

Acute right ventricular failure

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Acute right ventricular (RV) failure is a serious clinical challenge not only in cardiology intensive care but in adult critical care in general. It is related not only to left ventricular failure but also to a number of other causes of pulmonary vascular dysfunction and pulmonary hypertension (PH), for example, PH due to hypoxia, thromboembolism, mechanical ventilation or portopulmonary hypertension. Acute RV failure is often difficult to manage and it is associated with a worse prognosis. Mortality from cardiogenic shock due to RV infarction (>50%) exceeds that due to left ventricular disease. Acute RV failure is also related to increased mortality after surgical procedures. The general principles of management include pre-load optimization, increase in RV inotropy and after-load reduction by lowering pulmonary vascular resistance. This review focuses on the principles of RV failure management including general measures, vasoactive substances, ventilation strategies, surgical as well as mechanical approaches.

Key words: right ventricle, right ventricular failure, acute heart failure, pulmonary hypertension, management, pulmonary vasodilators

INTRODUCTION

Right ventricular failure (RVF) remains a real clinical challenge for cardiologists, intensive care and emergency medicine physicians as well as anesthesiologists. Very often it is difficult to manage due to hemodynamic instability and serious comorbidities. For decades, left ventricle has been much more studied, and most therapeutic measures for treatment of acute heart failure were focused on left ventricular failure. We briefly describe the causes and pathophysiological mechanism of RVF and focus on the management of this syndrome.

ETIOLOGICAL AND PATHOPHYSIOLOGICAL MECHANISMS OF ACUTE RVF

RVF pathophysiological mechanisms include alterations in pre-load and diastolic filling, reduced RV contractility (impaired inotropic function) and increased after-load. Causes of acute RVF are summarized in Table.

DIAGNOSIS OF RVF

Diagnostic methods of RVF include pulmonary artery catheterization (PAC) and echocardiography (either transthoracic or transesophageal), and, sometimes, cardiac magnetic resonance imaging. PAC and echocardiography are also useful for therapeutic monitoring. At PAC PH is defined as resting mean pulmonary artery pressure >25 mm Hg, and

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Table. Causes of acute RVF (1)

| | |
|---------------------------------------|---|
| Left ventricular failure | Ischemic heart disease, valvular heart disease, etc. |
| RV ischemia | RV infarction Relative due to RV pressure and / or volume overload |
| After-load increase | Pulmonary artery hypertension (primary and secondary) Hypoxic pulmonary vasoconstriction Post-cardiothoracic surgery Pulmonary embolism Pulmonary microthrombi (sepsis and acute lung injury) Pulmonary stenosis and RV outflow tract obstruction Mechanical ventilation Acute chest syndrome in sickle cell disease |
| Pre-load decrease | Hypovolemia / capillary leak Superior vena cava syndrome Tricuspid stenosis Cardiac tamponade Mechanical ventilation |
| Myocardial disease | Cardiomyopathies Arrhythmogenic RV dysplasia Sepsis |
| Congenital and valvular heart disease | Ebstein's anomaly Tetrology of Fallot Transposition of great arteries Atrial septal defect Anomalous pulmonary venous return Tricuspid regurgitation Pulmonary regurgitation Mitral valve disease |
| Pericardial disease | Constrictive pericarditis |
| Arrhythmias | |

pulmonary vascular resistance $>240 \text{ dyn. s. cm}^{-5}$ (3 Wood units) (2). At echocardiography PH is suggested when RV systolic pressure exceeds 35 mm Hg (severe $>50 \text{ mm Hg}$) (3).

TREATMENT OF ACUTE RVF

General supportive measures include infection prevention, thromboembolism and peptic ulcer prophylaxis, nutritional support, glucose control, daily spontaneous breathing trials, optimal haemoglobin levels, sodium restriction in volume overload states, body weight and volume status monitoring (1).

Volume management. As RV function is highly volume-dependent, optimizing pre-load is very important. However, too much pre-load will decrease

LV output due to ventricular interdependence and cause hypotension. Therefore, fluid administration should be monitored invasively or noninvasively. RV volume overload is treated by using diuretics or hemofiltration.

Vasopressors. Norepinephrine may be an effective systemic pressor in patients with acute RV dysfunction and RV failure, as it improves RV function by improving systemic vascular resistance (SVR) and by increasing cardiac output, despite potential increases in pulmonary vascular resistance (PVR) at higher doses. In patients with vasodilatory shock and pulmonary vascular dysfunction, low-dose vasopressin may be useful in difficult cases that are resistant to usual treatments, including norepinephrine. Both recommendations are considered to be weak (4).

Inotropes. A weak recommendation can be made that low-dose dobutamine (up to 10 µg/kg/min) improves RV function and may be useful in patients with pulmonary vascular dysfunction, although it may reduce SVR. Dopamine may increase tachyarrhythmias and is not recommended in cardiogenic shock (strong recommendation). A strong recommendation is that phosphodiesterase (PDE) III inhibitors improve the RV function and reduce PVR in patients with acute pulmonary vascular dysfunction, although systemic hypotension is common, usually requiring co-administration of pressors. A weak recommendation can be made that levosimendan may be considered for short-term improvements in the RV function in patients with biventricular failure (4).

Pulmonary vasodilators. Those drugs include intravenous agents (prostacyclin, iloprost, sildenafil, milrinone, adenosine), inhaled agents (prostacyclin, iloprost, milrinone, nitric oxide) and oral agents (bosentan, sildenafil). A strong recommendation is made as follows: pulmonary vasodilators reduce PVR, improve CO and oxygenation, and may be useful when PH and RV dysfunction are present, notably after cardiac surgery. It is strongly recommended to use inhaled rather than systemic agents when systemic hypotension is likely, and concomitant vasopressors should be anticipated. A strong recommendation is made that a short use of nitric oxide improves oxygenation indices but not outcome in patients with the acute respiratory distress syndrome (ARDS). The use of pulmonary vasodilators to treat PH associated with RV dysfunction in ARDS has a weak level of recommendation (4).

Ventilatory strategies. Atelectasis and ventilation at high lung volumes should be avoided. Lower airway plateau pressure reduces the incidence of RV failure. Prone ventilation may also reduce plateau pressure and pCO₂ sufficiently to improve the RV function. High-frequency oscillation reduces pre-load and may reduce the RV function (5).

Mechanical support. Mechanical therapies include RV-assist devices, extracorporeal membrane oxygenation, intraaortic balloon counterpulsation and atrial septostomy. They may have a role as rescue therapies in reversible pulmonary vascular dysfunction or while awaiting definite treatment (weak recommendation) (4).

CONCLUSIONS

Acute RV failure is associated with worse outcomes in critical care. Judicious volume management is important avoiding volume overload. It is essential to maintain adequate aortic root pressure to prevent RV ischemia. Vasopressors are useful including low-dose norepinephrine as a first-line agent. Potentially useful inotropes include dobutamine and PDE III inhibitors. Pulmonary vasodilators are useful to reduce RV after-load, including PH and RV failure after cardiac surgery. Inhaled agents are preferred. Mechanical therapies may be useful in experienced centres.

Received 2 July 2012

Accepted 1 August 2012

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ŪMINIS DEŠINIOJO SKILVELIO NEPAKANKAMUMAS

Santrauka

Ūminis dešiniojo skilvelio (DS) nepakankamumas yra rimta klinikinė situacija ne tik kardiologijos intensyvosios terapijos skyriuose, bet ir apskritai gydant suaugusiųjų ūmines būkles. Jį gali sukelti ne tik kairiojo skilvelio nepakankamumas, bet ir daugelis kitų plaučių kraujagyslių disfunkcijos ir plautinės hipertenzijos priežasčių, pavyzdžiui, plautinė hipertenzija dėl hipoksijos, tromboembolijos, mechaninės ventilacijos ar portopulmoninė hipertenzija. Ūminį DS nepakankamumą dažnai sunku gydyti, ir jis siejamas su blogesne prognoze. Mirštamumas nuo kardiogeninio šoko, kurį sukėlė DS infarktas, viršija mirštamumą nuo kairiojo skilvelio ligų ir yra >50 %. Ūminis DS nepakankamumas taip pat susijęs su dažnesnėmis pooperacinėmis mirtimis. Bendrieji gydymo principai – prieškrūvio optimizavimas, DS kontraktiškumo gerinimas ir plaučių kraujagyslių rezistentiškumo, tai yra pokrūvio, mažinimas. Šiame apžvalginame straipsnyje daugiausia pateikiama informacijos apie DS nepakankamumo gydymą aptariant bendrąsias priemones, vazoaaktyvius vaistus, dirbtinės plaučių ventilacijos principus, taip pat chirurginius ir intervencinius metodus.

Raktažodžiai: dešinysis skilvelis, dešiniojo skilvelio nepakankamumas, ūminis širdies nepakankamumas, plautinė hipertenzija, gydymas, plaučių vazodilatatoriai