

Case Report

Excessive maternal iced-tea intake - reversible cause of premature ductus arteriosus constriction

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ABSTRACT

Premature ductus arteriosus constriction is a rare entity associated with high neonatal morbidity and mortality. Most cases are idiopathic, with the major known cause being maternal use of nonsteroidal anti-inflammatory drugs. Polyphenol-rich foods have been proven to have a deleterious fetal hemodynamic effect that can lead to premature ductal constriction. We report a case of a neonate diagnosed with premature ductal arteriosus closure, whose only risk factor was an excessive maternal intake of commercial iced-tea during pregnancy. The authors aim to raise awareness to the impact of polyphenol-rich food consumption on ductus arteriosus, since maternal dietary advice can have a major preventive role. Furthermore, a timely diagnosis and intervention can prevent significant morbidity and mortality.

Keywords: Ductus arteriosus, Polyphenols, Gestation

INTRODUCTION

The ductus arteriosus (DA) is a crucial element in the fetal circulation, connecting the descending aorta to the main pulmonary artery and guiding oxygen-rich blood from the placenta to the systemic circulation. Premature constriction of DA during gestation is a rare phenomenon that can have hazardous complications such as right ventricular dysfunction, congestive heart failure, and even fetal death.¹

Maternal use of nonsteroidal anti-inflammatory drugs is the leading cause of isolated premature intra-uterine DA constriction but most cases remain unexplained.² The consumption of polyphenol-rich foods has been associated with premature DA constriction, due to the inhibition of

prostaglandin synthesis, which is responsible for fetal DA patency.^{3,4}

CASE REPORT

We report the case of a full-term male neonate who presented with tachypnea and grunting in the first hours of life. The mother was 31 years old, healthy and had no exposure to drugs or alcohol consumption. The pregnancy was uneventful, with adequate antenatal surveillance. The second trimester ultrasound showed an abnormal nuchal fold thickness, first and third trimester ultrasounds were normal. Amniocentesis was performed, with normal karyotype. He was born from an uncomplicated vaginal delivery, with an Apgar score of 9/10 at 1st and 5th minute of life, respectively. The family history was unremarkable.

At 3 hours of life, he presented with grunting and hypoglycemia (24 mg/dl) that persisted 30 minutes after milk ingestion. On physical examination, he had tachypnea (respiratory rate 90 bpm), subcostal indrawing and hypoxemia (SpO₂ 89%). He had good vitality, acrocyanosis but without lip cyanosis, adequate femoral pulses and peripheral perfusion. The cardiac and pulmonary auscultation were normal. The remaining physical examination was unremarkable. Admission in the neonatal intensive care unit was decided. Oxygen supplementation was started but with no improvement in oxygen saturation. There was no differential between pre and post-ductal oxygen saturation. Blood workup showed no signs of infection. A chest X-ray (Figure 1) was performed that showed an increase in cardiothoracic ratio (0.61) and a bilateral pneumomediastinum. He was observed by pediatric cardiology and the echocardiography revealed right ventricular concentric hypertrophy compressing the left ventricle (Figure 2), tricuspid regurgitation with right ventricle (RV) to right auricle pressure gradient of 60 mmHg, a completely closed arterial duct and a patent foramen ovale with right to left shunt. The diagnosis of premature (probably in utero) DA closure was made. History was revised to identify a causative agent. We inquired about herbal tea intake and other polyphenol-rich foods. The mother confirmed a high ingestion of commercial iced-tea (Lipton®) during gestation, up to one litre per day.

During hospital stay the neonate remained hemodynamically stable and there was a spontaneous ventilatory improvement, without oxygen therapy after day 7. Subsequent echocardiographic evaluations showed gradual regression of the cardiomyopathy and tricuspid insufficiency, and within two weeks the right ventricular size returned to normal. The neonate was discharged after 13 days, asymptomatic and with good cardiac function and maintained paediatric cardiology follow-up appointments, with favourable outcome.

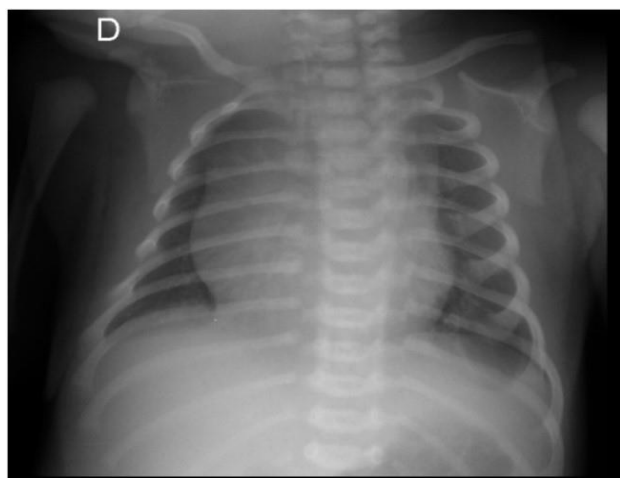


Figure 1: Chest x-ray showing an increased cardiothoracic ratio and a bilateral pneumomediastinum.



Figure 2: Right ventricle concentric hypertrophy, compressing the left ventricle.

DISCUSSION

In the fetal circulation, the DA connects the pulmonary artery to the aortic arch and allows 75% of the oxygenated right ventricular blood to bypass the high-resistance pulmonary vascular system to the systemic circulation.^{1,5} Patency of the DA in the fetal circulation is thought to be an active process, mostly regulated by a balance between opposing actions of oxygen and prostaglandin E₂.^{1,5} It is also known that during the third trimester, the DA becomes less sensitive to dilatory effects and more sensitive to constrictive effects of various chemicals. DA patency is maintained through high circulating levels and ductal synthesis of prostaglandins.⁶ In a healthy neonate, DA closure occurs on the first 24-72 hours of life.⁷

Premature constriction of DA is a rare event that can lead to subsequent complications such as right ventricular dysfunction, secondary tricuspid regurgitation, congestive heart failure or even death.^{1,6} It can range from a partial constriction to a total occlusion. It must be suspected in the presence of fetal or neonatal heart failure, as seen in our case.⁸

Most cases of premature DA closure are idiopathic. The enzyme cyclo-oxygenase (Cox-1 and 2) is responsible for maintaining the production of prostaglandin E₂. Therefore, a known cause of duct constriction is the use of Cox-2 inhibitors like indomethacin, non-steroidal anti-inflammatory drugs and steroids. Ductal sensitivity to Cox inhibitors increases with increasing gestational age.⁶

In the past years, studies have found that excessive intake of polyphenol-rich foods can also increase the risk of premature ductal closure. Polyphenol-rich foods include tea, dark chocolate, orange juice, red/purple grapes, berries and coffee. There is increasing evidence that these substances have an anti-inflammatory and antioxidant effect, secondary to inhibition of the metabolic route of prostaglandin, especially of Cox-2.^{3,4,9}

Fetal exposition to polyphenols in late gestation may trigger alterations in fetal ductal dynamics, such as higher

ductal velocities and lower pulsatility indexes, as well as larger RVs, and lead to premature ductal constriction. The cardiac alterations are reversible if the consumption is interrupted.^{3,10}

In the reported case there were no other identified risk factors other than an excessive commercial *iced-tea* consumption. After birth (with interruption of polyphenol exposure) there was complete reversal of the ductal constrictive effect and its hemodynamics consequences.

CONCLUSION

The medical community is often unaware of these harmful fetal effects, and therefore a number of these foods are freely consumed throughout gestation. Clinicals should be alert and provide maternal nutritional advice to decrease consumption of polyphenol-rich foods in late pregnancy. Furthermore, a timely diagnosis and intervention can be crucial to prevent severe morbidity and mortality.

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