# Vasopressin in perioperative management of congenital diaphragmatic hernia: a case report

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**Perioperative care of infants with diaphragmatic hernias can be a challenge because of pulmonary hypertension and systemic hypotension. The objective of this study was to report the usefulness of vasopressin infusion in improving pulmonary and systemic haemodynamics in an infant with congenital diaphragmatic hernia. Oxygenation failure, pulmonary hypertension and refractory systemic hypotension in infants with diaphragmatic hernia are managed by ventilation and conventional inotropes (dobutamine and dopamine). Vasopressin is a recent addition that exerts vasodilatory effects on the pulmonary circulation and vasotonic effects on the systemic circulation. The net effect is a reduction in pulmonary vascular resistance and improvement in cardiac output and reduced need for inotropes. This paradoxical response (vasodilation in some vascular beds) distinguishes it from other vasoconstrictor agents. The infant was administered intravenous vasopressin infusion for severe pulmonary hypertension that needed inhaled nitric oxide and for systemic hypotension that needed multiple inotropes under close echocardiographic monitoring. Informed parental consent and appropriate institutional ethics approval were obtained. Addition of vasopressin led to improved oxygenation and weaning off from nitric therapy. Improvement in cardiac output and blood pressure facilitated the weaning off from inotropes. Close echocardiographic monitoring was performed to ascertain the haemodynamic effects of vasopressin. Under echocardiographic monitoring, vasopressin is a useful adjunct for managing pulmonary and systemic perioperative haemodynamic instability in infants with diaphragmatic hernia. *Ann Pediatr Surg* 13:47–49 © 2017 Annals of Pediatric Surgery.**

**Annals of Pediatric Surgery** 2017, **13**:47–49

**Keywords: congenital diaphragmatic hernia, echocardiography, vasopressin**

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**Received** 20 April 2016 **accepted** 22 June 2016

## Introduction

Clinical outcomes in infants with congenital diaphragmatic hernia (CDH) may be affected by pulmonary hypoplasia, pulmonary hypertension and associated presence of congenital heart disease. Additionally, some infants may develop severe oxygenation failure and refractory systemic hypotension, making the management extremely challenging. Inhaled nitric oxide as a pulmonary vasodilator and conventional inotropes (dobutamine and dopamine) are generally used to establish and maintain haemodynamic stability. Vasopressin has been shown to be efficacious in relieving hypoxic pulmonary vasoconstriction and for the treatment of refractory systemic hypotension [1–3].

This case report details the usefulness of intravenous vasopressin infusion given under close functional echocardiographic monitoring in improving pulmonary and systemic haemodynamics.

## Case report

An antenatal ultrasound performed in a G3P1 mother at $20^4$ weeks' gestational age showed a left-sided CDH. An obstetric MRI 6 weeks later noted the spleen, a part of the liver and most of the small bowel and most of the transverse and descending colon and stomach to be in the thorax. The ratio between the observed and the expected total lung volume was 0.33.

This baby girl was born by caesarian section at $36^5$ weeks' gestational age due to nonreassuring foetal trace, and weighed 3344 g. She was immediately intubated and mechanically ventilated and a Replogle tube was inserted to decompress the stomach. Umbilical venous and arterial catheters were inserted for parenteral nutrition and invasive blood pressure monitoring. Sedation and muscle relaxation was administered using morphine and atracurium infusions. Conventional ventilation was changed to high-frequency ventilation after 30 min because of oxygenation failure and $CO_2$ retention. A wide presaturation and postsaturation gradient suggested pulmonary hypertension, which was confirmed by echocardiography; no congenital heart disease was noted. Inhaled nitric oxide was started at 20 ppm, which reduced the saturation gradient. Systemic hypotension was managed by escalating doses of dobutamine and dopamine. Vasopressin infusion was started at a dose of 0.04 U/kg/h (equivalent to 0.0006 U/kg/min). Table 1 and Fig. 1 depict the evolution of various parameters before and after vasopressin therapy. A dramatic improvement was noted over the next few hours in the oxygenation index, which was accompanied by improvements in biventricular cardiac output, blood pressure and pulmonary hypertension. The suprasystemic pulmonary pressures turned subsystemic and remained so after cessation of vasopressin. Nitric therapy and inotropes were gradually weaned off. The infant was operated on the fifth postnatal day. A type C defect in the left hemidiaphragm was managed with primary repair [4]. Vasopressin was continued during surgery, with no desaturations/pre–post saturation gradient or system hypotension noted intraoperatively.

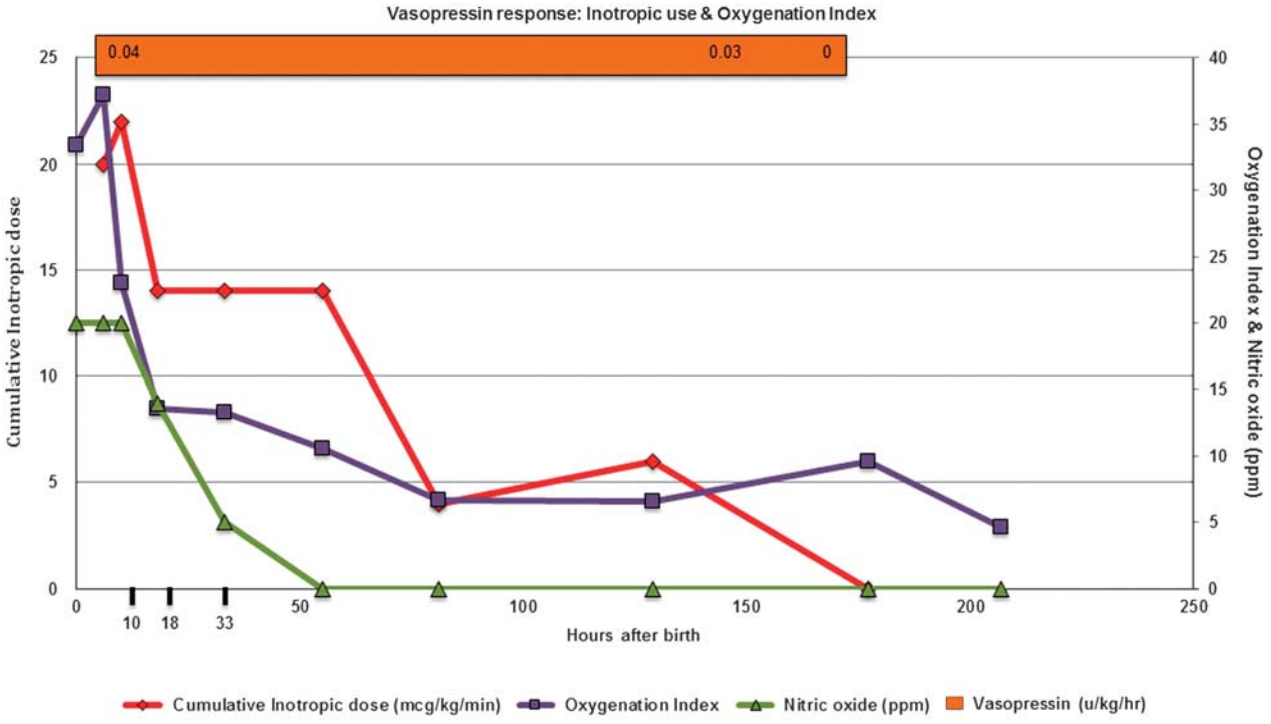
1687-4137 © 2017 Annals of Pediatric Surgery DOI: 10.1097/01.XPS.0000489147.17551.89

**Table 1 Echocardiographic parameters during the perioperative course**

| Days of life | PDA size (mm) | TR velocity (m/s)[a] | RVSP (mmHg) | Right ventricular SV (ml/kg) | % RTL shunt through PDA | SBP | RVSP/SBP | Left ventricular SV (ml/kg) |
|---|---|---|---|---|---|---|---|---|
| 1 | 3.9 | 3.6 | 65.7 | 0.9 | 63 | 62 | 1.06 | 0.5 |
| 2 | 3.6 | 3.4 | 56.2 | 1.12 | 28 | 72 | 0.78 | 0.8 |
| 3 | 3.5 | 2.9 | 44 | 1.36 | 19 | 62 | 0.7 | 1.1 |
| 4 | 2.9 | 2.8 | 41 | 1.34 | 20 | 72 | 0.6 | 1.2 |
| 5 | | | | Surgery | | | | |
| 6 | 2.9 | 3.2 | 50 | 1.5 | 0 | 75 | 0.6 | 1.3 |
| 8 | 2.6 | 2.1 | 22.6 | 1.8 | 0 | 70 | 0.3 | 1.45 |
| 11 | 2 | 2.1 | 22.6 | 1.9 | 0 | 75 | 0.3 | 1.5 |
| 28 | Closed | 1.7 | 16.6 | 1.9 | 0 | 68 | 0.2 | 1.5 |

PDA, patent ductus arteriosus; RTL, right to left; RVSP, right ventricular systolic pressure; SBP, systolic blood pressure; SV, stroke volume; TR, tricuspid regurgitation.
[a]Using modified Bernoulli's equation.

**Fig. 1**




Evolution of oxygenation index, nitric therapy and inotropic support before and after vasopressin administration.

The patient was weaned off it from the third post-operative day over a further 48 h and she was extubated 24 h after cessation of vasopressin. No rebound pulmonary hypertension or systemic hypotension was noted. The postoperative period was complicated by a mild would infection, which was accompanied by a raised C-reactive protein level, and was managed with vancomycin for a period of 5 days. Hyponatremia was managed with fluid restriction and sodium supplementation; it resolved rapidly after vasopressin cessation and she was discharged home after establishment of oral feeds. The study was approved by the institution's ethics board, and written informed consent was obtained from the parents.

## Discussion

Newborn infants with CDH can be extremely sick and sometimes require extracorporeal membrane oxygenation (ECMO). In this case, vasopressin therapy was associated with significant and dramatic improvement in pulmonary and systemic haemodynamics (improved oxygenation, reduced pulmonary hypertension and weaning off from nitric therapy along with improved blood pressure and weaning off from inotropes). Vasopressin usage was monitored by functional echocardiography.

Vasopressin analogue (terlipressin) use in CDH and pulmonary hypertension was first reported by Papoff *et al.* [5]. Others have reported its usage in nitric-unresponsive pulmonary hypertension secondary to meconium aspiration and in critically ill catecholamine-unresponsive patients with hypotension due to septic shock [1,6]. Its vasodilatory actions in the pulmonary circulation are mediated by the activation of endothelial oxytocin receptors, which in turn triggers activation of nitric oxide synthase [7]. Towards the systemic side, it

exerts vasotonic effects, which are mainly mediated by activation of V1 vascular receptors and modulation of ATP-sensitive potassium channels. This paradoxical action on differing circulations, in some ways, makes it an ideal agent in situations of pulmonary hypertension and oxygenation failure coexisting with systemic hypotension. Its role becomes significant especially in infants too sick to be transported for ECMO or when such a facility is not available. In a recent retrospective case series assessing the efficacy of vasopressin in stabilizing haemodynamics in CDH infants, vasopressin was seen to increase blood pressure, decrease the pulmonary to systemic pressure ratio, and oxygen requirement. In six of 13 infants, ECMO was no longer needed. Transient hyponatremia resolved with fluid restriction and sodium supplementation [8].

As experience with vasopressin is limited, close haemodynamic monitoring is desirable to document the pulmonary and systemic cardiovascular parameters. Relevant parameters are depicted in Table 1. Briefly, pulse Doppler assessment of the main pulmonary artery can give information about the right ventricular output. Greater than 30% duration of right-to-left shunting through the patent ductus arteriosus as a percentage of time of the cardiac cycle suggests significantly elevated pulmonary pressure [9]. Maximum velocity of the tricuspid regurgitation jet is used to estimate the right ventricular systolic pressure using modified Bernoulli's equation. Left ventricular stroke volume is a useful systemic index that complements invasively measured blood pressure. Vasopressin therapy resulted in the improvement of biventricular parameters (lowering of ductal right-to-left shunting and tricuspid regurgitation velocity and improved cardiac outputs).

Vasopressin is a useful adjunctive therapy for the management of critically ill infants with CDH-associated pulmonary hypertension and systemic hypotension.

## Acknowledgements

### Conflicts of interest

There are no conflicts of interest.

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