NECROTIZING ENTEROCOLITIS AFTER ATRIAL FLUTTER: CASE REPORT

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Abstract

Necrotizing enterocolitis (NEC) has a multifactorial etiology and predominantly affects preterm infants, with only 13% of cases occurring in term infants. Hypoxia, sudden cardiovascular changes can also affect mesenteric perfusion. It is assumed that decreased mesenteric blood flow, followed by reperfusion, can contribute to intestinal damage, thereby increasing NEC incidence rates in term infants. This report presents a case of NEC development during follow-up for atrial flutter (AF). Repeated cardioversion caused sudden hemodynamic changes, causing NEC.

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ABSTRACT:

Necrotizing enterocolitis (NEC) has a multifactorial etiology and predominantly affects preterm infants, with only 13% of cases occurring in term infants. Hypoxia, sudden cardiovascular changes can also affect mesenteric perfusion. It is assumed that decreased mesenteric blood flow, followed by reperfusion, can contribute to intestinal damage, thereby increasing NEC incidence rates in term infants. This report presents a case of NEC development during follow-up for atrial flutter (AF). Repeated cardioversion caused sudden hemodynamic changes, causing NEC.

INTRODUCTION:

Necrotizing enterocolitis (NEC) has a multifactorial etiology and predominantly affects preterm infants, with only 13% of cases occurring in term infants (1) However, the etiology of NEC is not fully understood, and several mechanisms are thought to contribute to its development. It is assumed that intestinal ischemia

occurs when NEC affects a degraded or immature gut barrier in a susceptible host exposed to infectious agents alongside mediator cytokines or growth factors (2). Prematurity, hypoxia, mucosal immaturity, gastrointestinal tract immaturity, bacterial proliferation, and enteral feeding (especially formula), are the major predisposing factors for NEC.

In addition to hypoxia, sudden cardiovascular changes can also affect mesenteric perfusion. It is assumed that decreased mesenteric blood flow, followed by reperfusion, can contribute to intestinal damage, thereby increasing NEC incidence rates in infants affected by hypoxic-ischemic encephalopathy, polycythemia, or structural congenital heart disease. Thus, the pathophysiology of cardiovascular dynamics in fetal tachycardia is a potential cause of intestinal injury. All these changes decrease intestinal perfusion, which can lead to ischemic necrosis. This report presents a case of NEC development during follow-up for atrial flutter (AF).

CASE REPORT:

A male newborn—from a 28-year-old mother, who had experienced an unremarkable first pregnancy—was delivered via cesarean section at 37 weeks' gestational age. He exhibited Apgar scores of nine and ten at the first and fifth minutes, respectively. During antenatal follow-up, at the 35^{th} week non-stress test, his heartrate was 228bpm; AF was diagnosed; and digoxin and sotalol treatments were implemented.

The patient's main physical examination results were as follows: bodyweight: 3,000 grams (50^{th} – 90^{th} percentile); head circumference: 35cm (50^{th}–90^{th} percentile); body temperature: 36°C; heart rate: 180bpm; arterial blood pressure: 65/40mmHg; respiratory rate: 52 breaths per minute. Other physical examination findings were normal.

AF was detected via an electrocardiogram (ECG) (Fig. 1). The patient's right heart chambers were broadly dilated, and he presented mild tricuspid and mitral valve insufficiency. An atrial septum defect with a left to right shunt was observed upon echocardiographic examination. Within four hours of delivery, atrial overdrive pacing applied by transesophageal catheter. Tachycardia cycle length was 120-130 ms with 4:1 conduction to ventricle. Overdrive pacing was not successful, atrial fibrillation and sinus beats achieved but all converted to flutter in few seconds. Flecainide was implemented after the procedure. Since there was no response to the flecainide treatment by the second day, cardioversion was performed three times with 3 joule/kg energy. Flecainide was stopped, and amiodarone infusion was initiated (After 5mg / kg IV loading, 10 mcq / kg / day IV infusion) in combination with propranolol (3mg/kg/day) treatment. Clexane prophylaxis was initiated to prevent thrombosis. Under amiodarone treatment, cardioversion was performed with 2 Joule/kg for twice on the third postnatal day, but after termination of tachycardia, reentry started immediately.

16 hours after the last cardioversion, the patient developed abdominal distention and tenderness, poor sucking, hypotonicity, and bloody stool (Fig. 2). Pneumatosis intestinalis was not observed via direct abdominal X-ray. Coagulation parameters were normal, but thrombocytopenia was detected in the laboratory examination. NEC was diagnosed; broad spectrum antibiotics were initiated; and the patient's nutrition was discontinued. Trophic nutrition began on the fifth day of treatment, and antibiotherapy was ceased on the seventh day of treatment. The patient, who was fed orally by sucking, was discharged on the 13^{th} postnatal day with amiodarone and low molecular weight heparin treatments. In the Holter records of the patient at the time of discharge, the average heart rate was 146 (107-164) beats / min. Normal sinus rhythm was detected. Heart failure did not develop, as BNP values were found within normal limits.

DISCUSSION:

In newborns, AF generally occurs in the first seven days of life. Important factors indicating its clinical course are the beginning signs, the duration of AF, and the degree of ventricle response to AF. While AF is preserved by a circular macro reentrant pathway in the atrial wall, the atrioventricular (AV) node is not part of the reentry circuit. Atrial rates range from 300 to 500bpm, and this is usually associated with 2:1 AV conduction and ventricular rates between 150 and 250bpm. Normal or near-normal ventricular rates are also seen in AF with slower (3:1 or 4:1) AV conduction. In the absence of structural heart disease, AF is seen almost exclusively in infants during the third trimester or at birth. AF is generally tolerated, and fetal

hydrops and death are rare (3).

The goal of treatment is to either suppress the arrhythmia or, if this is not achieved, control the ventricular rate to a level closer to normal. If AF continues to occur, sinus rhythm can be restored via transesophageal overdrive pacing or synchronized electrical cardioversion, the latter of which was required by this patient. AF recurrence in neonates is unusual, and long-term treatment is rarely required.

AF can cause hypoperfusion and deoxygenation. When perfusion is limited, or when oxygenation is directed to vital organs, the "diving reflex" phenomenon can redistribute the blood flow for vital organs. Mesenchymal perfusion is reduced to protect these organs. This causes intestinal ischemia—that is, NEC.

Tachycardia due to AF also impairs intestinal perfusion as it disrupts cardiac hemodynamics. Previously, it was thought that both tachycardia-related hypoperfusion and reperfusion injury caused patients to develop NEC. In the extant literature, the first patient followed up due to supraventricular tachycardia (SVT) was born at 36 weeks and was diagnosed with SVT on the first postnatal day. NEC was observed on the seventh postnatal day, though the baby's tachycardia improved after antiarrhythmic therapy. The second baby followed up in the extant literature was born at 36 weeks and was diagnosed with SVT on the third postnatal day. In this baby, NEC also appeared on the seventh postnatal day. Both cases were discharged after NEC treatment. No other predisposing factors that could lead to NEC were observed in the cases in the literature or in the present case (4).

Previous research has reported that antenatal tachycardia impairs intestinal perfusion in neonates. It has also been asserted that, in some cases, cardiovascular changes caused by cardioversion may lead to NEC (5). In the literature, a male baby, diagnosed with SVT, underwent cardioversion, and, 14 hours later, he developed upper and lower gastrointestinal bleeding with a distended abdomen (6). That study, therefore, suggests continuous tachycardia may impair intestinal perfusion. In the present case, bloody stools were similarly detected 16 hours after cardioversion.

CONCLUSION:

NEC is a multifactorial disease occurring mostly in premature babies. In those treated for arrhythmia, as in this case, NEC may be observed due to impaired mesenteric perfusion. Through this case report, the researchers wanted to draw attention to the compelling instance of NEC that emerged during follow-up in this patient treated for AF.

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