BRIEF COMMUNICATION

Post Infarction Cardiogenic Shock: Is It Clinically Important to Differentiate Hemodynamic Patterns?

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ABSTRACT

Despite recent advances in the treatment of cardiogenic shock, it still remains the main cause of death in patients hospitalized for acute myocardial infarction. Although cardiogenic shock is classically described as a hemodynamic condition characterized by low cardiac output, increased filling pressure and elevated systemic vascular resistance, some patients present different patterns, as low systemic resistance, fever and leucocytosis, indicative of an important systemic inflammatory response. The clinical importance of having one hemodynamic pattern or the other is currently unknown, though the existence of two different hemodynamic patterns should lead to reconsider the support medical treatment in this severely ill group of patients. The aim of this work was to analyze the incidence of each type of cardiogenic shock (classic and distributive), its clinical characteristics and its in-hospital outcome.

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Key words > Myocardial Infarction - Shock – Cardiogenic Shock

Abbreviations >

AMI	Acute myocardial infarction	**IABP**	Intra-aortic balloon pump
CI	Cardiac index	**SVR**	Systemic vascular resistance
CO	Cardiac output	**TNF-α**	Tumor necrosis factor alpha
CS	Cardiogenic shock		

INTRODUCTION

Despite recent advances in the treatment of cardiogenic shock (CS), it still remains the leading cause of death in patients hospitalized for acute myocardial infarction (AMI). (1)

Typically, its pathophysiology implies a vicious cycle triggered by loss of critical contractile mass leading to decreased cardiac output (CO) producing tachycardia, peripheral hypoperfusion (reflex vasoconstriction) and increased ventricular filling pressures (classic CS). However, some patients present a different hemodynamic pattern, resulting from the activation of a systemic inflammatory response generated by the infarction, characterized by fever, leukocytosis and not high (even low) systemic vascular resistance (SVR) despite the use of vasopressor drugs (distributive CS), as described by Hochman et al. (2) So far there are no scientific studies analyzing the clinical importance of having one hemodynamic pattern or the other. The purpose of this study was to analyze the frequency of each type of CS (classic and distributive), their clinical characteristics and in-hospital outcome.

METHODS

The study consisted in the consecutive, retrospective analysis of 350 patients with ST-segment elevation AMI of 12-hours evolution treated with primary percutaneous coronary intervention during the period 2008-2011. Patients with AMI complicated with CS who had a Swan-Ganz catheter inserted within 24 hours of admission were included in the study. The first hemodynamic measurement was considered for the definition of CS (classic vs. distributive). Patients with echocardiographic diagnosis of severe tricuspid regurgitation and patients with mechanical or infectious complications were excluded from the study.

Definitions

Shock: Systolic pressure = 90 mmHg associated with signs of peripheral hypoperfusion and/or requiring inotropic or vasoconstrictor therapy, in the absence of arrhythmias or hypovolemia.

Hemodynamic measurement: Classic CS: pulmonary

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capillary wedge pressure = 18 mm Hg, CI = 2.2 L/min/m2, SVR > 1200 dyn × s × cm-5, distributive CS: SVR < 1200 dyn × s × cm-5.

Statistical analysis

Data were retrospectively collected from selected clinical histories, recorded in an Excel form and analyzed with the EPI 2000 software. Qualitative variables were expressed as percentages and compared using the chi-square test (with Yates correction when necessary) or Fischer´s exact test, while quantitative variables were expressed as median with interquartile range (25-75%) and analyzed with Student´s t test or Wilcoxon´s test as applicable. A p value < 0.05 was considered significant.

RESULTS

Among the 22 patients included in the study, 11 had hemodynamic pattern of classic CS and 11 of distributive CS. Population and clinical characteristics described in Table 1 show that there were no significant differences between both types of CS. The comparison of hemodynamic parameters between both types of CS revealed that distributive CS had greater CO and CI and lower SVR without differences in the remaining measured parameters (Table 2). There were no significant differences in the use of inotropic drugs, IABP, mechanical respiratory assistance and mortality (Table 3).

DISCUSSION

Cardiogenic shock is still the leading cause of death in patients hospitalized with AMI. Its incidence ranges between 6% and 8% of AMI cases and is associated with 40-50% mortality despite revascularization and IABP. (3, 4)

Swan-Ganz catheterization allows confirming its diagnosis, assessing ventricular filling pressures and guiding medical treatment to achieve standardization of the measured values. While routine use of Swan-Ganz catheterization in patients with decompensated chronic heart failure has not shown benefit in reducing mortality and its overall use has declined globally, (3) its indications in the context of CS as AMI complication is well accepted and recommended in management guidelines (Class IIa, level of evidence B in the American guideline (5) and Class IIb, level of evidence C in the European guideline). (6)

It is well known that within the pathophysiology

Table 1. Basal characteristics

	Total (n = 22)	Classic shock (n = 11)	Distributive shock (n = 11)	p
Age, years	63 (55-77)	64 (63-77)	57 (52-78)	ns
Male	90%	80%	100%	ns
Diabetes	14%	18%	9%	ns
Fever	41%	27%	55%	ns
Leukocytosis	90%	89%	91%	ns
Leukocyte count (× mm3)	15500 (13100-19300)	13700 (11400-21100)	16200 (16000-20050)	ns
Maximum CPK (IU/L)	3308 (2102-7059)	7412 (4400-11900)	2820 (1460-5420)	ns
Anterior AMI	42%	55%	27%	ns
Previous AMI	18%	27%	9%	ns

CPK: Creatine phosphokinase. AMI: Acute myocardial infarction. ns: not significant.

Table 2. Hemodynamic parameters

	Classic (n = 11)	Distributive (n = 11)	p
Mean arterial pressure (mm Hg)	67 (63-90)	81 (71-100)	ns
Heart rate (beats/min)	89 (71-114)	107 (90-120)	ns
Mean PAP (mm Hg)	32 (26-34)	28 (27-33)	ns
PCWP (mm Hg)	20 (17-22)	17 (13-20)	ns
RA pressure (mm Hg)	10 (8-11)	11 (10-12)	ns
Cardiac output (L/min/m2)	3.46 (2.8-4.5)	5.8 (3.8-6.1)	< 0.01
CI (L/min/m2)	1.9 (1.5-2.2)	2.9 (2.1-3.3)	< 0.001
SVR (dyn × s × cm-5)	1270 (1200-2074)	947 (850-1080)	< 0.1
PVR (dyn × s × cm-5)	159 (125-227)	116 (104-165)	ns
LVSWI (g × m/m2)	14.9 (9.7-18.9)	22.9 (15.8-28)	ns
RVSWI (g × m/m2)	6.22 (1.85-7.78)	4.02 (3.3-4.6)	ns

PAP: Pulmonary arterial pressure. PCWP: Pulmonary capillary wedge pressure. RA: Right atrial. SVR: Systemic vascular resistance. PVR: Pulmonary vascular resistance. LVSWI: Left ventricular systolic work index. RVSWI: Right ventricular systolic work index. ns: Not significant.

Table 3. In-hospital outcome

	Total (n = 22)	Classic Shock (n = 11)	Distributive Shock (n = 11)	p
Inotropic drugs	82%	82%	82%	ns
Counterpulsation	41%	46%	36%	ns
MRA	69%	69%	69%	ns
Time of inotropic administration	5 (3-11)	4 (3-15)	7 (4-8)	ns
Time of Swan-Ganz	4 (3-9)	5,5 (3-10)	3 (2-7)	ns
Hospital stay	14 (8-22)	13 (9-29)	15 (8-22)	ns
In-hospital mortality	31,5%	36%	27%	ns

MRA: Mechanical respiratory assistance. ns: Not significant. Time in days.

of CS, CO reduction secondary to acute contractility loss of a significant part of cardiac muscle leads to tachycardia, peripheral vasoconstriction and increased filling pressures, which perpetuate a vicious cycle that needs compensation to avoid death. In this sense, IABP used for cardiac support until clinical improvement is a useful mechanical device based upon preload and afterload reduction increasing cardiac output and myocardial perfusion pressure. It is a guideline Class I recommendation, (5, 6) with level of evidence B. However, the results of the IABP -SHOCK II study published this year, (7) which randomized 600 patients with postinfarction CS after either use or not of IABP, remarkably showed a similar mortality at 30 days in both groups (39.7 vs. 41.3%, p = ns). One possible explanation for this finding might be that not all patients have the same postinfarction pathophysiology of CS and, therefore, may require different treatments. Hochman et al reported in the SHOCK trial that classic hemodynamic parameters were not seen in some patients, being the first to propose the existence of a new pathophysiological shock paradigm, (2) focusing on the systemic inflammatory response induced by infarction. This scenario, characterized by low systemic resistance, fever and leukocytosis, involves various inflammatory mediators such as interleukin 6 (IL -6) , tumor necrosis factor alpha (TNF-α), the complement, procalcitonin , neopterin, C-reactive protein and elevated values of nitric oxide and peroxynitrite secondary to increased activity of inducible nitric oxide synthase. IL -6 and TNF-α, have cardiac depressant effects and can even be elevated at admission in patients with Killip and Kimball A which then progress to CS. (8) TNF-α also induces endothelial dysfunction , which may further decrease coronary flow . (9) The elevation of nitric oxide leads to vasodilation, myocardial depression and interferes with catecholamine action. (10)

Modulation of the inflammatory response with drugs, as nitric oxide synthase inhibitors, has also been investigated. However, although preliminary studies showed that hemodynamic status and evolution could be improved in a small group of patients (11) larger studies (12) failed to show mortality reduction.

We observed in our study that according to the hemodynamic parameters measured, both the distributive and classic shock pattern are equally frequent. Both types of shock had similar in-hospital mortality and no clinical variables from which to infer each patient´s pattern, since even leukocytosis and fever were very common in both types of shock. In this sense, a limitation of our study is that due to the small sample size a beta type error cannot be ruled out , i.e. the lack of power to detect differences between the two groups.

CONCLUSION

The pathophysiological understanding of different forms of CS presentation following AMI would allow tailoring adjuvant therapeutic measures to reduce this entity´s high mortality.

RESUMEN

Shock cardiogénico posinfarto: ¿tiene importancia clínica discriminar patrones hemodinámicos?

El shock cardiogénico continúa siendo la principal causa de mortalidad en los pacientes hospitalizados por un infarto agudo de miocardio a pesar de los avances logrados en su tratamiento en los últimos años. Si bien clásicamente el shock cardiogénico se describe como un cuadro hemodinámico caracterizado por bajo volumen minuto cardíaco, aumento de presiones de llenado y elevación de las resistencias vasculares sistémicas, en algunos pacientes se observa una medición diferente, expresión de una respuesta inflamatoria sistémica importante, caracterizada por resistencias sistémicas bajas, fiebre y leucocitosis. Hasta el momento se desconoce la importancia clínica de tener un patrón hemodinámico u otro a pesar de que, quizá, la existencia de dos patrones hemodinámicos diferentes deba llevar a replantear el tratamiento médico de sostén en este grupo grave de pacientes. El presente trabajo se llevó a cabo con el objetivo de analizar la frecuencia de cada tipo de shock cardiogénico (clásico y distributivo), sus características clínicas y su evolución intrahospitalaria.

Palabras clave > Infarto del miocardio - Choque - Choque cardiogénico

Conflicts of interest
None declared.

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