Annals of Cardiology

Open Access

Case Report

Use of digoxin in children with low cardiac output syndrome and continuous renal replacement therapy

Montero-Yéboles R, MD[1]*; Díaz A, MD[1]; Lorenzo MJ, MD[1]; Ferrin A, MD[1]; Ruiz B, MD, PhD[2]; Pérez-Navero JL, MD, PhD[1]

[1]Pediatric Intensive Care Unit, Reina Sofia University Hospital, School of Medicine, University of Córdoba, Spain.

[2]Pediatric infectious diseases Unit, Reina Sofia University Hospital, School of Medicine, University of Córdoba, Spain.

Corresponding Author: Montero-Yéboles R

Department of Pediatrics at Reina Sofia University Hospital & Pediatric Research Unit, Maimónides Biomedical Research Institute of Córdoba (IMIBIC) at Córdoba University, Ave. Menéndez Pidal s/n. PC: 14004, Córdoba, Spain.

Tel: +34957010497, Fax: +34957010017; Email: rmyeboles@hotmail.com

Introduction

Digoxin is a cardiac glycoside with important positive inotropic, neurohormonal, and electrophysiologic effect, which is the basis for its use in patients with heart failure and ventricular dysfunction [1]. Digoxin is mainly eliminated by the kidney, therefore, patients with Low Cardiac Output Syndrome (LCOS) and impaired renal function are at risk for drug accumulation if not adjusted appropriately. Increased serum concentrations of digoxin put patients at risk for toxicity, which frequently manifests as electrolyte disturbances and haemodynamic compromise [2]. Moreover, patients undergoing renal replacement therapy are thought to have minimal effective renal elimination of digoxin. Consequently, digoxin plasma levels in this group of patients are unpredictable [2].

We present the case of a paediatric patient with Hypoplastic Left Heart Syndrome (HLHS) who developed ventricular dysfunction and LCOS, and Acute Kidney Injury (AKI) in the postoperative period after total cavopulmonary connection (Fontan procedure). LCOS did not respond to high doses of inotropes and diuretics and the AKI was treated with renal replacement therapy with Continuous Venovenous Haemodiafiltration (CCVHDF). Digoxin was added to the treatment to try to improve the haemodynamic dysfunction. Digoxinemia levels were monitored frequently to avoid toxicity, and digoxin doses were often adjusted to maintain plasma levels within the normal range (Table 1), achieving improvement of the cardiac function.

We conclude that, exceptionally, in patients with LCOS and AKI, when a high dose of inotropes is not adequate to improve the ventricular function, digoxin could be safely added under strict drug levels monitoring.

Article Details

Received: Sep 10, 2025

Accepted: Oct 27, 2025

Published: Nov 03, 2025

Annals of Cardiology - www.anncardiology.org

Citation: Montero-Yéboles R, Diaz A, Lorenzo MJ, Ferrin A, Ruiz B, et al. Use of digoxin in children with low cardiac output syndrome and continuous renal replacement therapy. Ann Cardiol. 2025; 2(2): 1012.

Table 1: Digoxin dose and plasma levels.

Day of treatment	Digoxin dose (µg/Kg/Day Iv)	Digoxinemia levels (Ng/Ml)
1	8	0.44
2	8	0.32
3	6.5	0.93
4	3.5	1.17
5	5.5	1.5
6	2.5	1.55
7	2.5	1.18
8	2.5	1.34
9	2.5	1.22
10	2	1.48
12	2	1.02
13	2	1.1
16	2	0.94
17	1.5	0.85
22	4	0.54
23	4	1.23
24	4	1.21
26	4	0.83
28	4	1.05
29	SUSPENDED	

Case report

We present the case of a four-year-old male with Hypoplastic Left Heart Syndrome. He had previous Norwood surgery in the neonatal period and bidirectional cavopulmonary shunt for median sternotomy.

A cardiac catheterisation at the age of three showed: no stenosis of pulmonary trunk and branches. PAP: 13/9 mmHg, mPAP 11 mmHg, PCP 12/6 mmHg, PCPm 11 mmHg. Fontan surgery was performed at this age with anastomosis of the inferior vena cava with a 20 mm Gore-tex tube and connection to the right pulmonary artery. A 5 mm fenestration between the extracardiac tube and the right atrium was performed.

Unfortunately, he developed Fontan failure with hepatomegaly, ascites, elevated central venous pressures, and large output from the chest drains. Echocardiography showed spontaneous closure of the fenestration. He progressively developed hypotension refractory to vasoactive drugs and anuria without response to diuretics, therefore, he was connected to CVVHDF three weeks after Fontan surgery, which was continued during his entire stay in the critical care unit.

CVVHDF parameters were blood flow 70 ml/min, replacement flow: 500 ml/h, dialysis flow: 400 ml/h and negative balance of 20 ml/h. Serial renal ultrasounds showed patent renal flow with no signs of thrombosis. Renal scintigraphy revealed acute tubular necrosis. Reoperation was performed to enlarge the fenestration of the extracardiac tube to reduce venous pressure and allow shunting through the fenestration towards the single atrium. After the intervention, he developed vasoplegia requiring volume, high doses of vasoactive drugs, terlipressin, methylene blue, and hydrocortisone. Given the refractory ventricular dysfunction, treatment with digoxin was started. Because the patient was on CVVHDF, digoxin plasma levels were frequently measured adjusting the dose accordingly (Table 1). As LCOS improved, treatment with digoxin was withdrawn. He always maintained digoxin levels within the normal range throughout treatment.

Discussion/Conclusion

Despite its efficacy, the use of digoxin has been steadily decreasing [3]. Different studies with adult patients have shown that Heart Failure (HF) and low serum digoxin concentrations are associated with a reduction in mortality and hospitalisation [1]. Low-dose digoxin in patients with left ventricular dysfunction has demonstrated improvement in the overall survival [3]. Low-dose digoxin has shown to be significantly more effective than ivabradine [4] in coronary patients with symptomatic diastolic heart failure and preserved systolic function.

Patients with LCOS and evidence of organ dysfunction require the use of inotropes, sometimes at very high doses. However, these therapies may have deleterious effects. An ideal inotropic drug should restore effective tissue perfusion by enhancing myocardial contractility without causing adverse effects. Such a drug is not available yet. Therefore, although experience with the use of digoxin in children with LCOS and renal failure is scarce, the addition of low dose digoxin to inotropic support could be an option before starting mechanical circulatory support as a last resort. In our patient with heart failure and acute renal failure resistant to inotropes, digoxin was started before considering mechanical circulatory support with extracorporeal membrane oxygenation, or with the Berlin Heart ventricular assist device.

In the absence of residual renal function among patients receiving CVVHDF, digoxin administration has been associated with negative outcomes because the proportion of digoxin clearance during CVVHDF remains uncertain [5].

Li X et al [6] have published that an intermittent low dose of digoxin has beneficial effects, and it is safe in patients with congestive heart failure on haemodialysis. Furthermore, continuous CVVHDF has been proposed as a treatment option in digoxin toxicity, especially in patients who suffer from severe renal dysfunction and/or do not have access to digoxin antidote [6]. Therefore, the use of digoxin in critically AKI paediatric patients, if indicated, requires close plasma levels monitoring to prevent adverse events. In our patient, digoxin doses were changed frequently to maintain plasma levels within the normal range.

We conclude that the use of digoxin with a proper plasma levels monitoring is safe and therapeutic effects can be achieved.

Declarations

Ethics approval and consent to participate: Local ethic committee approved this publication.

Consent: Written informed consent was obtained from the patient´s parents for publication of this case report.

Competing interests: The authors declare that they have no competing interests.

References

1. Ahmed AJ. Digoxin and reduction in mortality and hospitalization in geriatric heart failure: importance of low doses and low serum concentrations. Gerontol A Biol Sci Med Sci. 2007; 62: 323–329.

2. Delicata L, Gatt A, Paris JL, Bonello J. Symptomatic digoxin toxicity in a patient on haemodialysis. BMJ Case Rep. 2020; 13: e234899.

3. Wieser M, Rhyner D, Martinelli M, Suter T, Schnegg B, Bösch C, Wigger O, Dobner S, Hunziker L. Pharmacological therapy of heart failure with reduced ejection fraction. Ther Umsch. 2018; 75: 180–186.

4. Cocco G, Jerie P. Comparison between ivabradine and low-dose digoxin in the therapy of diastolic heart failure with preserved left ventricular systolic function. Clin Pract. 2013; 3: e29.

5. Gokalp C, Dogan AF, Aygun G, Kurultak I, Ustundag S. Continuous venovenous hemodialysis may be effective in digoxin removal in digoxin toxicity: a case report. Hemodial Int. 2020; 24: E58–E60.

6. Li X, Ao X, Liu Q, Yang J, Peng W, Tang R, Zhong Y, Meng T, Gan L, Zhou Q. Intermittent low-dose digoxin may be effective and safe in patients with chronic heart failure undergoing maintenance hemodialysis. Exp Ther Med. 2014; 8: 1689–1694.