LEVOSIMENDAN FOR POSTOPERATIVE LOW CARDIAC OUTPUT SYNDROME IN A CHILD WITH CONGENITAL MITRAL INSUFFICIENCY

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Abstract. The treatment of low cardiac output syndrome after cardiac operations in children is a real therapeutical challenge. Usually catecholamines and phosphodyesterase inhibitors are the drugs of choice. Levosimendan has also been used in this context but only in a small number of patients so the clinical experience is very limited. This paper presents the successful use of levosimendan in a pediatric patient with postoperative low cardiac output syndrome. The treatment with levosimendan, associated to a number of other drugs and therapeutic procedures increased the myocardial contractility, improved the cardiac output and eventually led to the patient’s complete recovery.

Key words: congenital mitral insufficiency, levosimendan

CASE REPORT

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The low cardiac output syndrome is encountered in up to 25% of the children operated for congenital heart abnormalities and determines a significant increase of both morbidity and mortality after pediatric cardiac surgery.¹ Myocardial contractile dysfunction is encountered during and after almost all cardiac operations. The treatment usually includes drugs with positive inotropic effects belonging to either the catecholamine or the phosphodyesterase inhibitors group. A relatively new drug, levosimendan, has not only inotropic but also vasodilatating and antiischemic effects, so its use might have several advantages over the “classic” inotropic drugs. ²

Case report

A one year old girl was admitted to our hospital for the surgical correction of a severe congenital mitral insufficiency and a persistent ductus arteriosus with severe pulmonary hypertension and NYHA grade III heart failure. The surgical procedure was long and difficult. Initially a ligation of the ductus arteriosus, a mitral valve repair (annuloplasty) and a folding of the left atrium were performed under extracorporeal circulation. The intraoperative echocardiography revealed a significant residual mitral valve insufficiency, so the mitral valve was replaced with a Braille no 23 biological prosthesis. The operation lasted 11 hours and 15 minutes. The total extracorporeal circulation time was 4 hours and 20 minutes. At the end of the surgical correction the separation from the extracorporeal circulation required the use of a complex hemodynamic support. This was no surprise considering the length and the complexity of the surgical repair, followed by a myocardial ischemia that was documented by both electrical and biochemical changes. The hemodynamic support consisted initially of a combination of intravenous enoxymone 15mcg/kg/min, adrenaline 0.025mcg/kg/min, sodium nitroprusside 0.5mcg/kg/min and inhaled nitric oxide 20 ppm. Postoperatively the patient was mechanically ventilated. The myocardial contractility was measured by echocardiography. The cardiac output was assessed by following the clinical signs and by measuring the central venous oxygen saturation of the blood (SvO2). The postoperative course was difficult mainly because of a prolonged and severe myocardial dysfunction that needed prolonged inotropic support.

Immediately after the operation the same combination of intravenous enoxymone 15 mcg/kg/min and inhaled nitric oxide 20 ppm was used. The ejection fraction was less than 25% and the Svco2 was 57.65%. During the first postoperative
day the ejection fraction increased to 30%, but SvcO2 remained low (59.9%). The patient became thrombocytopenic, so the enoxymone was replaced by levosimendan. The treatment with levosimendan was initiated without an initial bolus. A dose of 0.2mcg/kg/min was administered for 24 hours. The therapy with levosimendan was not followed by any side effects. The treatment with adrenaline 0.025mcg/kg/min and sodium nitroprusside 0.5mcg/kg/min was also continued. The ejection fraction remained 30%, but the SvcO2 increase to 75.4%.

In the postoperative day 2 the sodium nitroprusside was replaced with nitroglycerin 0.5mcg/kg/min and dobutamine in a dose of 10mcg/kg/min was associated. Additionally, a right pleural effusion was drained. During the postoperative days 3 and 4 the hemodynamic support therapy was continued with the same doses as in day 2, the ejection fraction remained around 30% and the SvcO2 was 66.8% and 64.8% respectively. During the postoperative day 5 SvcO2 increased to 88.3% and it was possible to decrease the adrenaline dose to 0.025mcg/kg/min and the dobutamine dose to 7mcg/kg/min, while the nitroglycerin remained at 0.5mcg/kg/min. The patient was extubated during the same day. In the postoperative day 6 the ejection fraction exceeded 35%, so it was possible to stop the adrenaline infusion. At the same time the nitroglycerin was also discontinued. The mediastinal drains were removed during the postoperative day 7 and the inotropic support was discontinued the next day. From the postoperative day 7 oral digoxin, in a dose of 0.01mg/kg/day and enalapril 0.18mg/kg/day were administered.

The postoperative course was additionally complicated by a ventilator associated pneumonia that required antibiotic treatment. By the postoperative day 11 the ejection fraction was still above 35% and in the 12-th day after the operation the patient was transferred to the surgical ward. The myocardial contractility recovered 2 weeks after the operation – ejection fraction 50% under the treatment with digoxin, enalapril and furosemide. 3 weeks after the operation the patient was discharged from the hospital in good condition.

Discussion

Myocardial contractile dysfunction and the low cardiac output it can cause are recognized as severe complications of cardiac surgery. We believe that in this case the myocardial dysfunction and the hemodynamic failure were caused by several factors: the cardiac malformation with severe pulmonary hypertension and the complex and prolonged surgical procedure, which required a long extracorporeal circulation time and was followed by myocardial ischemia. An additional unwanted event was the pulmonary infection, caused by the prolonged mechanical ventilation. The hemodynamic support was initiated after the completion of the surgical repair and consisted of a combination of cathelcolamines and phosphodiesterase inhibitors plus a combination of vasodilators. It is worth notice the long duration of the inotropic therapy, especially with cathelcolamines who are prone to develop tolerance, so the efficiency of the treatment can decrease after several days, especially when additional factors (acidosis, infection) are present.[3] In this case levosimendan was not the first option but was added as a last chance to increase the cardiac output without increasing the vascular resistance and, even if it improved the cardiac output this effect appeared slowly and did not lead to an immediate decrease of the cathelcolamine requirements.

The blood pressure remained stable during the therapy with levosimendan. A possible explanation might be that we did not give an initial bolus, but we started directly the maintenance dose and the patient concomitantly received a cathelcolamine infusion. The heart rate was relatively high as one can be expected in a patient with fever, anemia and cathelcolamine infusion. However levosimendan did not produce a further increase of the heart rate nor did it induce arrhytmias. We conclude that levosimendan might be a good therapeutic alternative in pediatric cardiac surgery. Its advantages seem to be the good, fast and lasting inotropic effect, the lack of major side effects and the possibility to be combine with other drugs, be they inotropes or antiarrhytmics. Further studies are necessary in order to give levosimendan a more precise place on the list of the inotopic drugs that can be used in the surgical setting. A treatment protocol, maybe similar to the one used for treating adult patients with heart failure might also be of great help in this challenging clinical situation.

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References