variable, it could be at the base or about 1 cm or more distal to the base(1).

The available literature regarding pathophysiology, mostly conjectural, suggests that the torsion of the appendix could either be a primary event or secondary to other pathologies. The proponents of 'primary' etiology blame it on the fan- shaped meso-appendix having a narrow base and the absence of azygotic folds that normally attach the appendix laterally(3). The other school of thought is that mucocele, lipoma, fecolith or inflammation causes distension of appendix rendering it unstable and more likely to twist. One postulation says that a fecolith could act as a point around which an irregularly contracting appendix might pivot(1). Absence of inflammation in few of the removed specimens supported the view(4). Another view is that inflammation of the appendix is the primary event with the resulting disten-sion of the distal part of appendix render- ing it unstable and making it prone to torsion(5).

One of the interesting speculations has been that intermittent appendicular torsion may be responsible for recurrent right iliac fossa pain in some children(4).

Y.K. Sarin,
D. Pathak,
Department of Pediatric Surgery,
Maulana Azad Medical College,
New Delhi, India.

REFERENCES

1. Gopal K, Kumar S, Grewal H. Torsion of the vermiform appendix. J Pediatr Surg 2005; 40: 446-447.
2. Merrett ND, Lubowski DZ, King DW. Torsion of the vermiform appendix: a case report and review of literature. Aust N Z J Surg 1992; 62: 981-983.
3. Dewan PA, Woodward A. Torsion of the vermiform appendix. J Pediatr Surg 1986; 21: 379-380.
4. Finch DR. Torsion of the appendix. B J Med Pract 1974; 28:391-392.
5. Beevors EC. Torsion of the appendix. Lancet 1920; 1: 597-598.

Post Intra Ventricular Hemorrhage Neonatal Cranial Diabetes Insipidus

Cranial diabetes insipidus (DI) is a rarely reported complication of severe intra-ventricular hemorrhage (IVH). It needs for its diagnosis in the neonatal period. One such case reported in this communication.

Case Report

A male baby was born at 29 weeks of gestation to a 35-year-old mother by emergency cesarean section which was necessitated because of prolonged rupture of membrane, maternal sepsis, foetal bradycardia and breech presentation. Despite antenatal steroid the baby had a stormy initial period as he developed respiratory distress and subsequently Persistent Pulmonary Hypertension of Newborn

(PPHN) needing high ventilatory supports, inotropes and prostacyclin to stabilise. He had a further episode of severe desaturation and bradycardia along with tonic convulsion on day 2. Cranial ultrasound scan confirmed suspected intracranial bleed with a bilateral Grade (IV) IVH and midline shift.

TABLE I–*Serum Sodium and Osmolality Before and After Treatment.*

	Before treatment			After treatment		
	Day 8	Day15	Day21	Day 22	Day 27	Day 35
Serum Na (mEq/L)	158	155	158	134	138	135
Serum osmolality (mOsm/kg)	314	309	319	273	281	269

On day 8 hypernatremia (Na = 158 meq/L) was noted which persisted despite increasing the total fluids to 180 ml/Kg/day. As he was also noted to have polyuria (5 to 7 ml/Kg/hr) and dropping body weight further bio-chemical tests were performed which revealed persistently raised serum osmolarity in comparison to urine osmolality (*Table I*). A central cause of DI was suspected due to significant weight loss, persistent hypernatremia, increased urinary output, low urine osmolality and grade (IV) bilateral IVH which was confirmed by intranasal Desmopressin (DDAVP) Challenge (*Table I*).

He was started on intranasal DDA VP the dose of which had to be adjusted frequently in accordance with his biochemical parameters. MRI scan done at corrected age of 38 weeks gestation revealed gross sequel of IVH with an intact hypothalamus and pituitary. A trial to stop DDAVP at the age of 7 months (corrected age 4.5 months) unfortunately failed. At present he is maintaining a stable biochemistry on a dose of DDAVP 3 μg (0.52 μg/kg) with steady weight gain on the 9th centile.

Discussion

The main etiological factors reported in neonatal cranial DI have been asphyxia, severe infections, CNS abnormalities and rarely IVH(1). Lack of Anti-diuretic Hormone (ADH) secretion due to impairment in the functions of magnacellular neurons from supraoptic and paraventricular nuclei is postulated to be the basic pathogenesis(2).

Diagnosis of cranial DI in neonate is extremely challenging and requires high index of suspicion(2). Hypernatremia is not unusual particularly in sick preterm but persistent hypernatremia not resolving despite increased fluid intake should be an important red flag. The combination of weight loss, hypernatremia, increased urinary output and low urine osmolality compared to serum has been suggested to indicate DI in newborns(3). These were present in our baby and awareness of this lead us to a trial of DDAVP which clinched the diagnosis. A medline search of articles in English literature revealed only two surviving baby with IVH as a cause of cranial DI(4,5). This might be related to the fact that IVH resulting in cranial DI are usually life threatening as well as the degree of difficulty in diagnosing it.

Rajiv Sinha,
Peter Martin,
St Peter's Hospital,
Chertsey, UK.

REFERENCES

1. Wang LC, Cohen ME, Duffner PK. Aetiologies of Central diabetes insipidus in children Pediatr Neurol 1994 ; 11: 273-277.
2. Krebes VL, Damiani D, Diniz EM, *et al*. Central diabetes insipidus as a complication of neonatal pathology: report of three cases. Acta Paediatrica Japonica 1998; 40: 146-149.
3. Giacoia GP, Watson S, Karathanos A. Treatment of neonatal diabetes insipidus with desmopressin Southern Med J 1984; 77: 75.
4. Atasay B , Berberoglu M, Gunlemez A, *et al*.

Management of central diabetes insipidus with oral desmopressin in a premature neonate. Journal of Pediatric Endocrinology & Metabolism 2004; 17: 227-230.

5. Yu VHY. Treatment of neonatal diabetes insipidus with desmopressin Aust Pediatr J 1980; 16: 284-286.

Recurrent Meningitis in a Family with C3 Deficiency

A 3-year-old girl presented to us in November 1998 with fever, headache and altered sensorium of two days duration. She was sick, irritable, drowsy and had neck rigidity. Blood counts showed leucocytosis. CSF came under high pressure and was turbid. It had 550 cells per mm^3 with 65% polymorphs and 35% lymphocytes. CSF proteins were 2 g/dL and glucose was 0.7 mg/dL. Gram stain showed gram positive cocci and CSF culture grew *Streptococcus pneumoniae*. She was treated with IV Ceftriaxone and made a complete and uneventful recovery. She presented again after 3½ with another episode of fever, lethargy and vomiting. CSF and blood culture again revealed streptococcus pneumoniae. She was again treated with IV Ceftriaxone and made complete recovery. She had a past history of two episodes of otitis media and one episode of *Escherichia coli* urinary tract infection.

In March 2002, her sibling, a boy aged 10 months presented with a short history of fever, vomiting and convulsions. Clinical diagnosis of acute meningitis was entertained. CSF and blood culture grew *Streptococcus pneumoniae*. He was also treated with IV Ceftriaxone and he made an uneventful recovery. He had chronic suppurative otitis media since the age of six months and also had recurrent chest infection and wheeze.

Family history showed three deaths due to probable meningitis (an uncle aged 28 years, a cousin aged nine years and sibling aged nine months).

In view of the history of recurrent infection and pneumococcal meningitis we investigated the two siblings for immunodeficiency. Both siblings had normal blood counts, immuno globulin levels and lymphocyte subsets. Total hemolytic complement activity, *i.e.,* CH 50 was 28.5%in the first child and 33.7% in the second (Normal range: 80-100%). Complement C3 levels were 0.06 g/L in both which were very low (Normal range 0.7 to 1.6 g/L). Other complement levels were within normal limits. Complement levels in parents were within normal limits. A diagnosis of familial C3 complement deficiency was made.

Both patients were put on prophylactic antibiotics and were immunized with *Pneumococcal* and *H. influenzae* vaccines.

Complement deficiencies can present at any age and make children more prone to infections with capsulated organisms leading to meningitis or sepsis.

Deficiency of Classical pathway defect (C1, C4 and C2) can predispose to autoimmune diseases like systemic lupus erythematosus, rheumatoid arthritis, and glomerulonephritis. They also predispose to infections with capsulated organisms, *i.e., Pneumococci* and *H.influenzae*.