

in General Medicine, 3rd edn. Eds Fitzpatrick TB, Eisen AZ, Wolff K, Friedberg IM. New York, McGraw Hill, 1971, pp 2417-2419.

8. Caffey JAB. Infection: Infantile syphilis. *In: Pediatric X-ray Diagnosis*, 5th edn. Chicago, Year Book of Medical Publishers, 1970, pp 955-960.

## Spontaneous Aortic Thrombosis in a Neonate

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In childhood, the incidence of thrombotic disease, both spontaneous and catheter-induced, is highest in the neonatal period(1). A case of spontaneous aortic thrombosis following acute gastroenteritis and severe dehydration in a neonate is presented.

### Case Report

A 2.5 kg, full-term female baby was delivered by emergency LSCS (for non-progress of labor) to a second gravida

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mother with Apgar scores of 9,10 and 10. There were no maternal antenatal high risk factors. Postnatally, the child was being bottle fed with cow's milk.

At 70 h of age, the neonate presented with diarrhea. Examination revealed an active infant with normal hydration. Investigations revealed: hemoglobin 17 g/dl; TLC 8,300/mm<sup>3</sup> (60% polymorphs, 40% lymphocytes and band to total neutrophil count ratio of 0.33); micro ESR 1 mm/1st hour. Stool microscopy revealed numerous polymorphs. Blood culture was sterile and stool culture grew *Escherichia coli* sensitive to gentamicin and nalidixic acid.

The infant was started on septran and gentamicin parenterally and nalidixic acid orally. Bottle feeds were continued and half-strength WHO-ORS advised as additional feeds for replacing ongoing diarrheal fluid losses.

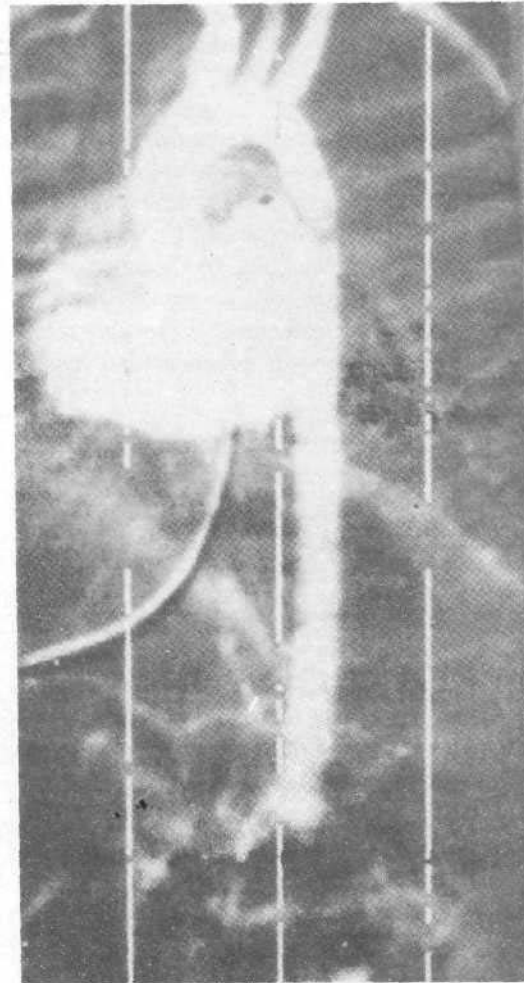
At 84 hours of age, the infant was in a state of shock. Anterior fontanelle was depressed and skin turgor was lost. Resuscitation with appropriate fluid replacement was carried out. Following resuscitation it was observed that while both radial pulses were palpable and the upper extremities were pink, the lower limbs from inguinal region downward (anteriorly) and sacral region downward (posteriorly) were cyanosed with both femoral pulses still unpalpable. Heart

rate was 180/minute, respiratory rate was 66/min and blood pressure in left arm was 80 mm Hg by flush method! Chest and precordial examination were normal. Liver was enlarged 4 cm below costal margin and spleen tip was palpable 0.5 cm below costal margin. Bowel sounds were normal. Neurologically the infant was lethargic with absent cry. Subtle seizures were noticed with hypertonia in both upper limbs. However, the both lower limbs were flaccid. Investigations revealed: arterial gases pH 6.59,  $pO_2$  124.4 mmHg,  $pCO_2$  19.3 mmHg, base excess 39 mmol/L,  $HCO_3(A)$  1.8 mmol/L; serum electrolytes—sodium 120 mmol/l, potassium 5 mmol/L and blood urea 30 mg/dl. Lumbar puncture was deferred. A provisional diagnosis of acute gastroenteritis with septicemia and meningitis with severe dehydration and aortic thrombosis was made. Parenteral cefotaxime and dilantin were added to ongoing treatment and oral nalidixic acid was omitted. Appropriate fluid replacement and correction of metabolic acidosis were carried out.

An urgent venous digital subtraction angiography (DSA) was planned. The umbilical vein was cannulated and the cannula advanced upto right atrium. CVP was 5 cm saline. DSA revealed complete obstruction of descending aorta below third lumbar vertebral level (*Fig. 1*).

At 94 hours of age arterial gases revealed pH 7.304,  $pO_2$  97.7 mm Hg,  $pCO_2$  16.1 mm Hg,  $HCO_3(A)$  7.8 mmol/L and base excess - 16.8 mmol/L. Urokinase was started intravenously via umbilical venous line for thrombolysis. A loading dose of 4000 IU/kg was given over 10 min by a constant infusion pump. Following this, maintenance therapy was given as a continuous infusion of 4000 IU/kg/h.

At 100 hours of age, capillary filling



*Fig.1. Digital subtraction angiogram of ascending and descending aorta showing complete block at level of aortic bifurcation into common-iliac arteries.*

time and color had improved over both thighs but femoral pulses had not yet appeared. Intractable seizures developed at 102 hours of age and the infant succumbed at 104 hours of age. Post-mortem lumbar puncture sent for culture grew *Streptococ-*

*cus pneumoniae*. The consent for autopsy was refused by the parents.

### Discussion

Aortic thrombosis in the neonate is rare. It may complicate umbilical artery catheterization(2), but can also occur spontaneously(3-8). The pathogenesis of aortic thrombosis is unknown, but, in the absence of causes for vessel wall abnormalities (*e.g.*, the presence of catheters), is probably related to blood flow disturbances and hypercoagulability.

Spontaneous aortic thrombosis has been reported in association with sepsis(4), congenital antithrombin III deficiency(6), and dehydration and shock(7). It has also been documented in an infant born to a diabetic mother(9).

The usual presenting features of aortic thrombosis are lower-limb mottling or cyanosis with absent femoral pulses or systolic gradient between the upper and lower limbs. Congestive heart failure, upper limb hypertension and hematuria have also been described(2,7).

Contrast angiography is the standard diagnostic procedure for the verification of large vessel thrombosis(10). It is obligatory before institution of thrombolytic therapy or performing vascular surgery. To avoid the adverse effects of hyperosmolar contrast media in the newborn, isotonic radio-opaque media (*e.g.*, iohexol) are preferable and administered whenever possible via the umbilical route.

Management of aortic thrombosis is a grey area. Spontaneous recovery without anticoagulant therapy or surgical removal has been described(11). However, this approach is not without risk. In adults, aortic thrombosis would usually be treated by sur-

gical restoration of vessel patency. This approach has also been used in two neonates with spontaneous aortic thrombosis with good results(8). However, in critically ill children with aortic thrombosis, surgical intervention may be difficult and medical thrombolysis with streptokinase or urokinase may be safer. Dosage for urokinase in the newborn are not established. It has been suggested that a loading dose of 4000 IU/kg be given intravenously over 10 min by a constant infusion pump; maintenance therapy should be initiated with a continuous infusion of 4000-6000 IU/kg/h, and dosage should be increased until one can demonstrate improved perfusion or clot dissolution by Doppler or real-time ultrasound studies(1). Bleeding complications must be looked for specifically during urokinase therapy. Thrombolytic therapy should be given only in a fully equipped and staffed neonatal intensive care unit.

### REFERENCES

1. Zipursky A, Schmidt BK. Neonatal thrombosis and embolism. *In: Neonatology: Pathophysiology and Management of the Newborn*. Ed Avery GB. Philadelphia, JB Lipincott Company, 1986, pp 687-690.
2. Henry CG, Gutierrez F, Lee JT, *et al*. Aortic thrombosis presenting as congestive heart failure: An umbilical artery catheter complication. *J Pediatr* 1981, 98: 820-822.
3. Rothstein JL. Embolism and thrombosis of the abdominal aorta in infancy and childhood. *Am J Dis Child* 1935, 49: 1578-1606.
4. Gross RE. Arterial embolism and thrombosis in infancy. *Am J Dis Child* 1945, 70: 74-76.
6. Bjarke B, Herin P, Blomback M. Neonatal aortic thrombosis. A possible clinical manifestation of congenital antithrombin

- III deficiency. *Acta Paediatr Scand* 1974, 63: 297-301.
7. Knowlson GT, Marsden HB. Aortic thrombosis in the newborn period. *Arch Dis Child* 1978, 53: 164-166.
  8. Me Faul RC, Keane JF, Nowicki ER, *et al.* Aortic thrombosis in the neonate. *J Thorac Cardiovasc Surg* 1981, 81: 334-337.
  9. Oppenheimer EH, Esterly JR. Thrombosis in the newborn: Comparison between infants of diabetic and non-diabetic mothers. *J Pediatr* 1965, 67: 549-556.
  10. Barnes RW. Noninvasive diagnostic techniques in peripheral vascular disease. *Am Heart J* 1979, 97: 241-258.
  11. Malin SW, Baumgart S, Rosenber HK, *et al.* Non-surgical management of obstructive aortic thrombosis complicated by renovascular hypertension in the neonate. *J Pediatr* 1985, 106: 630-634.

## Declining Trend in Tetanus Hospitalizations

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Tetanus, especially neonatal is one of the important causes of morbidity and mortality in many developing countries. In contrast, the incidence and mortality of tetanus has become negligible in developed countries

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like USA and UK. Concerted efforts have been made in India to contain tetanus through EPI and then UIP, in which thrust has also been given to antenatal immunization(1). In recent years, we gained an impression of reduction in tetanus related hospitalizations which prompted the current report.

### Material and Methods

The yearly admissions of tetanus, both neonatal and post-neonatal, in the last 11 years from 1983 to 1993 as well as the total pediatric admissions in the corresponding years in the Pediatric Department of LNJP Hospital were found out from the hospital records. The contribution of tetanus to Pediatric hospitalizations was worked out by expressing the tetanus admissions as a percentage of the total pediatric admissions. Statistical analysis was done by using linear regression method with time as an explanatory variable.

### Results

The yearly pediatric admissions, the tetanus admissions and contribution of