

Use of Prostaglandin E2 in Neonatal Emergency Transport: A Case Report

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Abstract A male newborn born in a private hospital with undiagnosed interrupted aortic arch presented with circulatory collapse on day 2 is reported. The neonatal emergency transport team (NET) used intravenous Prostaglandin E2 (PGE2) infusion at the referring hospital for suspected systemic ductus dependent congenital heart disease. He responded with improved circulation and was safely transported to neonatal intensive care unit (NICU) of Queen Elizabeth Hospital.

Key words Interrupted aortic arch; Neonatal emergency transport; Newborn; Prostaglandin E

Introduction

Prostaglandin E1 and E2 were shown to be effective in symptomatic ductus dependent congenital heart diseases.¹⁻⁴ We report a case of undiagnosed interrupted aortic arch presented with circulatory collapse who was given PGE2 infusion at referring hospital. He was stabilized and transferred to NICU by the NET.

Case Report

A 2.9 Kg, full term male was born to a healthy mother by normal vaginal delivery in a private hospital. The antenatal history was unremarkable and there was no risk of neonatal sepsis. The family did not have history of congenital heart disease. He was well after birth but was noted to have severe tachypnea and marked pallor at 33 hours of life. CXR was reported to be abnormal with features of chest infection and was suspicious of cardiomegaly (Figure 1). He was treated for chest infection

with intravenous Penicillin and Netromycin and received 60% supplementary oxygen by head box to keep SaO₂ around 90%. Blood tests at private hospital were as follows: CBC: Hgb: 12.4 g/dl, WBC: 19.7 x 10⁹/L, Platelet: 261 x 10⁹/L; ABG: pH: 6.95, PaCO₂: 3.63 KPa, PaO₂: 9.14 KPa, BE: -23.4, HCO₃: 6 mmol/L. He was intubated for gasping respiration at 38 hours of life with ventilator setting FiO₂: 0.9, Pressure 17/3 cm H₂O, Rate 30 / min. He also received multiple doses of intravenous sodium bicarbonate and dopamine infusion of 5 ug/kg/min.

Our Neonatal Emergency Transport Team (NET) was consulted and we arrived at the referring hospital 1 hour later. He was pale and grey in colour, and his rectal temperature was 36°C. He was in severe respiratory distress and was on artificial ventilation. His circulation was poor with capillary refill time greater than 3 seconds. Peripheral pulses were not detectable and femoral pulses were feeble. The apical heart rate was 180/min, and his apex was displaced to 6th left intercostal space lateral to the left mid-clavicular line. The precordium was active with strong right ventricular impulses; the first heart sound was normal followed by single second heart sound, and gallop rhythm was heard. But there was no heart murmur. His oxygen saturation by pulse oximetry and blood pressure by dinamap were not recordable. His umbilical arterial blood pressure was 39/35 mmHg and these waveforms were flat in appearance. His liver was 3 cm below the right costal margin.

Base on the above clinical findings, continuous

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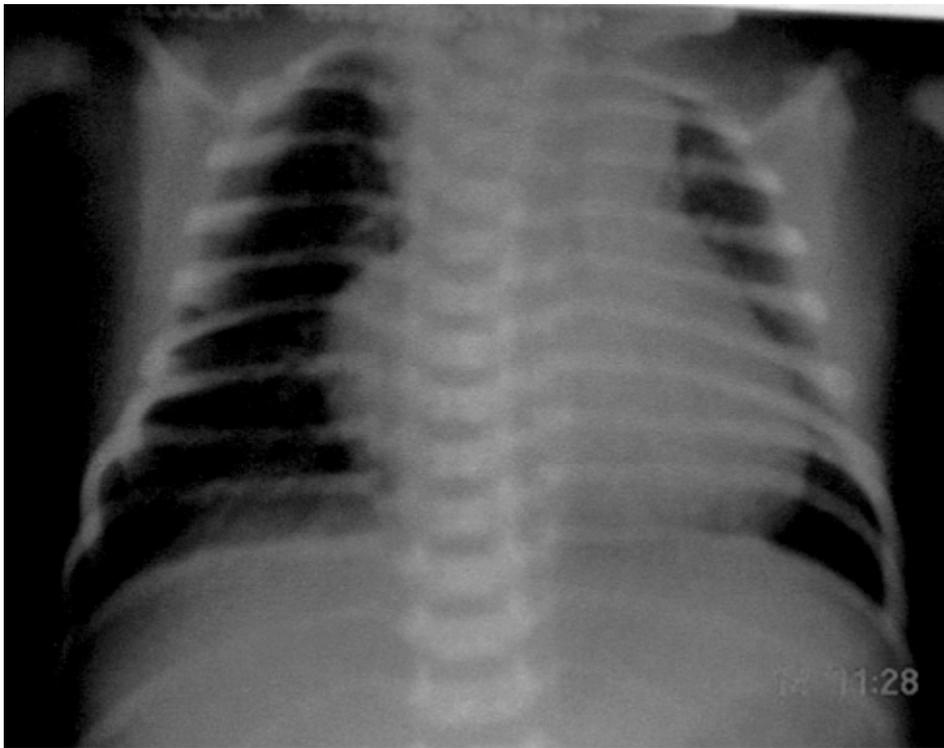


Figure 1 CXR at private hospital. Congested Lung fields and cardiomegaly.

intravenous PGE2 infusion was started at 0.01 ug/kg/ min for suspected severe symptomatic systemic ductus dependent congenital heart disease such as coarctation of aorta or interrupted aortic arch. He was also given intravenous furosemide, dopamine infusion 10 ug/kg/min, and intravenous sodium bicarbonate infusion to correct metabolic acidosis. By 15 minutes after starting PGE2 infusion, there was improvement in both arterial blood pressure from 39/35 mmHg to 51/39 mmHg and umbilical arterial waveforms. An ejection systolic murmur was heard at the left upper sternal border though his femoral pulses were still very weak. He was transported to our NICU for management. Parents were informed of his critically ill condition before departure.

On admission, the paediatric cardiologist echocardiographic evaluation confirmed the NET clinical diagnosis of systemic ductus dependent congenital heart disease namely interrupted aortic arch, 9 mm perimembranous ventricular septal defect, and 2.4 mm patent ductus arteriosus with depressed left ventricular function (LVFS: 22%) while on 10 ug/kg/min dopamine infusion. His femoral pulses were palpable three hours after starting PGE2 infusion and the umbilical arterial blood pressure was 70/41 mmHg. However, biochemical

evaluation confirmed coagulopathy, renal and cardiac impairments resulting from the preceding circulatory collapse. At 43 hours of life, he was transferred to Grantham Hospital for surgical intervention.

Discussion

Prostaglandin E1 was shown to be effective in pulmonary ductus dependent cyanotic congenital heart diseases, systemic ductus dependent acyanotic congenital heart diseases and some systemic – pulmonary common mixing congenital heart diseases.¹⁻⁴ PGE2 is as effective as PGE1, and there is oral preparation for PGE2.^{4,5} Despite various screening programmes for congenital heart disease before and after birth, a large proportion of infants with congenital heart defects remain undetected and come to the attention of the medical profession only after they develop symptoms.⁶ These symptomatic infants can present in extremis and die rapidly. Thus any delay in recognition and inefficient stabilization of these symptomatic neonates increases morbidity and mortality.⁷

Neonatal Emergency Transport team (NET) is a well-trained specialized team functioning as an ambulatory

neonatal intensive care unit. The team should be able to handle and stabilize ill neonates of various medical, surgical problems unaided in an unfamiliar environment, and transport ill neonates safely to the receiving NICU. In most situations, the exact diagnosis of congenital heart defects will be rarely available to the NET during the initial evaluation at the referring hospital. Therefore, the use of PGE1/ PGE2 infusion has to be based on clinical findings. Symptomatic ductus dependent congenital heart lesions that require PGE infusion usually fall into two main categories (Table 1). These are neonates with severe persistent cyanosis with/without heart murmur and circulatory collapse or severe heart failure with small volume pulses.⁸ They are symptomatic at the time when the ductus closes. Therefore, the attending NET should take good history and look for these symptoms and signs and perform appropriate investigations such as CXR, ECG and ABG to substantiate the working diagnosis. NET should also be able to discuss with the referring doctor, give appropriate advice, anticipate possible problems and to bring with the team the required medications. Other possible diagnoses should also be considered and covered such as severe sepsis, in born error of metabolism, myocarditis, arrhythmia and persistent pulmonary hypertension of newborn.

Use of PGE infusion will depend on the patient's clinical condition. A relatively well patient with ductus dependent lesion may well survive a transport without PGE, but it is unlikely that a patient in extremis will. Therefore, the threshold for starting PGE should be lower in the symptomatic group. Most ductus dependent lesions will respond PGE infusion promptly. Freed et al has shown that cyanotic pulmonary ductus dependent neonates showed good PaO₂ response to PGE1 infusion within 30 minutes while those acyanotic systemic ductus dependent neonates took average 1.5 hours to have maximal response.³ Thus, the infusion should be considered continued somewhat longer in acyanotic neonates before deciding that PGE will not be effective. In our case, the baby showed initial response with better arterial blood pressure in 15 minutes

but his femoral pulses were only well felt and blood pressure well improved by 3 hours. It has been suggested that favourable determinants of responsiveness are initially low arterial PaO₂ and an age of less than 96 hours, while unfavourable determinants of responsiveness are an irreversibly closed ductus, severe acidemia and collapse.³ There is no clear evidence that interarterial infusion is more effective than central or peripheral venous infusion.⁴ Also, PGE1 is effective in opening the ductus in both high and low oxygen concentrations.⁴ The infusion concentrations for PGE1 and PGE2 are different. The dose is from 0.01 to 0.1 ug/kg/min for PGE1 and 0.003 to 0.01 ug/kg/min for PGE2 respectively.⁹

PGE is not contraindicated in most congenital cardiac lesions except obstructed total anomalous pulmonary venous drainage. The side effects of PGE1 and PGE2 are similar. The most important reported short term side effects of PGE1 are those affecting the cardiovascular system (hypotension, vasodilatation, flushing and rhythm disturbances); the central nervous system (twitching and convulsion); respiratory system (apnoea and hypoventilation); the gastrointestinal system (frequent stools and diarrhoea); and pyrexia.¹⁰ Side effects are not directly related to dosage. It is advisable to intubate neonates who are on PGE infusion during transport for unpredictable apnoea. The infusion dose should then be reduced to avoid unnecessary complications. There were reports on ductus aneurysmal formation in neonates who had been given PGE1 and thus the ductus will be at risk of aneurysmal rupture when it is closed by surgery.⁵

Conclusion

This case report has shown that PGE2 is effective in symptomatic systemic ductus dependent congenital heart disease namely interrupted aortic arch. The NET was able to initiate PGE2 infusion correctly and promptly based on clinical findings. The attending NET or doctors taking care

Table 1 Examples of ductus dependent congenital heart diseases (Two different presentations.)

Persistent Cyanosis	Circulatory collapse/Congestive heart failure
Pulmonary atresia	Coarctation of aorta
Critical pulmonary stenosis	Interrupted aortic arch
Tetralogy of Fallot	Aortic atresia
Tricuspid atresia	Mitral atresia
Transposition of great vessels	Hypoplastic left heart syndrome

of ill neonates should start PGE1/PGE2 promptly when ductus dependent congenital heart defect is suspected. Early and efficient stabilization is important for better outcome in this group of neonates.

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