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Short-Acting Endovenous Beta-Blockers and Echocardiography-Guided Heart Rate Optimization in A Type 4a Myocardial Infarction Complicated by Cardiogenic Shock

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Abbreviations:

HR: Heart Rate; SAP: Systolic Arterial Pressure; DAP: Diastolic Arterial Pressure; MAP: Mean Arterial Pressure; EDV: End-Diastolic Volume; ESV: End-Systolic Volume; LVOTd: Left Ventricular Outflow Tract Diameter; LVOT VTI: Left Ventricular Outflow Tract Velocity-Time Integral; EF: Ejection Fraction; SV: Stroke Volume; SVI: Stroke Volume Index; BSA: Body Surface Area; CO: Cardiac Output; CI: Cardiac Index; CPO: Cardiac Power Output; CPI: Cardiac Power Index; PET: Pre-Ejection Time; ET: Ejection Time; IVRT: IsoVolumic Relaxation Time; ePAWP: Estimated Pulmonary Artery Wedge Pressure; TAPSE: Tricuspid Annular Plane Systolic Excursion; FAC: Fractional Area Change Right(A-V) grad – Right Atrio-Ventricular Pressure Gradient; PASP: Pulmonary Artery Systolic Pressure; sRVCPI: Simplified Right Ventricular Contraction-Pressure Index; pAcT: Pulmonary Acceleration Time

Formulas:

SV = ((LVOTd)/2*(LVOTd)/2*(VTI)*3.14)/100 SVi = SV / BSA
CO = (SV*HR)/1000 CI = (SV*HR)/1000 CPO = (CO*MAP)/451 CPI = (CI*MAP)/451
ePAWP1 = 1.24*(E/e')+1.9 mmHg
sRVCPI2 = TAPSE*Right(A-V) grad

1. Abstract

1.1. Background

Landiolol, a short-acting, highly-β1-selective beta-blocker without negative inotropic effect, could offer the opportunity to modulate heart rate and/or rhythm in different critical settings. Echocardiographic estimate of hemodynamic parameters is a useful way to monitor critically ill patients and evaluate the effects of the ongoing therapy.

1.2. Case Summary

a 55-year-old man with end-stage renal failure underwent an elective percutaneous coronary intervention that was complicated by type 4a myocardial infarction and resulted in cardiogenic shock. The patient developed hypotension and sinus tachycardia, needing vasoactive support with norepinephrine. In order to optimize cardiac output, endogenous beta-blocker landfill was administered, modulating infusion rate with close echocardiographic monitoring in order to find the optimal chronotropism.

1.3. Discussion

this case highlights how integrating dynamic echocardiographic assessment with a short-acting beta-blocker can guide real time hemodynamic optimization. Llandilo enabled heart rate modulation, improving estimated cardiac output and facilitating weaning from norepinephrine. This approach underscores the potential role of targeted beta-blockade in fine-tuning cardiac performance in selected critically ill patients.

2. Introduction

Echocardiography, a widely available method to guide diagnosis and monitor critically ill patients, is progressively gaining space and relevance in intensive care unit (ICU) due to the possibility of performing non-invasive estimates of plenty of hemodynam-

ic parameters [1]. In this regard, echocardiographic monitoring has already been proven as a valuable tool in different clinical settings [2]. Landiolol, a highly-β1-selective beta-blocker, lends itself to a profitable use in hemodynamically unstable patients thanks to its extremely short half-life [3]. This molecule has already been studied in different clinical settings, demonstrating a safe hemodynamic profile and a negative chronotropic power. In the present case, we integrated a real-time echo dynamic assessment with the clinical and hemodynamic effects of landfill in a critically ill patient.

3. Case Presentation

A 55-year-old man with end-stage renal failure (in chronic replacement therapy) due to IgA nephropathy, was admitted to the Cardiology ward in order to undergo an invasive coronary angiography after a myocardial single-photon emission computed tomography (SPECT) suggestive for reduction in coronary reserve, performed in the context of kidney transplant evaluation. He had no previous cardiologic history, with a transthoracic echocardiography showing preserved left ventricular ejection fraction (LVEF), normal indexes of right ventricular systolic function and no significant valve disease. The coronary angiogram showed a severe calcific obstructive disease involving mid left anterior descending (LAD) artery; therefore, a percutaneous coronary intervention (PCI) with rotational atherectomy and stent implantation was performed. The procedure was complicated by a no-reflow phenomenon causing a final LAD TIMI-flow 0, despite the use of intracoronary adenosine, adrenaline and multiple balloon dilations.

The patient, normotensive (blood pressure - BP - 120/80 mmHg, heart rate - HR - 100 bpm) with no clinical signs of hypoperfusion, was then admitted to the ICU. The echocardiography showed akinesia of LAD territory with severe reduction of LVEF (25%), no signs of elevated left ventricular filling pressure, normal indexes of right ventricular systolic function and normal estimated pulmonary pressures. Soon after, marked hypotension occurred together with hyperthermia; an overt mechanical complication was excluded and rapid volemic expansion together with vasoactive support with norepinephrine 0.10 mcg/Kg/min were initiated. As the patient remained hypotensive with a proportional pulse pressure (PPP) of 18% (BP 85/70 mmHg, HR 95 bpm), a new echographic evaluation was performed, showing an estimated cardiac output (eCO) of 3.6 L/min (left ventricular outflow tract velocity-time integral - LVOT VTI - of 10 cm with an anterograde stroke volume - aSV - of 38 mL), no signs of elevated left ventricular filling pressure (E/A ratio 0.8, isovolumic relaxation time - IVRT - 125 ms) and a clear fluid responsiveness with a 20% increase in LVOT VTI after passive leg raise (PLR), although with a possible fluid intolerance (IVRT 66 ms). Considering these findings and the concomitant mild anemia, a blood transfusion was administered and infusion of landiolol 2 mcg/Kg/min was started. After few hours, the re-evaluation showed stable levels of haemoglobin, no clinical signs of hypoperfusion and a substantially stable BP (80/60 mmHg, PPP 25%) with a significant decrease in HR (85 bpm): a new echo graphic assessment was then performed, showing a slight increase in eCO (3.9 L/min, LVOT VTI 12 cm) with signs of elevation in left ventricular filling pressures (IVRT 60 ms). According to these findings, a decrease in norepinephrine infusion rate was attempted and well tolerated and it was possible to stop the vasoactive support after few more hours. The following day, another echo graphic examination was performed, which showed a reduction in eCO, as the patient's HR lowered to 78 bpm: landiolol was accordingly reduced aiming to a HR of 85 bpm and then stopped. During the following days, the patient's hemodynamic improved significantly, so that it was possible to plan a functional rehabilitation program; moreover, diagnosis of IgA-induced vasculitis was made. Unfortunately, the patient died about 10 days later, because of left ventricular free wall rupture occurred during straining.

4. Discussion

The present case is a notable demonstration of how echocardiographic evaluation can guide all- round therapeutic managements, even more when using a short-acting and rapid-onset drug such as landiolol. The first insight is related to the possibility of non-invasively assessing CO variations by echo graphically measuring LVOT VTI to estimate anterograde left ventricular stroke volume. This parameter is of paramount importance as it allows the clinician to determine the presence of a low- output state, which is an indispensable requirement for the diagnosis of cardiogenic shock in a non- hypovolemic patient [4,5]. Next to this diagnostic feature, repeated measurements can be useful in discerning whether or not a therapeutic intervention has been hemodynamically helpful, as clearly reported in other settings [2,6]. In the present case, it was possible to ascertain that the initial landiolol- induced reduction in heart rate brought to a slight increase in eCO (3.5 L/min vs 3.9 L/min) with, realistically, a lower myocardial oxygen demand. Moreover, the subsequent finding of a decrease in eCO with a further drop in HR, led to the reduction in dosage of landiolol, according to the physio pathologically robust hypothesis that a correct compensatory and adaptive HR was about 85 bpm (see Supplementary Data Table 1-3). This last consideration opens up to possible future scenarios regarding HR modulation using endogenous drugs in order to maximize left ventricular cardiac output and to optimize energy expenditure. The second insight is related to the use of the short-acting endogenous beta-blocker landiolol. This molecule, with its 4-minute half-life and high β1-selectivity (255:1 = β1: β2), has already been studied in specific clinical contexts, such as septic shock and supraventricular tachyarrhythmias, with evidence of hemodynamic safety, negative chronotropic power and antiarrhythmic efficacy [7,8]. In the present case, landiolol was used in a patient with no supraventricular or ventricular arrhythmias nor overt infection, but with a clear phlogistic state due to the recent myocardial infarction, the chronic need for renal replacement therapy and the underlying inflammatory phenotype related to the rheumatological disorder.

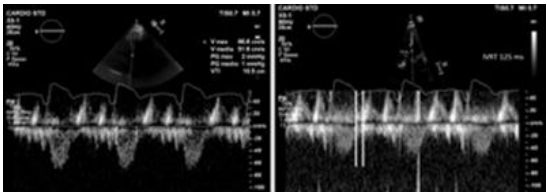

Figure 1: LVOT VTI (left) and IVRT (right) at CICU admission, before landiolol administration.

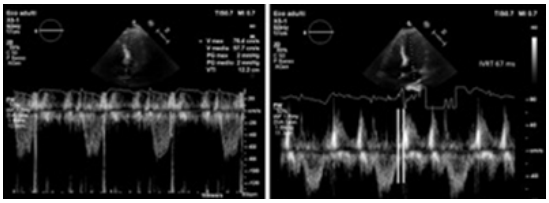

Figure 2: LVOT VTI (left) and IVRT (right) 6 hours after landiolol administration (HR 85 bpm).

Table 1: First echodynamic assessment (before landiolol administration).

Parameter	Value
HR (bpm)	94
SAP (mmHg)	85
DAP (mmHg)	69
EDV (mL)	133
X`ESV (mL)	100
LVOTd (mm)	22
LVOT VTI (cm)	10
EF (%)	25
SV (mL)	38
CO (L/min)	3.6
CI (L/min/mq)	2.05
CPO (W)	0.59
CPI (W/mq)	0.34
PET (ms)	87
ET (ms)	210
E-wave (cm/s)	47
A-wave (cm/s)	55
E/A ratio	0.85
IVRT (ms)	125
ePAWP (mmHg)	10
TAPSE (mm)	18
FAC (%)	38
TAPSE/PASP	0.72
sRVCPI	342
pAcT (ms)	129

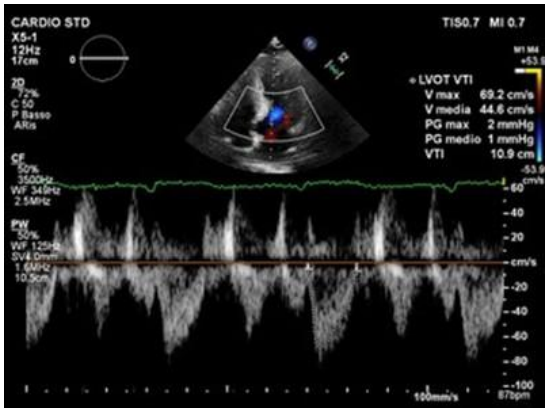

Figure 3: LVOT VTI 18 hours after landiolol administration (HR 78 bpm).

Table 2: Second echodynamic assessment (6 hours after landiolol administration).

Parameter	Value
HR (bpm)	85
SAP (mmHg)	80
DAP (mmHg)	60
EDV (mL)	155
ESV (mL)	110
LVOTd (mm)	22
LVOT VTI (cm)	12
EF (%)	30
SV (mL)	46
CO (L/min)	3.9
CI (L/min/mq)	2.22
CPO (W)	0.57
CPI (W/mq)	0.33
PET (ms)	61
ET (ms)	203
E-wave (cm/s)	56
A-wave (cm/s)	60
E/A ratio	0.93
IVRT (ms)	67
ePAWP (mmHg)	27
TAPSE (mm)	18
FAC (%)	38
Right(A-V) grad (mmHg)	/
TAPSE/PASP	/
sRVCPI	/
pAcT (ms)	140

Table 3: Third echodynamic assessment (18 hours after landiolol administration).

Parameter	Value
HR (bpm)	78
SAP (mmHg)	90
DAP (mmHg)	60
EDV (mL)	164
ESV (mL)	115
LVOTd (mm)	22
LVOT VTI (cm)	11
EF (%)	30
SV (mL)	40
CO (L/min)	3.1
CI (L/min/mq)	1.77
CPO (W)	0.48
CPI (W/mq)	0.27
PET (ms)	106
ET (ms)	245
E-wave (cm/s)	49
A-wave (cm/s)	45
E/A ratio	1.1
IVRT (ms)	100
ePAWP (mmHg)	8
TAPSE (mm)	16
FAC (%)	25
Right(A-V) grad (mmHg)	18
TAPSE/PASP	0.61
sRVCPI	288
pAcT (ms)	140

As previously described in a different setting [9], an intravenous beta-blocker allowed heart rate modulation and oxygen consumption optimization in a critically ill patient (SCAI C according to CSWG-modified classification10), also enabling the reduction of myocardial shear stress, a detrimental factor in such a scenario, as the subsequent dramatic events clearly demonstrated. Moreover, after achieving the desired heart rate with landiolol titration, with subsequent increase in eCO, it was possible to reduce and interrupt the infusion of norepinephrine, further improving the diastolic state of the left ventricle.

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