PDA), aortopulmonary window, atrioventricular septal defect, single ventricle (*single ventricle*) without pulmonary stenosis, rarely atrial septal defect;
   - Total/Partial Anomalous Pulmonary Venous Connection;
   - valvular regurgitation: mitral regurgitation, aortic regurgitation;
   - outflow obstruction: *cor triatriatum*, pulmonary stenosis, mitral stenosis;
   - outflow obstruction: aortic valve stenosis, subaortic stenosis, supravalvular aortic stenosis, aortic coarctation.
2. cardiac causes in a structurally normal heart:
   - cardiomyopathy, myocarditis, arrhythmias; hypertension;
3. non-cardiac:
   - anemia;
   - sepsis
   - hypoglycemia; diabetic ketoacidosis;
   - hypothyroidism, other endocrinopathies;
   - renal failure;
   - muscular dystrophies.[1]

Clinical picture

- acute cardiorespiratory collapse;
- respiratory distress;
- tachycardia (unless the cause of heart failure is a conduction system block);
- hepatomegaly;
- poor peripheral circulation, marbled skin, cold sweat;
- oedema, pericardial, pleural and peritoneal effusion;
- excessive or unexpected weight gain;
- feeding difficulties, low weight gain.[2]

Treatment

Treatment of acute heart failure

- ventilatory support according to the condition;
- volume expansion, inotropic support;
  - catecholamines: dopamine, noradrenaline, adrenaline, dobutamine;
  - vasopressors are drugs that induce vasoconstriction and thus increase mean arterial pressure (dopamine, noradrenaline, adrenaline);
  - inotropics are drugs that increase cardiac contractility (dobutamine, milrinone);
  - some drugs have both vasopressor and inotropic effects;
  - the effect of these drugs is mediated by adrenergic and dopamine receptors, among others:
    - alpha-1 adrenergic receptors: in the vessel wall → vasoconstriction; in the heart → prolongation of contraction duration without increasing heart rate; (noradrenaline, adrenaline > dopamine);
    - beta-1 adrenergic receptors: in the heart → stronger contraction and faster heart rate (inotropic and chronotropic effect) with minimal vasoconstriction; (dobutamine, adrenaline > noradrenaline, dopamine in medium and higher doses);
    - beta-2 adrenergic receptors: in vessel wall → vasodilation; (dobutamine, adrenaline);[3]
  - **dobutamine**: pure adrenergic agonist (β1 > β2 > α receptors); improves myocardial contractility; reduces end-diastolic pressure in the left ventricle and increases blood pressure by increasing cardiac output; may induce hypotension by peripheral vasodilation (β2 receptors);
  - **dopamine**: adrenergic and dopaminergic receptor agonist in a dose-dependent manner; low doses → vasodilation including coronary and renal arteries; medium doses → inotropic and chronotropic effect, but also increase in pulmonary capillary pressure; high doses → α-receptor-mediated vasoconstriction predominates, afterload increases;

    - **noradrenaline**: endogenous catecholamine; stimulates β and α adrenergic receptors → inotropic and chronotropic effect and peripheral vasoconstriction;
    - **milrinone**: phosphodiesterase inhibitor; inotropic effect (but not via β1 receptors) and peripheral vasodilation;[4]
- fluid restriction to about 2/3;
- furosemide i.v. 1 mg/kg every 6-12 hours;
- optimization of oxygenation, not hyperoxia;
- correction of anaemia.[2]

Treatment of chronic heart failure

- normal amounts of fluids (not restriction) in an effort to maximize energy intake;
- optimizing caloric intake (hypercaloric nutrition, supplements, nasogastric tube feeding);
- oral diuretics: furosemide 2-6 mg/kg/day (up to 2-3 doses), potassium-sparing diuretics or potassium supplementation;
- in myocardial dysfunction, ACEi and/or digoxin may be considered;
- correction of anaemia.[2]

Links

References

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