Heart rate control and hemodynamic improvement with Ivabradine in cardiogenic shock patients on mechanical circulatory support

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Background Cardiogenic shock (CS) is a life threatening condition due to primary cardiac dysfunction. First line therapy involves drug administration (including inotropes and/or vasopressors) up to mechanical circulatory support. Tachycardia is a compensatory mechanism in response to hypotension and low cardiac output or a side effect related to inotropic drugs. Ivabradine selectively acts on IKf channel in the sinoatrial node to reduce sinus heart rate without affecting inotropism. Its use in small non-randomized series of patients with CS was safe and well tolerated[1].

Methods We present the use of ivabradine in six patients with CS undertaking veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Data regarding haemodynamic and echocardiographic monitoring were collected before, at 12, 24 and 48 hours after ivabradine administration.

Results Ivabradine was administered through naso-gastric tube with a median time of 23 hours [IQR 18-28] since VA-ECMO implantation at the starting dose of 2.5 mg twice a day. Haemodynamic and echocardiographic parameters' variations are shown in table (panel A). Ivabradine was well tolerated and led to a significant reduction of heart rate after first administration ($p < 0.01$) (panel B). Echo-derived stroke volume increased significantly ($p < 0.001$) (panel C); so did cardiac index ($p < 0.001$) and left ventricular cardiac power index (p 0.005) (panel D). VA-ECMO rate pump and blood flow significantly decreased (respectively p 0.002, p 0.001). No significant changes were observed in arterial blood pressure ($p > 0.05$). Norepinephrine was down-titrated in all patients (p 0.01). Patients presented with cardiac arrest died due to neurological injury whereas the others were weaned off VA-ECMO and discharged alive.

Conclusions Ivabradine administration resulted in an effective reduction of heart rate leading to ventricular stroke volume allowing the reduction of extracorporeal flow support and vasopressors administration.

Reference

1.Chiu MH, Howlett JG, Sharma NC. Initiation of ivabradine in cardiogenic shock. ESC Heart Fail 2019;**6**(5):1088-1091.

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	Baseline	12 hours	24 hours	48 hours
HR (bpm)	101,0±2,4	80,2±7,6§	78,2±8,4	80,5±7,3
SBP (mmHg)	95,2±8,2	96,8±11,2	98,8±11,7	103,0±14,7
MAP (mmHg)	66,0±7,7	66,8±7,1	67,3±7,0	70,5±10,1
SV (ml)	15,7±7,9	27±1,5§	30,3±4,6	41±2,4§
CI (L/min/m^2)	1 ± 0,28	1,2 ± 0,17	1,34 ± 0,13 §	2,21 ± 0,18§
LVCPI (W)	0,14 ± 0,03	0,18 ± 0,02	0,16 ± 0,07	0,32 ± 0,04 *
VA-ECMO Pump Speed (rpm)	2595 [2500-2613]	2400 [2387-2427]*	2395 [2338-2400]	2400 [2360-2400]
VA-ECMO Blood Flow (L/min)	2,75 [2,70-2,80]	2,63 [2,43-2,66]	2,45 [2,36-2,55]	2,25 [2,20-2,33]§
Noradrenaline (mcg/kg/min)	0,12 [0,08-0,13]	0,09 [0,04-0,12]	0,07 [0,04-0,10]§	0,05 [0,04-0,10]§

§ p< 0.001 versus baseline; * p <0.01 versus baseline