Urinary ascites and anuria due to fungal balls

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

References

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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 resuscitation trolley. 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 resuscitator, eliminating the need for a transport incubator. Our resuscitators 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.

Table 1

<table>
<thead>
<tr>
<th>Haematological results</th>
<th>UA end diastolic flow</th>
<th>UA end diastolic flow</th>
<th>UA end diastolic flow</th>
</tr>
</thead>
<tbody>
<tr>
<td>present (group 1); n = 108</td>
<td>(100 mmHg)</td>
<td>(100 mmHg)</td>
<td>(100 mmHg)</td>
</tr>
<tr>
<td>Leucopenia</td>
<td>9 (3.4)</td>
<td>7 (2.6–5.4)</td>
<td>7 (2.3–4.4)</td>
</tr>
<tr>
<td>Leucocytosis</td>
<td>6 (5.6%)</td>
<td>5 (19.2%)</td>
<td>5 (11.1%)</td>
</tr>
<tr>
<td>Haemoglobin (g/l)</td>
<td>164 (20)</td>
<td>151 (35)</td>
<td>14 (31.1%)</td>
</tr>
<tr>
<td>Pack cell volume (%)</td>
<td>4 (5.6%)</td>
<td>4 (5.6%)</td>
<td>4 (5.6%)</td>
</tr>
<tr>
<td>Anaemia</td>
<td>1 (0.9%)</td>
<td>2 (7.7%)</td>
<td>1 (2.5%)</td>
</tr>
<tr>
<td>Platelet count (x1000/mm3)</td>
<td>280 (9) (74.1)</td>
<td>108 (32–42)</td>
<td>119 (53.7)</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>4 (3.7%)</td>
<td>12 (46.2%)</td>
<td>20 (44.4%)</td>
</tr>
<tr>
<td>NRBCs/100 WBCs</td>
<td>19 (6–95)</td>
<td>129 (2–2890)</td>
<td>247 (2–1680)</td>
</tr>
<tr>
<td>Raised NRBC count and thrombocytopenia</td>
<td>2 (1.8%)</td>
<td>11 (42.3%)</td>
<td>20 (44.4%)</td>
</tr>
</tbody>
</table>

Table 1 Neuronal values

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.

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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References


Haematological consequences of placental insufficiency

Abnormal development of the placental vasculature is responsible for maternal and fetal impacts of uteroplacental insufficiency. 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 birth weight centile (p < 0.05). The platelet count was predominantly determined by UA blood flow resistance, and red blood cell indices by birth weight 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 intrauterine hypoxaemia. With increasing severity of placental dysfunction, enhanced red cell mass in response to hypoxaemia is 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 in identifying growth restricted neonates at high risk for these haematological disturbances.

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References
