CASE REPORT

Congenital ductus arteriosus aneurysm: an unusual cause of transient neonatal hypertension

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SUMMARY

Case 1: A term male child was re-admitted on day 10 of life due to acute onset of respiratory distress. Physical examination revealed tachypnoea, tachycardia and blood pressure (BP) above the 95th centile in all four limbs. Cardiovascular examination revealed a short systolic murmur on the sternal border. Abdomen showed hepatomegaly of 3 cm below the costal margin. Chest X-ray showed a cardiothoracic ratio of 0.65 with normal vascularity. Ultrasound and Doppler of the kidneys and brain were normal. The high parasternal view showed a large ductus arteriosus aneurysm (DAA) of 2.0×2.5 cm. The baby was managed with inotropes and antihypertensives. CT angiogram showed 1.6×0.6 cm thrombosed DAA, which was extending from the posterior descending aorta to the ampulla. With the resolution of aneurysm BPs normalised and antihypertensives were stopped at 6 weeks of age. **Case 2:** A premature male neonate weighing 1.2 kg was admitted to the neonatal intensive care unit for respiratory distress syndrome. On the 4th day of life during routine measurement of vitals, the BP was consistently above 95th centile in all four limbs. Blood tests revealed thrombocytopenia that persisted inspite of single donor transfusions. The evaluation for sepsis was negative. The ultrasound and Dopplers of the kidneys and brain were all normal. A transthoracic echocardiogram showed a large DAA measuring 5×1.8 mm. Hypertension was managed with antihypertensives. Serial transthoracic echocardiogram showed organising DAA. CT angiogram showed 6 mm×2 mm thrombosed DAA. As the arterial BP normalised, antihypertensives were stopped on day 15 of life. The baby was discharged on day 29 of life and on follow-up BP remained normal.

BACKGROUND

Systemic hypertension (HTN) is an uncommon and under-recognised problem in the newborn. The estimated incidence of HTN in neonates is 1%.¹ Most infants with HTN are asymptomatic, and the onset is often insidious. Neonatal HTN will only be recognised by regular and routine measurement of blood pressures (BPs) in sick and premature infants. The aetiology and presentation of HTN in the newborn is varied and often the diagnosis and management is delayed or incomplete. Congenital ductus arteriosus aneurysm (DAA), a localised sacular or tubular dilation of the ductus arteriosus presenting as HTN in the newborn is not previously reported. In this case report, we present two cases of DAA presenting as HTN in the newborn.

CASE PRESENTATION

Case 1

An inborn male neonate was delivered by elective lower segment caesarean section (LSCS) to a primiparous mother at a gestational age of 40 weeks. The baby's birth weight was 3.29 kg with normal APGARS at 1 min and at 5 min. The baby developed respiratory distress (RD) soon after birth needing admission. The patient was managed with continuous positive airway pressure (CPAP) and the RD was passive on day 2 of life. Investigations revealed thrombocytopenia, which was managed conservatively as there was no clinical bleeding or bleeding tendency. The BP records were normal for birth weight and age. Breast feeding was established and the infant was shifted to the mothers' side on day 7, however got readmitted on day 10 of life due to acute onset of RD. Physical examination revealed tachypnoea, tachycardia and BP recordings (invasive and non-invasive) above the 95th centile in all four limbs. Cardiovascular examination revealed a short systolic murmur on the sternal border. Abdomen showed hepatomegaly of 3 cm below the costal margin. Chest X-ray showed a cardiothoracic ratio of 0.65 with normal vascularity. The complete haemogram, serum electrolytes and renal function tests were normal. There was metabolic acidosis on arterial blood gas with a base deficit of 15. Ultrasound and Doppler of the kidneys and brain were normal. Transthoracic echocardiogram performed in view of RD revealed structurally normal heart with poor systolic function. The measured shortening fraction was 20%. The high parasternal view showed a large DAA of 2.0×2.5 cm ([figure 1](#)). The baby was treated with dobutamine, milrinone and furosemide for poor cardiac function. Oral nifedipine 0.5 mg/kg/dose was started to control the HTN and later propranolol was added in view of poor response. Respiratory support included CPAP for 72 h. CT angiogram performed on day 15 of life in view of uncontrolled HTN showed 1.6×0.6 cm thrombosed DAA. The DAA was extending from the posterior descending aorta to the ampulla. Serial echocardiogram showed spontaneous resolution of ductus aneurysm over a period of 6 weeks. With the resolution of aneurysm BPs normalised and antihypertensives were stopped at 6 weeks of age.

Case 2

An inborn male neonate was born to a primiparous mother at a gestational age of 32 weeks and a birth weight of 1.2 kg by emergency LSCS. The baby

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Figure 1 High parasternal view showing a large ductus arteriosus aneurysm (DAA, ductus arteriosus aneurysm; DAo, descending aorta; PA, pulmonary artery).

was admitted to the neonatal unit and treated for respiratory distress syndrome (RDS) with surfactant and CPAP for 24 h. On the 4th day of life during routine measurement of vitals, BP was consistently above 95th centile in all four limbs. Blood tests revealed thrombocytopenia that persisted inspite of single donor transfusions. The evaluation for sepsis was negative. Complete haemogram, serum electrolytes and other renal functions were normal. The ultrasound and Doppler of the kidneys and brain were all normal. A routine transthoracic echocardiogram carried out in view of prematurity and RDS showed a large DAA measuring 5×1.8 mm. The systemic HTN was managed with frusemide of 1 mg/kg and nifedipine of 0.25 mg/kg/dose. Serial transthoracic echocardiogram showed organising DAA. CT angiogram performed on day 6 of life showed 6×2 mm thrombosed DAA (figures 2 and 3). As arterial BP normalised, antihypertensives were stopped on day 15 of life. The baby was discharged on day 29 of life and on follow-up BP remained normal.

Figure 2 CT angiogram showing thrombosed ductus arteriosus aneurysm.

Figure 3 CT angiogram showing thrombosed ductus arteriosus aneurysm.

In neither of the patients, there was any family history of HTN or intake of antihypertensive medications by the parents or first-degree relatives.

INVESTIGATIONS

Case 1

Ultrasound and Doppler of the kidneys and brain were normal.

High parasternal view showed a large DAA of 2.0×2.5 cm.

CT angiogram showed 1.6×0.6 cm thrombosed DAA.

Case 2

Ultrasound and Doppler of the kidneys and brain were normal.

Transthoracic echocardiogram showed large DAA measuring 5×1.8 mm.

CT angiogram showed 6×2 mm thrombosed DAA.

DIFFERENTIAL DIAGNOSIS

Cardiac conditions to consider in the differential diagnosis of neonatal HTN include thoracic aortic coarctation. Absence of differential BPs rules out this possibility.

Renovascular conditions to consider include

- ▶ Thromboembolism
- ▶ Renal artery stenosis
- ▶ Midabdominal aortic coarctation
- ▶ Renal venous thrombosis
- ▶ Compression of renal artery
- ▶ Idiopathic arterial calcification
- ▶ Congenital rubella syndrome

Renal parenchymal diseases to consider include

- ▶ Polycystic kidney disease
- ▶ Multicystic dysplastic kidney disease
- ▶ Tuberous sclerosis
- ▶ Ureteropelvic junction obstruction
- ▶ Acute tubular necrosis
- ▶ Cortical necrosis
- ▶ Interstitial nephritis
- ▶ Haemolytic-uraemic syndrome

Normal renal Doppler and ultrasound rules out the renal causes of HTN in the newborn.

Bronchopulmonary dysplasia and pneumothorax could cause HTN in newborns.

Normal electrolytes ruled out the endocrine conditions of neonatal HTN such as:

- ▶ Congenital adrenal hyperplasia

- ▶ Hyperaldosteronism
- ▶ Hyperthyroidism
- ▶ Pseudohypoaldosteronism type II in the index cases.

Medications/intoxications that may cause neonatal HTN include

- ▶ Dexamethasone/adrenergic agents/vitamin D intoxication/theophylline/caffeine
- ▶ Pancuronium/phenylephrine/maternal cocaine or heroin use

Tumours that should be considered in the differential diagnosis of neonatal HTN include the following:

- ▶ Neoplasia/Wilms' tumour/mesoblastic nephroma/neuroblastoma/pheochromocytoma

We did not investigate for these tumours in our cases.

Neurological conditions to consider in the differential diagnosis of neonatal HTN include the following:

- ▶ Pain
- ▶ Intracranial HTN
- ▶ Seizures
- ▶ Familial dysautonomia
- ▶ Subdural haematoma

Miscellaneous conditions to consider in the differential diagnosis of neonatal HTN include the following:

- ▶ Closure of abdominal wall defect
- ▶ Adrenal haemorrhage
- ▶ Hypercalcaemia
- ▶ Traction
- ▶ Extracorporeal membrane oxygenation
- ▶ Birth asphyxia
- ▶ Urological neoplasms

TREATMENT

Both the babies were managed with antihypertensives which included nifedipine and furosemide.

OUTCOME AND FOLLOW-UP

Both the cases were discharged with normal BP and now in regular follow-up with normal BP measurement.

DISCUSSION

Systemic HTN is an uncommon and under-recognised problem in the newborn. The estimated incidence of HTN in neonates is 1%.¹ Most infants with HTN are asymptomatic, and the onset is often insidious. Neonatal HTN will only be recognised by regular and routine measurement of BP in sick and premature infants. The aetiology and presentation of HTN in the newborn is varied and often the diagnosis and management is delayed or incomplete. Congenital DAA, a localised saccular or tubular dilation of the ductus arteriosus presenting as HTN in the newborn is not previously reported.

In our two cases, transient HTN was the presenting feature of DAA. Presence of aneurysm on the two-dimensional echocardiogram, persisting thrombocytopenia, absence of other

systemic causes of HTN and gradual control of HTN with the slow resolution of aneurysm favour an association of transient HTN with ductal aneurysm. The serial echocardiograms and serial BP recordings strengthen the association of DAA with HTN. We postulate compression on the sympathetic trunk as the reason for HTN in both these neonates as the thoracic sympathetic trunk travel in proximity to the posterior descending aorta. Thrombus formation in the aneurysm explains platelet consumption and hence persisting thrombocytopenia.

Most of the cases of DAA resolve spontaneously and only those with complications including compressive symptoms, rupture, propagation of thrombus and associated syndromes need surgical management.² DAA per se causing HTN is not described in the literature. Koneti *et al*³ reported four stages of DAA, that is, stage of acute compression, stage of loculation, thrombus formation and resolution. Our case presentations are during the period of compression and thrombus formation. The reduction of the size of DAA at the end of 6 weeks causing relief of compression to the adjacent thoracic structures may explain the association of 'stage of resolution' with 'normalisation of BP'.

Learning points

- ▶ Diagnosis of neonatal hypertension requires regular monitoring as the symptoms of neonatal hypertension are subtle and thus can be missed easily.
- ▶ Congenital ductus arteriosus aneurysm is a rare cause for the transient hypertension in neonates.
- ▶ Neonates with hypertension should always be evaluated for ductal aneurysm.
- ▶ Controlling the hypertension with medications until the end of spontaneous resolution is usually needed.

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