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LOW CARDIAC OUTPUT IN INTENSIVE CARE UNIT

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Epidemiology and mortality

Low cardiac output (LCO) is a common reason for ICU admission or an underlying disease that complicate the patient's outcome. The principal cause of low cardiac output remains ischemic heart disease (1,2). Other causes are systemic or pulmonary hypertension, arrhythmias, dilated cardiomyopathy, myocarditis, and finally valvular diseases. Low cardiac output is often associated or complicated with diabetes, renal failure, respiratory complications, and older age (2). LCO may be systolic or diastolic, and combined as well. The cardiac dysfunction may be right or left side heart failure.

Mortality is very high, and it is recently estimated to be approximately 13%. Several authors reported a mortality rate 24% (2,3,4).

Pathophysiology of low cardiac output

Cardiac output is determined by the combination of five factors: preload, afterload, contractility, rate, and rhythm. If any of these factors change, may have an important impact on cardiac output. As the result of low cardiac output, tissue hypoperfusion and congestion are the commonest pathophysiology changes faced. This situation activates the rennin-angiotensine-aldosterone axis, sympathetic response, and natriuretic peptide (5). The last one promotes vasodilatation, reduces afterload, and increases urinary output. Later the net result is vasoconstriction, sodium and water retention, and increased filling pressures. The increased wall stress can further deteriorate a preexisting ischemic zone and can also contribute in low cardiac output (6). By the other hand, the permanent high catecholamine levels induce cell death and decrease the contractility (7).

Afterload is often changed by hypertrophic cardiomyopathy, aortic stenosis, and intense peripheral vasoconstriction. The increased afterload doesn't permit the ventricular ejection, increasing so the end-systolic pressure and volume.

The preload contributes on venous return and cardiac filling. The preload can be diminished by hypovolemia (bleeding or other volume loses), intense vasoconstriction or vasodilatation (neurogenic, anaphylactic), and by so called central hypovolemia (reduced diastolic function by pneumothorax, cardiac tamponade, constrictive pericarditis). The decreased preload reduces end-diastolic-volume and the stroke volume. Stroke volume and heart rate determine cardiac output.

If the contractility decreases, the filling pressure increase and the wall stress as well. The increased heart rate impairs the diastolic myocardial blood furniture causing ischemia. The rhythm disturbances induce diastolic and systolic dysfunction.

Clinical features and diagnosis modalities of LCO

There are four clinical situations of LCO (8). These includes as follows:

1. Hypertension secondary decompensate heart function: this situation is determined by high blood pressure.
2. Cardiogenic shock: this situation is generally characterized by hypotension (systolic blood pressure under 90 mmHg), tachycardia, and low urine output.
3. Pulmonary edema: severe respiratory failure with rapid and rebel desaturation, and crackles.
4. Acute deterioration of chronic heart failure.

The diagnosis is the past medical history, thoracic X-ray, electrocardiogram, echocardiography, and B-type natriuretic peptide (BNP) measurement. A special attention can be paid to B-type natriuretic peptide (9,10). This hormone is released after the increased wall stretched and volume overload. The increased level of BNP correlates with LCO and prognosis as well.

Monitoring the patient suffering LCO

All the vital parameters must be regularly checked.

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The electrocardiogram, respiratory rate and saturation, heart rate and rhythm, blood pressure and urine output, are usually recorded. Invasive blood pressure monitoring is usually recommended. Central lines are useful for drug administration and for right atrium pressure monitoring. Transesophageal-ecocardiography is also useful for cardiac function monitoring, but this method can not give any indices about systemic resistances. Pulmonary artery catheters are also commonly used, but several authors recommended their limitation (11,12,13).

Treatment of LCO

Beside of oxygen administration 3 classes of drugs are generally used:

1. Vasodilators (sodium nitroprusside, nitroglycerin, nesiritide) are useful in left ventricular decompensation and hypertensive crisis.
2. Diuretics
3. Inotrops can be used in hypotension and LCO with or without pulmonary congestion (dopamine, dobutamine, amrinone, millrinone)

LCO is an emergency requiring urgent treatment. Immediate practical treatment is as follows.

4. Oxygen given by mask, non invasive mechanical ventilation, and finally endotracheal intubation.
5. The patient needs to be positioned upright, reducing the preload and venous stasis.
6. Morphine may be a suitable choice because it can reduce filling pressure. It must be titrated very carefully in order to avoid overdosing.
7. Loop diuretics 40-120 mg decrease ventricular filling, and can ameliorate the oxygenation.
8. Vasodilators (14) are suitable may improve left ventricular failure by reducing filling pressure and afterload. Several studies referred to nitroprusside, nitroglycerin, nicardipine etc.
9. Inotropes are generally required to improve contractility, reducing filling pressure, decreasing afterload (millrinone, dobutamine). Digoxin remained not a suitable choice in LCO after myocardial infarction.

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