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Use of Levosimendan in acute heart failure and its effect on renal function

Nefrología 2009;29(6):616-617.

Dear Editor,

Levosimendan is a relatively new inotropic agent, used in cases of decompensated heart failure, which has shown, in some reported cases, improvement in the patients' renal function.

We report the case of a 70-year-old male patient, monitored for grade III chronic kidney disease with no affiliated aetiology, with CCr of 45ml/min in the last examination. His medical history includes the following: monoclonal gammopathy of an uncertain significance; femoral cutaneous neuropathy; and multifactorial heart failure (ischaemic heart failure, non-ischaemic dilated cardiomyopathy, moderate pulmonary hypertension, heart disease with a pacemaker), with several episodes of decompensation causing his hospitalisation. He followed a regular treatment of Acenocoumarol, Carvedilol, Enalapril,

Torsemide, Digoxin, Atorvastatin, Alopurinol, Folic acid, Vitamin B₁₂, Ferrous sulphate and Neorecormon.

His hospitalisation was caused by a new decompensation of his heart failure, a deterioration of his dyspnoea, important levels of orthopnoea, episodes of paroxysmal nocturnal dyspnoea, ascites and oedematisation of limbs with a progressive decrease of diuresis. Moreover, he mentioned profuse haemorrhage due to a haemorrhoidal condition, and the examination confirmed a deterioration of the renal function.

During examination the patient experienced tachypnoea, with labial cyanosis, jugular ingurgitation and bad tolerance to the supine position. BP: 116/54. The cardiopulmonary auscultation showed rhythmic tones at 69 bpm, with R3 and a systolic heart murmur of 1/6 in the mitral and aortic areas; presence of rhonchi and diffuse wheezing, with crepitations in both pulmonary bases. The patient showed signs of hepatomegaly and inferior limbs oedematized until the knee with fovea. Diuresis remained at a daily level of around 2 litres. The examination revealed a deterioration of his renal function with Cr 3mg/dl, CCr estimated at: 24ml/min (baseline levels of 2mg/dl and 45ml/min, respectively). Urea 173mg/dl, Na 132mg/dl and K 6.4mg/dl. Hb: 5.7g/dl, requiring blood transfusion. The ECG showed a biventricular pacemaker rhythm at 70 bpm, and the thorax X-ray revealed an image of severe cardiomegaly with signs of vascular redistribution. Colonoscopy was carried out with anal fissure being the sole finding. The ECG showed severely dilated left cavities with severe global hypokinesia and FE estimated at 21%.

Intensive diuretic treatment was carried out with a poor response, leading to the decision to treat with Levosimendan in perfusion at a dose of 12.5 mg IV for 24 hours. The patient progressed satisfactorily for his congestive condition: dyspnoea disappeared and renal function

improved, showing creatinine figures of 1.8 mg/dl at discharge.

The patient was examined after 6 months, and the continuing improvement of his renal function was confirmed: Cr 1.5mg/dl, CCr 52ml/min.

In the clinical management of AKD, the fundamental factors are to maintain euvoemia, an effective cardiac output and adequate pressure of the renal perfusion. For this reason, we apply volume expansion, which is switched to the administration of vasopressors (dopamine and noradrenaline being the first choice) when it proves to be inadequate in restoring blood pressure. When peripheral hypoperfusion is caused by a condition of acute heart failure due to systolic dysfunction, the positive inotropic medicines with a vasodilatory effect through IV are a therapeutic option; Levosimendan being at the top of this group of drugs.¹

This drug has a triple action mechanism: first, it improves cardiac contractility by sensitising calcium with troponin C. On the other hand, it produces arterial and

venous vasodilatation by activating the potassium channels sensitive to adenosine triphosphate (ATP) of the plain vascular muscle fibre and, lastly, it produces phosphodiesterase III inhibitors.² It is administered through IV and it is initially prescribed at a loading dose of 6-12 μ /kg for 10 minutes, with a subsequent dose of 0.05-0.2 μ -7 kg/min for 24 hours. Its main side effects are owed to its vasodilatory effect: cephalalgia, nausea, hypotension, etc.³

Various studies exist in the literature, in which Levosimendan is used in patients hospitalised with heart failure. This confirms its beneficial effect for renal function, including its comparison with the effects of other types of medicine, such as dobutamine, while it maintains its effect during the 3 monitoring months, as in our case.^{4,5}

Therefore, to conclude, Levosimendan is a new alternative therapy in the treatment of decompensated heart failure with a secondary renal failure, improving the parameters of renal function and maintaining this effect for several months.

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