Review paper

The management of patients with cardiogenic shock

Postępowanie u chorych ze wstrząsem kardiogennym

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Abstract

Cardiogenic shock is a life-threatening condition with a very poor prognosis. Many disorders may contribute to the development of shock. Of them, acute myocardial infarction is the main culprit. Despite tremendous progress in the field of interventional cardiology, in-hospital and long-term mortality still exceeds 50%. Data from large controlled trials on cardiogenic shock are scarce due to ethical reasons. Nevertheless, there is a chance for acceptable long-term survival in patients treated aggressively. The aim of this review is to summarise the key points of differential diagnosis and to emphasise that in most cases of shock due to acute myocardial infarction primary coronary intervention is the treatment of choice.

Streszczenie

Wstrząs kardiogenny jest stanem zagrożenia życia o bardzo złym rokowaniu. Wiele sytuacji klinicznych przyczynia się do rozwoju wstrząsu, ale ostry zawał serca stanowi najczęstszą przyczynę. Pomimo olbrzymiego postępu w kardiologii interwencyjnej śmiertelność szpitalna i długoterminowa nadal przekracza 50%. Z powodów etycznych nie dysponujemy danymi z kontrolowanych badań klinicznych. Niemniej agresywne leczenie stwarza szansę na uzyskanie akceptowalnego poziomu śmiertelności w tej grupie chorych. Celem pracy jest próba podsumowania najważniejszych elementów diagnostyki różnicowej oraz wykazanie, że pilna rewaskularyzacja za pomocą angioplastyki wieńcowej jest leczeniem z wyboru w większości przypadków.

Introduction

Cardiogenic shock (CS) is a life-threatening condition of heterogeneous aetiology with acute left or right heart failure as a key point leading to decreased and insufficient peripheral perfusion and multiple organ failure. Many disorders contribute to CS development; however, acute coronary syndromes (ACS), i.e. acute myocardial infarction (MI), are the leading cause [1, 2] (Table 1). Cardiogenic shock complicates up to 10% of ST-segment elevation myocardial infarction (STEMI) [3] and 2.5% of non-STEMI cases [4]. Typically, 50% of patients with STEMI develop CS during the first 6 h and 75% during the first 24 h from myocardial infarction symptoms onset [5]. Although in-hospital and short-term mortality is very high, ranging from 70–80% in patients treated conservatively [6] to 40–60% in patients receiving early reperfusion therapy [7], there is a chance for acceptable long-term survival in those treated aggressively [8].

The aim of this review is to outline pathophysiological changes during CS and to present step by step initial diagnosis, treatment strategies according to aetiology, and to discus novel research directions.

Aetiology of cardiogenic shock

Several acute conditions may contribute to the decrease in peripheral tissue perfusion. Most of them are primary cardiac. The most frequent are clinical manifestations of coronary artery disease. They include acute heart failure due to myocardial infarction and its complications, *myocarditis*, cardiomyopathies, blunt trauma, brady- and tachyarrhythmias, and severe mitral or aortic regurgitation. Some of them are related to prosthetic valve dysfunction, unintentional

Table 1. Causes of cardiogenic shock. Based on ref. [1, 11]

Myocardial injury: – acute myocardial infarction and its complications (the most common) – acute myocarditis – mechanical damage (commotio cordis) – cardiomyopathies, i. e. Takotsubo cardiomyopathy
Heart rhythm disturbances: – extreme bradycardia – tachycardia, i.e. ventricular tachycardia, atrial fibrillation with rapid ventricular response
Acute valve insufficiency: – aortic and mitral regurgitation, i.e. aortic root dissection, endocarditis, chordal rupture – prosthetic valve dysfunction
"Obturative" shock: – pericardial effusion/tamponade – thrombus, intracardiac tumour – pneumothorax – pulmonary embolism – pulmonary hypertension – intra-abdominal hypertension

or intentional drug overdose, or, in rare cases, transplant rejection. In many cases the heart is not initially involved; however, it cannot remain free of the haemodynamic complications in pericardial tamponade, *pneumothorax*, pulmonary embolism, etc. Regardless of the initial aetiology, the final path includes the heart's failure as a pump [1] (Table 1).

Pathophysiology

Recent investigations provide convincing data that in CS acute left- or right-ventricular failure is in parallel with neurohormone and cytokine system activation and peripheral vasculature compensatory response, which are all trapped in a vicious circle. Systemic inflammatory response due to myocardial and peripheral ischaemia mediated by catecholamines, vasopressin, angiotensin II, interleukin 6, tumour necrosis factor α, nitric oxide, and many others interfere with reflex vasoconstriction and left ventricular contractility, and therefore sustain and aggravate both myocardial injury and haemodynamic collapse. This complex pathophysiology has been presented in detail in an excellent paper by Reynolds and Hochmann in Circulation [9].

Clinical presentation and initial diagnosis

There are a variety of symptoms, signs, and measurements that define CS and characterise its severity. Although some haemodynamic parameters, i.e. cardiac index (CI) < 2.2 l/min/m^2 and pulmonary capillary wedge pressure (PCWP) > 18 mm Hg, are helpful to confirm the diagnosis and to monitor the response to the therapy applied, they are not mandatory in the initial phase of CS. A history of recent chest pain, persistent arterial hypotension < 90 mm Hg, tachycardia, cool extremities, diuresis less than 20 ml/h, and altered mental status are typical signs of peripheral hypoperfusion and should focus the attending physician on the active search for possible causes of CS [1, 10, 11]. Prompt 12-lead ECG and physical examination remain the basics to establish or to eliminate many conditions presented in Table 1. ECG provides initial information on STEMI/NSTEMI or arrhythmias and may suggest the suspicion of pulmonary embolism or cardiac tamponade. Some negative prognostic factors may also be noted, such as heart rate, QRS complex duration, and the voltage sum of ST-segment depression [12]. Bed-side echocardiography plays a key role in confirming diagnosis, identifying high-risk patients, and excluding mechanical complications of MI or acute valve disease and identifying signs of pulmonary embolism to plan the revascularisation and further treatment strategy (class I C according to European Society of Cardiology guidelines, [10]). The most powerful echocardiographic predictors of short- and long-term mortality are stroke volume index, stroke work index, and the severity of mitral regurgitation [13]. Some baseline laboratory parameters (blood smear, cardiac troponins, serum creatinine, electrolytes, bilirubin, C-reactive protein, natriuretic peptides, glucose, transaminases activity, and blood gases) should be repeated frequently to assess the dynamics of multiple organ failure. Chest computed tomography (CT) scan may help in differential diagnosis when ACS is not probable and provide life-saving data if aortic dissection or pulmonary embolism is present. Continuous monitoring of heart rate, blood pressure, respiratory rate, temperature, ECG, blood oxygen saturation, and urine output is mandatory. Invasive blood pressure measurement (class IIa C) is useful in haemodynamically unstable patients or in patients receiving inotropic agents. Central venous catheter (class IIa C) should be used when catecholamine infusion is present or to obtain central venous pressure (CVP) and venous oxygenation measurements. However, CVP may be inadequate during positive end-expiratory pressure ventilation or in patients with significant tricuspid regurgitation. Some haemodynamic parameters (CVP, right atrial pressure, PAWP) provided by Swan-Ganz catheter (class IIb B) are helpful in monitoring response to treatment and allow the calculation of mean arterial pressure, stroke volume, systemic and pulmonary vascular resistance, and many others. An ongoing discussion on the usefulness of the Swan-Ganz catheter has been summarised in a meta-analysis by Shah *et al.* [14] and in a review by Payen and Gayat [15] with the conclusion that the Swan-Ganz catheter should not be used in a routine manner and that most of the parameters can be obtained by other non-invasive measurements, including echocardiography and central venous catheter.

Treatment of patients with cardiogenic shock

An aetiology-guided treatment should start during first medical contact. A temporary pacing should be initiated in patients with bradycardia, and an electrical cardioversion should be performed in most cases of ventricular tachycardia or atrial fibrillation with rapid ventricular response. Patients with acute valve insufficiency or acute aortic root dissection should undergo surgical repair regardless of the cause provided in Table 1. Other non left-ventricular causes are rare; however, attempts should be made to ensure proper right-sided filling pressures by fluid resuscitation and removing the primary cause (i.e. tamponade, pulmonary embolism, etc.). Maintaining appropriate oxygenation using both non-invasive and invasive ventilation techniques is mandatory to prevent respiratory failure and further shock progression.

Cardiogenic shock due to acute coronary syndrome

The current treatment of patients with CS related to acute coronary syndromes has been summarised in both American and European guidelines for the management of patients with STEMI [10, 16]. In general, there is agreement that prompt haemodynamic stability achieved by immediate revascularisation and supporting pharmacotherapy is the primary goal. Invasive and non-invasive methods must start simultaneously, because the classic DeLuca's finding that every minute of delay counts remains valid [17]. Before aggressive antithrombotic and antiplatelet therapy and primary percutaneous coronary intervention (PCI) is initiated mechanical complications of MI must be excluded.

Mechanical complications of myocardial infarction

A free wall rupture (FWR) complicates 1–6% of STEMI. Older people without reperfusion therapy or treated with lytics, systemic steroids, or non-steroid anti-inflammatory drugs are prone to the development of this complication. In acute cases chest pain, blood pressure drop, and pulseless electrical activity are usually followed by death. In subacute FWR a continuous blood leakage to the pericardium may lead to tamponade; however, a surgical intervention is possible when performed on time. In chronic form a false aneurysm may be formed. An early invasive strategy, optimal blood pressure control, and quick relief from the anginal pain are the most efficient methods of FWR prevention. A ventricular septum rupture (VSR) is rare in a primary PCI era (less than 1% of all STEMI cases, mainly in multi-vessel disease cases). A progression of biventricular heart failure, or right bundle branch or complete heart block and a loud systolic murmur make a typical clinical picture. A papillary muscle rupture often accompanies the right or circumflex coronary artery closure and results in an acute mitral regurgitation (MR) manifested as pulmonary oedema or CS. The treatment of choice in all cases mentioned is an immediate surgical intervention because the mortality in patients treated conservatively reaches 90% [18–20].

Myocardial infarction of the right ventricle

A right ventricular MI may coincide with up to 50% of inferior MIs and is characterised by an increase in right atrial and ventricular pressures, and decreased PCWP and stroke volume. When isolated it is usually well tolerated because the right ventricle receives support from the left one via interventricular septum movements and thus by increasing right ventricular pressures. In large MIs often accompanied by atrial fibrillation or atrioventricular blocks this mechanism fails and severe CS develops with high mortality [21, 22].

Reperfusion and revascularisation

Emergency revascularisation, either by PCI or coronary artery bypass graft (CABG), irrespective of time delay or prior lysis, is recommended in all cases in CS due to acute myocardial infarction, in both European and American guidelines (class I B) [10, 16]. The beneficial impact of an early revascularisation strategy was observed regardless of baseline left ventricular function [23]. In the Should We Emergently Revascularise Occluded Coronaries for Cardiogenic Shock Trial (SHOCK) there was no significant difference in 30-day mortality; however, early revascularisation strategy was superior to conservative management (6-month mortality: 50.3% vs. 63.1%, $p = 0.027$; 12-month mortality 53.3% vs. 66.4%, $p < 0.03$) and this trend was sustained in 3-year and 6-year observations [7, 8, 24]. The 30-day mortality was lower in a subgroup of patients younger than 75 (41.4% vs. 56.8%, $p < 0.02$). Data by Dauerman *et al.* from the Global Registry of Acute Coronary Events (GRACE) show lower in-hospital mortality in revascularised patients (45% vs. 69%, $p < 0.001$) [25] regardless of age as well as in Dzavik's *et al.* sub-analysis (48% vs. 81%, $p < 0.0003$) [5]. Similar observations on in-hospital mortality in older patients with CS come from the Polish Registry of Acute Coronary Syndromes (PL-ACS): 54.6% vs. 69.9%, $p < 0.0001$ [26]. These outcomes are even better when platelet glycoprotein IIb/IIIa inhibitors (abciximab) are added to the adjunctive pharmacotherapy [27]. In cases of primary PCI failure or in patients who are not eligible for PCI due to multivessel disease or/and the left main involvement CABG should not be postponed (class I C) [10, 28].

Adjunctive pharmacotherapy

Antithrombotic therapy with aspirin and heparin should be administered as a standard of care in ACS

Table 2. Adjunctive intravenous pharmacotherapy in patients with cardiogenic shock according to their haemodynamic status. Based on ref. [10, 29]

ESC class	Haemodynamic parameters and agents applied	Comment
	CI < 2.2 l/min/m^2; PCWP < 14 mm Hg Fluid infusion	
I C	CI < 2.2 l/min/m^2; PCWP > 18 mm Hg; SBP > 85 mm Hg Nitroglycerine, 10–200 µg/min	Possible when no severe valve stenosis is present
	Sodium nitroprusside, 0.3–8 µg/kg/min	If longer than 10–30 h serum thiocyanide levels monitoring needed
	Nesiritide, 2 µg/kg bolus followed by 0.015–0.03 µg/kg/min	
	CI < 2.2 l/min/m^2; PCWP > 18 mm Hg; SBP < 85 mm Hg; CVP > 10 mm Hg	
IIa C	Dobutamine, 2–20 µg/kg/min	
IIa C	Dopamine, 1–20 µg/kg/min	
IIb B	Norepinephrine, 0.2–1 µg/kg/min	
IIb C	Epinephrine, 0.05–05 µg/kg/min	
IIb B	Milrinone, 25–75 µg/kg bolus followed by 0.375–0.75 µg/kg/min	
IIb B	Enoximone, 0.25–0.75 µg/kg bolus followed by 1.25–7.5 µg/kg/min	
IIb C	Levosimendan, 12 µg/kg bolus followed by 0.05–0.2 µg/kg/min	No bolus when SBP < 100 mm Hg
	Fluid retention	
I C	Furosemide, max. 240 mg/day	Continuous infusion better
No recommendation	Tolvaptan	Decreases dyspnoea, mortality unchanged, serious liver injury possible

patients. Both clopidogrel and novel ADP-receptor inhibitors may be deferred until coronary angiography is performed, to decrease bleeding complications during emergent coronary artery by-pass grafting. Negative inotropes and blood pressure-lowering agents should be avoided. An appropriate arterial oxygenation (including non-invasive and invasive positive pressure ventilation), adequate glycaemic control and near-normal pH are mandatory to support the results of invasive treatment. A variety of pharmacological agents improving cardiac output and increasing blood pressure are available. In cases with CS and PCWP < 14 mm Hg and CI < 2.2 l/min/m^2 a continuous fluid infusion is recommended to fulfil the vascular bed. When PCWP exceeds 18 mm Hg vasodilators may be helpful in cases without hypotension and severe valve stenosis. In patients with CI < 2.2 l/min/m^2, PCWP > 18–20 mm Hg, and systolic blood pressure (SBP) < 85 mm Hg a continuous infusion of inotropic agents through a central venous catheter should be initiated. An adequate CVP (ca 10–14 mm Hg) is mandatory to avoid peripheral hypoperfusion. Haemodynamic benefit may be diminished by potential side effects (ventricular arrhythmia, tachycardia). The most popular agents, their dosage, and special issues are summarised in Table 2 [10, 29].

Mechanical support

Mechanical support to improve systemic blood flow and to decrease peripheral hypoperfusion may be a life-saving option in patients in whom CS persists despite early revascularisation and optimal medical therapy. A history of left ventricular support begins with extracorporeal membrane oxygenation (ECMO), and evolves via intra-aortic balloon pump (IABP) and Hemopump to TandemHeart, Impella, and Ventricular Assist Devices (VAD). Currently, various modifications are available on the market.

Intra-aortic balloon pump

Forty years of experience after IABP introduction has made it easy to implant a device with relatively low cost and complication rate. Peak diastolic pressure is increased and the end-systolic pressure is decreased during continuous inflation-deflation cycles, which results in a reduction of afterload and improved coronary flow. Previously reported as a first line strategy (class I C) [30] with scientific support from the SHOCK Trial Registry [31] and the National Registry of Myocardial Infarction-2 (NRMI-2) [32], IABP lost its position in guidelines due to several meta-analyses [33, 34] and IABP-SHOCK II Trial [35] results with no further support of any IABP benefit. Unfortunately, while IABP is not efficient to ensure long-term survival, there is no well defined reason why the improvement in the haemodynamic status after IABP insertion is not a suitable predictor of better survival. Several limitations of the SHOCK Trial Registry may be a partial explanation: IABP inserted after PCI, IABP group represents a high-risk population, bleeding complication, stroke or in-

flammatory response to IABP components. Of note, classic indications for IABP, i.e. mechanical complications of MI, are still valid.

Novel devices and areas of future research

In theory, veno-arterial extracorporeal membrane oxygenation may maintain cardiac output up to 6 l/min and exchange carbon dioxide and oxygen. Whether it has a beneficial effect on long-term survival in STEMI complicated by CS remains unclear due to the scarcity of data. In a single-centre study in a population with deep CS of a heterogenous aetiology, the majority of 68 patients who were successfully weaned from ECMO were discharged alive. In-hospital mortality in the STEMI subgroup was 40.5% [36], which was also confirmed in a small STEMI-only oriented study on 27 subjects (37% 30-day mortality in those successfully weaned from ECMO) [37] and in another one on 33 patients (1-year mortality 36.4% in patients treated with ECMO and IABP vs. 76% in those without ECMO; $p < 0.001$) [38]. Although these data are encouraging, better survival was assigned for successful weaning from ECMO. Whether it was an ECMO-related benefit remains undetermined. However, ECMO may be followed by different modifications of ventricular assist device. Data from large well powered randomised controlled trials on novel devices in CS management are lacking. The reports available come from observational studies and retrospective analyses or small trials [39–43]. The Impella (Abiomed, Germany) is a catheter-based, percutaneously implanted axial pump facilitating the blood flow from the left ventricle to the aorta. The TandemHeart (Cardiac Assist, USA) is a percutaneously or surgically implanted by-pass from the left atrium to the femoral artery via centrifugal continuous flow pump. Both systems may be implanted by interventional cardiologists prior to or after PCI; however, TandemHeart requires the operator to be skilled in transseptal puncture because the by-pass is implanted via the femoral vein through the interatrial septum at an activated clotting time of over 400 s. Despite encouraging results, bleeding complications, in-hospital, and long-term mortality still remain unacceptably high in this population [44]. This is best summarised in a meta-analysis by Cheng *et al.*, with a conclusion that despite better haemodynamic profile mortality does not decrease significantly [45]. However, these devices may be a bridge to heart transplantation or – if the donor is lacking – to total artificial heart implantation and staged transplantation with an excellent long-term survival [46–48].

Another important area of research involves mild therapeutic hypothermia via percutaneous catheter located in the inferior vena cava. It has been shown to improve the neurological outcome in out-of-hospital cardiac arrest survivors, and neuroprotection is the primary goal of its emergent application [49]. Its effect on myocardial performance during the acute phase of MI remains unknown. In animal models the results are conflicting [50, 51]. In trials that proved hypothermia benefit patients with CS were excluded [10]. Moreover, hypothermia might have a deleterious influence on haemodynamic status due to hypotension, bradycardia, vasoconstriction, or shivering and its management (deep analgo-sedation or neuromuscular blocking agents). This may raise concerns about the efficacy and safety in patients with initial haemodynamic collapse. Inter-hospital differences in cooling protocols, no standard in sequence (prior to or after primary PCI), and lack of data from well-powered trials may cause difficulties in drawing conclusions. Therefore, extrapolating data must be done with caution. Nevertheless, Norwegian and Czech reports encourage further investigate this issue [52, 53].

Conclusions

Can the 50% mortality barrier be broken? An effort to achieve early reperfusion with final TIMI 3 flow, experience in PCI techniques, and new generations of stents and multilevel platelet activation inhibition has resulted in a mortality decrease in recent decades. Unfortunately, some patients with patent infarct-related artery do not improve their haemodynamic status and have a very poor prognosis. Many conclusions on CS were drawn from SHOCK analyses. Randomised controlled trials involving patients with CS are difficult to conduct due to ethical reasons, so the national registries play an important role [54]. It is not clear whether research in mechanical support combined with therapeutic hypothermia in cardiac arrest comatose survivors or experimental therapies in the inhibition of the systemic inflammatory response (i.e. inducible nitric oxide synthase), or even bone marrow stromal cells auto-transplantation, will further improve survival. So far, we can "only" exclude mechanical complications, optimise oxygenation, and achieve TIMI 3 flow, preferably by immediate culprit lesion stenting and staged procedure (PCI or CABG) for multivessel disease (unless there is TIMI flow less than 3 in non-culprit vessels with significant narrowing). A need for a prompt mechanical support is intuitively defined; however, no strong data supporting novel techniques is present.

Conflict of interest

The authors declare no conflict of interest.

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