CASE REPORT

Urinary ascites and anuria caused by bilateral fungal balls in a premature infant

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A case is reported of anuria and urinary ascites secondary to bilateral ureteropelvic obstruction by fungal balls. Management consisted of bilateral nephrostomy drainage with local irrigation with amphotericin B, and systemic antifungal treatment without surgery. Aspiration by paracentesis was performed for the urinary ascites and continuous drainage through an 8 Fr pig tail catheter for the urinoma. The literature on renal fungus balls in neonates and infants is reviewed.

The most common urinary fungus is *Candida*, producing asymptomatic candiduria, cystitis, and, with renal involvement, systemic candidiasis, primary renal candidiasis, and bezoar formation. Most patients have underlying disease or risk factors that favour the occurrence of systemic candidiasis. Predisposing factors described in the literature are prematurity, use of broad spectrum antibiotics, and use of intravenous catheters.¹ Renal fungal balls may be a cause of urinary tract obstruction and acute renal failure in these patients. Although several cases of anuria in premature neonates or infants secondary to bilateral ureteropelvic junction obstruction related to fungal balls have been reported, no cases of urinary ascites associated with fungal balls have been described. We report a unique case of anuria and urinary ascites in a premature infant secondary to bilateral ureteropelvic junction obstruction caused by fungal balls.

CASE REPORT

A 3 month old female infant presented with anuria for 23 hours and abdominal distension. The patient was born as the second twin at 30 weeks gestation and had been treated with broad spectrum antibiotics, parenteral nutrition, central vascular access, and mechanical ventilation for hyaline membrane disease in the neonatal intensive care unit for two months. One month after leaving intensive care, she was readmitted because of anuria and abdominal distension. Mild fever was observed, and laboratory data revealed a white blood cell count of 39.0×10^9 cells/l, haemoglobin concentration of 85 g/l, and creatinine concentration of 168.0 μ mol/l. Ultrasonography showed bilateral hydronephrosis with echogenic contents within the renal pelves, a large cystic mass in the left perinephric space, and ascites (fig 1A). Antegrade pyelography showed both collecting systems to be dilated, with multiple filling defects and extravasations from the upper calyces of the left kidney (fig 1B). Computed tomography showed multiple, small, low density regions in both renal parenchymes, and high density material filled the dilated pelves. A urinoma in the left prerenal space and ascites in the pelvic cavity were also observed (fig 2). Percutaneous nephrostomy on both sides was performed immediately after antegrade pyelography, and urine specimens were sent for analysis and fungal culture. Urinalysis showed many yeast-like cells, and *Candida albicans* was cultured from the specimens. Systemic antifungal treatment

with amphotericin B was started at a test dose of 0.1 mg/kg. The dose was increased gradually in daily increments over a three day period until a full dose of 0.5 mg/kg was achieved. Both renal pelves were irrigated at a rate of 40 mg/h or 45 mg/h every five hours daily with amphotericin B solution (50 mg/l). Continuous irrigation with 1 litre of solution with a slow drip infusion was maintained in both renal pelves. Aspiration by paracentesis was also performed for the urinary ascites, and continuous drainage through an 8 Fr pig tail catheter for the urinoma. After three weeks, the urinary tract obstructions were relieved, which was confirmed by ultrasonography and antegrade pyelography, and the percutaneous nephrostomy catheters were removed. Four weeks later, excretory urography showed good urinary drainage without filling defects within both renal pelves, and the 8 Fr pig tail catheter was removed. Fungal cultures from the urine were negative, and the systemic antifungal treatment was stopped six weeks after it had been started. There were no serious side effects associated with the treatment. The baby was discharged and convalescence was uneventful at the six month follow up.

DISCUSSION

Systemic candidiasis after intensive care in premature newborns is a well recognised complication, and fungal obstruction of the upper urinary tract in infants is potentially fatal. However, the diagnosis of fungal obstruction in high risk neonates is difficult to make and is often delayed or missed as there are no specific clinical features. Young age, small size, the presence of candidaemia, and withholding antifungal treatment are poor prognostic factors of fungal obstructive uropathy in neonates and infants.² Mortality in the cases reported to date has been lower after aggressive surgical and medical management. Because yeasts are not always identified in specimens obtained from patients, ultrasonography has been the most useful imaging modality in the early diagnosis of fungal balls.³

For patients with renal fungus balls without complete obstruction, surgical intervention such as placement of a nephrostomy tube is rarely necessary. In non-obstructing bilateral fungal balls, treatment with a combination of liposomal amphotericin B and fluconazole may remove the balls and obviate the need for surgical intervention.⁴ Oral fluconazole may be a safe and effective alternative for the management of systemic candidiasis in neonates.⁵ Complications requiring surgery, such as urinary tract obstruction, are uncommon. However, if fungal balls are found to be causing pelvic obstruction, treatment by insertion of bilateral nephrostomy catheters, instillation of amphotericin B into the renal pelvis, and parenteral and/or oral administration of antifungal drugs should be started.⁶ No guidelines are yet available for the indication, the mode of treatment, and length of treatment in neonates and infants with obstructing fungal balls. Sometimes, surgical removal of bezoars may be necessary to eradicate the fungal infection,⁷ but not all patients need such an intervention.⁸

Figure 1 (A) Ultrasonograph showing a urinoma (asterisk) in the left prerenal space. (B) Left antegrade pyelography showing a dilated collecting system and multiple filling defects (black asterisk) in the renal pelvis, and extravasations (white asterisk) from the upper calyces.

Invasive fungal infections are a major cause of morbidity and mortality in premature newborns. Because it may not always be possible to remove risk factors, a high index of suspicion, prompt diagnosis, and early institution of anti-fungal treatment are recommended. Our patient was successfully treated with percutaneous nephrostomy and amphotericin B irrigation, coupled with systemic antifungal treatment without surgical removal. Urinary ascites were aspirated by paracentesis, and the urinoma was continuously drained through an 8 Fr pig tail catheter. Anuria resulting from fungal balls in the upper urinary system has rarely been

Figure 2 Computed tomography showing both multiple renal cysts in renal parenchymes and high density material filling dilated pelves. A urinoma measuring 3×4 cm can be observed in the left prerenal space (asterisk).

reported. This is a very unusual case of anuria and urinary ascites in a premature infant caused by fungal balls.

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PostScript

LETTERS

Cold comfort for babies

CESDI Project 27/28 investigated the standards of care provided to infants who died after birth at 27–28 weeks gestation in England, Wales, and Northern Ireland during 1998–1999, comparing them with controls who survived.¹ Temperature on admission to

the neonatal unit was below the British Association of Perinatal Medicine/Royal College of Physicians (BAPM/RCP) standard of 36°C² in 73% of babies who died and 59% of controls. The CESDI report confirms the findings of the EPICure study,³ which showed that low admission temperature was an independent risk factor for neonatal death after adjustment for other known risks. Alarming, the CESDI report concludes that, as most units were

unable to achieve the standard, “the feasibility of achieving this standard must be questioned”! Vohra *et al*⁴ and Bjorklund and Hellstrom-Westas⁵ have shown that admission temperature is significantly increased if evaporative heat loss is prevented during resuscitation by occlusive wrapping after birth.

Since 2000, we have aimed to place all infants below 29 weeks gestation in polythene bags on the resuscitaire, before proceeding

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with their stabilisation. The policy was not followed universally at first, but since 2002 all babies below 29 weeks have been resuscitated in a plastic bag. The unit moved to a new hospital in March 2002, but there has been no change in delivery room temperature, or in the experience of medical and nursing staff attending resuscitation. Transport incubators were used in the old hospital to transfer the baby to the unit but this is now carried out on the resuscitaire.

The baby is slid into the bag up to the neck while still wet. The head is covered with a hat. No blankets are used, allowing radiant heat to warm the infant through the bag. Clinical inspection and auscultation can be performed through the bag, and, if vascular access is needed, a small hole can be cut in the bag. The infant is transported to the neonatal unit on the resuscitaire, eliminating the need for a transport incubator. Our resuscitaires have battery powered radiant heaters for the journey but these have not been important. The data shown here were gathered before the batteries were installed. Avoiding a move to a transport system has eliminated the associated risk of accidental extubation. On arrival in the unit, the baby is weighed and then placed in a warm humidified incubator before the bag is removed. Axillary temperature is then measured using an electronic thermometer.

Figure 1 shows the ranges of admission temperatures for inborn babies of 23–28 weeks gestation, over the past eight years. Now that all babies are placed in a plastic bag, we have abolished hypothermia. The mean (SD) admission temperature is now 37.0 (0.7) °C. Some infants now have mildly raised temperature on admission. They are the subject of further study but in most there are markers suggesting maternal infection.

Figure 1 Ranges of admission temperatures for inborn babies of 23–28 weeks gestation.

Table 1 Neonatal blood values

	UA end diastolic flow present (group 1; n = 108)	UA end diastolic flow absent (group 2; n = 26)	UA end diastolic flow reversed (group 3; n = 45)
WBCs/mm ³ (×1000)	9.1 (3.4)	7.3 (2.6–54.2)	7.8 (2.3–44.6)
Leucopenia	6 (5.6%)	5 (19.2%)	5 (11.1%)
Leucocytosis	5 (4.6%)	4 (15.4%)	14 (31.1%)‡
Haemoglobin (g/l)	164 (20)	151 (35)†	146 (25)†
Packed cell volume (%)	52.1 (6.5)	48 (11.5)†	46 (8.7)†
Anaemia	1 (0.9%)	2 (7.7%)	1 (2.2%)
Polycythaemia	21 (19.4%)	4 (15.4%)	4 (8.9%)
Platelets/mm ³ (×1000)	208.9 (74.1)	108 (32–412)*	119 (53.7)*
Thrombocytopenia	4 (3.7%)	12 (46.2%)‡	20 (44.4%)‡
NRBCs/100 WBCs	19 (0–595)	129 (2–2890)*	247 (12–1680)*, **
Raised NRBC count and thrombocytopenia	2 (1.8%)	11 (42.3%)‡	20 (44.4%)‡

Data are presented as mean (SD), median (range) or number (%).

UA, Umbilical artery; WBC, white blood cell; NRBC, nucleated red blood cell. *p < 0.05 compared with group 1, **p < 0.05 compared with group 2 (all Mann-Whitney U tests); †p < 0.05 compared with group 1 (one tailed t test); ‡p < 0.05 compared with group 1 (Fisher's exact test).

Using this simple, inexpensive technique the BAPM/RCP standard is readily achieved, independent of the clinical state and size of the infant. All units should have a policy for auditing admission temperature and a strategy for eliminating hypothermia during resuscitation, as this may be just as important as other more complex and expensive interventions.

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Haematological consequences of placental insufficiency

Abnormal development of the placental vasculature is responsible for maternal and fetal impacts of uteroplacental insufficiency.¹ Umbilical artery (UA) Doppler allows the non-invasive assessment of the severity of this vascular abnormality. UA end diastolic velocities are positive in mild placental insufficiency but are absent or reversed if 60–70% of the tertiary villous vessels are damaged. This observational study examines the relation between UA end diastolic velocity in growth restricted fetuses and haematological indices at birth. Singleton growth restricted neonates (birth weight < 10th centile) had a complete blood count within two hours of delivery. Results were related to the UA end diastolic velocity.

Among 179 participants, UA end diastolic velocity was positive in 108 fetuses (60%), absent in 26 (14%), and reversed in 45 (25%). Progressive abnormality of the UA waveform was associated with significant effects on white cell, red cell, and platelet counts (table 1).

White blood cell and nucleated red blood cell counts correlated positively ($r = 0.56$, $p < 0.001$) and were most strongly associated with base deficit and birthweight centile ($p < 0.05$). The platelet count was predominantly determined by UA blood flow resistance, and red blood cell indices by birthweight centile. Interestingly, the nucleated red blood cell count showed a negative correlation with the haemoglobin concentration ($r = -0.28$, $p < 0.001$) and platelet count ($r = -0.31$, $p < 0.001$).

Our results indicate that neonatal haematological consequences of placental insufficiency are complex and go beyond the expected polycythaemic response to intra-uterine hypoxaemia.² With increasing severity of placental dysfunction, enhanced red cell numbers in response to hypoxaemia are no longer observed, and metabolic compromise may even be associated with decreased red cell mass, thrombocytopenia, and increased nucleated red blood cells and white blood cells. UA Doppler is useful for identifying growth restricted neonates at high risk for these haematological disturbances.

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