Ivabradine and dobutamine associated as a pure inotropic drug in cardiogenic shock?

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Introduction: Dobutamine remains gold-standard treatment in cardiogenic shock. However, it exacerbates tachycardia, worsening heart failure. Ivabradine, a specific inhibitor of If channel, could reduce this deleterious effect in association with dobutamine in patients with cardiogenic shock.

We report the case of a 41-year-old woman admitted in intensive care unit for a severe heart failure with hemodynamic shock. She had no medical history.

She suffered from thoracic and epigastric pain and cholecystis was initially diagnosed with an indication of sphincterotomy. However, her clinical status progressively worsened with severe dyspnea and global heart failure requiring appropriate treatment. ECG showed inverted T waves in the lateral leads and echocardiography showed a dilated cardiomyopathy with severe systolic alteration (LVEF: 35%). Coronary angiogram was strictly normal. Finally, no evidence was found on cardiac MRI for ischemic process or myocarditis. She progressively worsened with renal and hepatic dysfunction. Troponin and inflammation markers remained negative. It was necessary to introduce dobutamine and intravenous diuretics but we noticed an initial increase in heart rate concomitantly with blood pressure. We added ivabradine in order to reduce heart rate without effect on blood pressure (fig). Her clinical status improved and dobutamine could be stopped after 5 days and beta-blockers were then introduced.

Discussion: Heart rate is a well-known marker of prognosis and tachycardia worsened by dobutamine could be deleterious to evolution of patient with cardiogenic shock. Ivabradine could be helpful in reducing heart rate without effect on blood pressure. However, this drug is indicated in stable heart failure but, to this day, hemodynamic instability is excluded. New prospective studies seem necessary to evaluate this benefit.

Conclusion: In cardiogenic shock, association of dobutamine and ivabradine could be interesting to create a pure inotropic drug.