Anuric unless catheterized

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We present a premature baby with critical illness and subsequent rent in the bladder. A 28-weeks, 1500g male neonate was born in poor general condition to a 19-year-old gravida 2 para 1 whose pregnancy was uneventful. The baby was transferred to this hospital intubated with the diagnosis of acute perinatal asphyxia, respiratory distress syndrome, presumed sepsis and shock. The clinical condition improved after general resuscitative measures, including administration of surfactant, inotropes, broad-spectrum antimicrobials and analgesia (morphine). Placement of umbilical arterial and venous catheter was uneventful. However from day 3, the abdomen became markedly distended, tender and shiny with omphalitis complicated by a spreading cellulitis. Central lines were removed. The baby became anasarcous and anuric, non-responsive to fluid challenge and frusemide. Passed urine freely after catheter was in position. Serum urea rose to 9mmol/L and creatinine, 194mmol/L. Ultrasound (US) showed fullness of the pelvicaliceal systems, ascites and 15-25cc of urine in the bladder. There was no gross or microscopic hematuria. Cultures remained sterile. After 7 days of maximum support, the baby was extubated and catheter was removed. Serum urea and creatinine, and abdominal US became normal. General condition remained good except that twice unless catheterized, the baby became anuric with ascites and elevated serum urea and creatinine. The possibility of a bladder rupture was then entertained. Micturating cystourethrogram (MCUG) demonstrated a leak of contrast from the right posterior bladder wall (**Figure 1**). The urethra was normal. Observational MCUG 5 days later was normal. No recurrence of the problem was noted on follow up to 15 months of age.

Bladder rupture in the absence of demonstrable obstruction is a distinct rarity.[1] It may not be readily identifiable in a septically ill neonate unless there is a high index of suspicion. Peritoneal and urine electrolytes (not requested in our baby) compared with serum values, will prove the presence of urinary ascites and autodialysis.[2,3] The clue which lead to our diagnosis was the episodes of anuria, ascites, rising serum urea and creatinine abating after indwelling catheterization. Micturating cystourethrogram confirmed our diagnosis. It is probable that septic embolization arising from the infected umbilicus could have extended to the bladder resulting in necrosis and weakening of the wall. The umbilical arteries originates from the internal iliac arteries and its branches are the

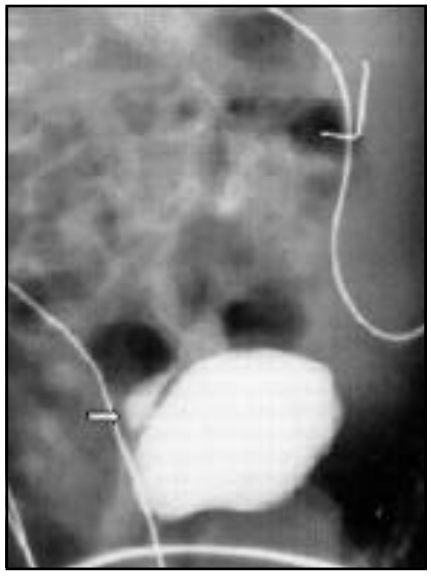

Figure 1 - Micturating cystourethrogram (lateral view) leak of contrast from the right posterior bladder wall.

superior and inferior vesical arteries supplying the bladder.[4] Detrusor areflexia as a result of asphyxia,[5] and hypotonic thin-walled nature of an immature bladder, and morphine infusion can cause bladder overdistension. Whether these trigger factors precipitated the bladder disruption remained speculative. We felt that the bladder drainage allowed time for the point of rupture to spontaneously heal.

Diagnosis of this unusual event should be considered in sick newborns presenting with anuria, and ascites resolving after catheterization. Prompt recognition should result to a satisfactory outcome.

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