CASE REPORT

Necrotising enterocolitis after supraventricular tachycardia: an unusual precursor to a common problem

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SUMMARY

Supraventricular tachycardia (SVT) is the most common neonatal arrhythmia. Necrotising enterocolitis (NEC) is a disease with multifactorial aetiology, most common in preterm infants. There are three previous case reports in the literature of NEC following neonatal SVT. We present two cases of late preterm infants with NEC, characterised by pneumatosis on abdominal X-ray and ultrasound, following recurrent SVT without haemodynamic instability. The infants were successfully treated with antibiotic therapy and bowel rest.

BACKGROUND

Necrotising enterocolitis (NEC) is a disorder with multifactorial aetiology, predominantly affecting preterm infants, with only 13% of cases occurring in term infants.¹ Predisposing factors include prematurity, gastrointestinal tract immaturity and mucosal immaturity, and enteral feeding.² Although NEC can occur in infants who have never been fed, enteral feeding is presumed to contribute to the pathogenesis of NEC by providing a substrate for bacterial proliferation in the gut.³⁻⁴ Cardiovascular instability leading to ischaemic injury of the gastrointestinal tract has also been postulated as a precursor to NEC.⁵⁻⁶ It is hypothesised that a reduction in mesenteric blood flow and subsequent reperfusion could contribute to bowel injury, hence the increased incidence of NEC in infants affected by hypoxic-ischaemic encephalopathy, polycythaemia and structural congenital heart disease.⁷⁻⁸ Despite the recognised risk factor of congenital heart disease in NEC, an association between supraventricular tachycardia (SVT) and NEC has only been reported in three cases previously.⁹⁻¹¹

CASE PRESENTATION

Case 1

A 3-day-old 36-week gestation 2.1 kg infant was transferred from an outside hospital for management of SVT. The antenatal history was notable for an anatomy ultrasound demonstrating possible Dandy-Walker malformation, prominent lateral ventricles, polydactyly and intrauterine growth restriction (IUGR). Postnatally the infant was diagnosed with Miller-Dieker syndrome. The baby was born via emergency caesarean section for IUGR and poor interval growth. The Apgar scores were 5 at 1 min, 6 at 5 min and 7 at 10 min.

On day of life 3, the baby had an episode of SVT with a heart rate of 280 beats per minute (figure 1). Four doses of intravenous adenosine were required for reversion to sinus rhythm, beginning at 0.1 mg/kg and increasing in increments of 0.05 mg/kg up to a maximum dose of 0.25 mg/kg. There was no loss of cardiac output and the baby maintained clinical stability, although an elevated lactate (3.8 mmol/L) was documented following the episode. The echocardiogram demonstrated a patent foramen ovale and a small patent ductus arteriosus. The baby was commenced on propranolol, with the dose increased due to recurrent SVT episodes. Feeds were commenced on day of life 3, and graded up to full volumes by day of life 8.

On day of life 7, the infant had rectal bleeding. The abdomen was distended and mildly tender but the infant was haemodynamically stable and afebrile. Abdominal X-ray did not show pneumatosis, portal venous gas or free air, however abdominal ultrasound demonstrated focal bowel wall echogenicities consistent with pneumatosis. The baby was made nil per os (NPO) and commenced on intravenous ampicillin, tobramycin and metronidazole in line with local antibiotic guidelines.

Due to NPO status, the propranolol was switched to intravenous dosing, which resulted in hypotension. Hence this was changed to intravenous esmolol. The baby completed a course of 7 days of NPO and intravenous antibiotics and was recommenced on feeds, as per unit-specific feeding protocols (figure 2). Cardiorespiratory stability was maintained throughout and there was no recurrence of NEC at 3 months.

Case 2

A 36-week large for gestational age infant was transferred on day 1 of life for further management of SVT. The maternal grandmother was known to have mitral valve prolapse; there was no further family history for congenital heart disease or arrhythmias. The infant was born via spontaneous vaginal delivery and Apgar scores were 7 at 1 min and 9 at 5 min.

The baby was noted to be febrile, hypoglycaemic and tachycardic in the labour and delivery unit. No risk factors for infection were present, however a blood culture was sent with commencement of intravenous ampicillin and tobramycin. The heart rate was greater than 250 beats per minute for 2 hours,

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Figure 1 ECG demonstrating supraventricular tachycardia (SVT).

after which a diagnosis of SVT was made. Vagal manoeuvres (ice and deep suctioning) were unsuccessful. Four doses of adenosine were administered with no effect, increasing from 0.1 mg/kg to 0.2 mg/kg. With a fifth dose of adenosine (0.25 mg/kg) given via an umbilical venous catheter the baby reverted to normal sinus rhythm. The infant was haemodynamically stable throughout the episode with normal blood pressure and perfusion. At approximately 12 hours of life, lactate was noted to be elevated (4.8 mmol/L) and subsequently normalised to 1.5 mmol/L.

An ECG revealed Wolff-Parkinson-White syndrome (figure 3) and the infant was started on propranolol, with the dose increased due to recurrent SVT. The echocardiogram demonstrated a fenestrated atrial septum and restrictive patent ductus arteriosus.

On day of life 7, the infant developed bloody stools with an abdominal radiograph and ultrasound showing colonic pneumatosis intestinalis (figure 4). The abdominal examination was unremarkable aside from some mild erythema at the left lower quadrant. The infant was placed NPO and commenced on a 7-day course of vancomycin, tobramycin and metronidazole. As a result of the NPO status, the propranolol was changed to equivalent intravenous dosing which required incremental increases to treat recurrent SVT. The infant completed 7 days of antibiotics and restarted feeds, which were graded up with no concerns. The intravenous propranolol was transitioned to oral flecainide. The infant was discharged home at 19 days of age on oral flecainide and full oral feeds with no recurrence at the time of follow-up at 2 months of age.

DISCUSSION

There are three previous case reports in the literature of NEC associated with SVT. The first is a 41-week term infant with a coexisting structural heart defect, dysmorphic features and skeletal abnormalities who presented with refractory SVT at 5

Figure 3 ECG demonstrating Wolff-Parkinson White syndrome. SVT, supraventricular tachycardia.

months of age and developed NEC, manifesting with abdominal distension and requiring a right hemicolectomy. This infant had further episodes of SVT associated with recurrence of NEC and the need for multiple laparotomies.⁹ The second case report describes a 33-week preterm infant with an antenatal diagnosis of SVT and subsequent hydrops fetalis who proceeded to have several postnatal episodes of SVT in the first 26 hours of life. The infant developed fulminant NEC after initiation of enteral feeds presenting with generalised symptoms of sepsis and decreased bowel sounds with abdominal tenderness, and subsequently died.¹¹ The third case is a 35-week preterm infant with an antenatal diagnosis of SVT, who continued to have several episodes of SVT postnatally requiring propranolol, digoxin and sotalol for control. The infant remained haemodynamically stable throughout these events but developed NEC on day 11, presenting with abdominal distension and requiring a laparotomy and resection of necrotic small bowel.¹⁰

In contrast, both infants at our centre were near term gestation, with no structural congenital heart disease on echocardiogram. Both infants required a higher dose of adenosine for reversion to normal sinus rhythm. There is emerging evidence that higher doses of adenosine are more effective in terminating SVT in young infants.¹² Both infants maintained clinical haemodynamic stability throughout the episodes, with normal blood pressure, perfusion and pulses, although mild elevation in lactate measurements was noted in both cases. This indicates that mesenteric blood flow could potentially be impaired and thus predispose to NEC, even in the absence of clinically evident shock. When perfusion is limited or oxygenation is redirected to vital organs, the phenomenon of 'diving reflex' can result, leading to a redistribution of blood flow to vital organs. During haemodynamic instability or impaired oxygenation, increased vascular resistance reduces blood flow to the mesentery.¹³ SVT could potentiate gut hypoperfusion and subsequent hypoxic-ischaemic injury to the mucosa as a result of persistent tachycardia and low cardiac output. Reperfusion injury is also a possibility with reversal of

NICU Guideline for the Initiation and Advancement of Enteral Feeding					
Weight	Initiation of Feeds	Day of Feeding	Volume of Feeds	Volume of Increase	Steps to Full Feeds
500-1000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
1000-1500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
1500-2000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
2000-2500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
2500-3000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
3000-3500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
3500-4000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
4000-4500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
4500-5000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
5000-5500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
5500-6000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
6000-6500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
6500-7000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
7000-7500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
7500-8000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
8000-8500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
8500-9000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
9000-9500g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs
9500-10000g	1.0ml/kg/24hrs	Day 1-3	0.1ml/kg/24hrs	0.1ml/kg/24hrs	0.1ml/kg/24hrs

Figure 2 Feeding protocol at The Hospital for Sick Children.

Figure 4 Abdominal X-ray in a patient with necrotising enterocolitis demonstrating widespread pneumatosis intestinalis.

SVT and stabilisation of cardiac output.¹³ We hypothesise that a reduction in mesenteric blood flow and ensuing reperfusion contributed to mucosal injury in the gastrointestinal tract and subsequent NEC in these cases.

The only identifiable risk factors for NEC in the patients in our centre were the episodes of SVT. In addition, the infant in case one had IUGR, with a birth weight of 2.1 kg. IUGR has not consistently been shown to be associated with NEC, in the absence of abnormal umbilical artery flow on antenatal ultrasounds. However, case control studies, along with large database analyses, have demonstrated an association between IUGR and NEC in a multivariate logistic regression model.^{14 15} While there was pneumatosis on X-ray or ultrasound in both cases, neither infant required surgical intervention. Conservative treatment with bowel rest, triple antibiotic and slow reintroduction of feeds was successful, with both infants well and without disease recurrence at the time of writing. SVT treatment was maintained with a change from oral propranolol to intravenous β -blocker therapy during the NEC treatment period.

Learning points

- Necrotising enterocolitis (NEC) is an important neonatal morbidity with multifactorial aetiology.
- Supraventricular tachycardia (SVT) may predispose to NEC via disturbance of mesenteric blood flow.
- Index of suspicion for NEC should remain high in infants who have experienced SVT.

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REFERENCES

- 1 Wiswell TE, Robertson CF, Jones TA, *et al.* Necrotizing enterocolitis in full-term infants. A case-control study. *Am J Dis Child* 1988;142:532–5.
- 2 Lin PW, Stoll BJ. Necrotising enterocolitis. *Lancet* 2006;368:1271–83.
- 3 Berseth CL. Feeding strategies and necrotizing enterocolitis. *Curr Opin Pediatr* 2005;17:170–3.
- 4 SIFT Investigators Group. Early enteral feeding strategies for very preterm infants: current evidence from Cochrane reviews. *Arch Dis Child Fetal Neonatal Ed* 2013;98:F470–2.
- 5 Chen Y, Chang KT, Lian DW, *et al.* The role of ischemia in necrotizing enterocolitis. *J Pediatr Surg* 2016;51:1255–61.
- 6 Nowicki P. Intestinal ischemia and necrotizing enterocolitis. *J Pediatr* 1990;117:S14–19.
- 7 Al Tawil K, Sumaily H, Ahmed IA, *et al.* Risk factors, characteristics and outcomes of necrotizing enterocolitis in late preterm and term infants. *J Neonatal Perinatal Med* 2013;6:125–30.
- 8 Motta C, Scott W, Mahony L, *et al.* The association of congenital heart disease with necrotizing enterocolitis in preterm infants: a birth cohort study. *J Perinatol* 2015;35:949–53.
- 9 Hajivassiliou CA, Pitkin J. Recurrent necrotizing enterocolitis associated with episodes of supraventricular tachycardia. *J Pediatr Surg* 1998;33:1569–70.
- 10 Hanna MH. Necrotizing enterocolitis after recurrent supraventricular tachycardia. *J Neonatal Perinatal Med* 2013;6:359–61.
- 11 Khalak R, Chess PR. Fulminant necrotizing enterocolitis in a premature neonate treated for supraventricular tachycardia. *J Perinatol* 1998;18:306–7.
- 12 Quail MA, Till J. Question 3 does a higher initial dose of adenosine improve cardioversion rates in supraventricular tachycardia? *Arch Dis Child* 2012;97:177–9.
- 13 Caplan MS, Jilling T. The pathophysiology of necrotizing enterocolitis. *Neoreviews* 2001;2:103e–9.
- 14 Dorling J, Kempley S, Leaf A. Feeding growth restricted preterm infants with abnormal antenatal Doppler results. *Arch Dis Child Fetal Neonatal Ed* 2005;90:F359–63.
- 15 Yee WH, Soraisham AS, Shah VS, *et al.* Incidence and timing of presentation of necrotizing enterocolitis in preterm infants. *Pediatrics* 2012;129:e298–304.

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