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Awareness of neonatal heat illness: a fatal outcome in a late preterm infant

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SUMMARY

Extreme heat impacts on child health and well-being due to immature thermoregulation, less sweating and a high surface-to-body mass ratio that make them more vulnerable to heat-related illness. Severe dehydration, especially if struggling with breastfeeding, renal failure and electrolyte imbalance, and neurological and cardiorespiratory failure may result. This late preterm was born healthy during a heatwave and developed lethargy and minimal responsiveness from day of life (DOL) 5, developed seizures at DOL 12, then demised at DOL 20. All hospitals involved in the diagnostic workup and treatment presumed severe sepsis and treated accordingly. However, microbiology results were always negative. In infants, especially if preterm, it is challenging to distinguish septic shock from heat-related illness, as well as other neonatal diagnoses. Medical history, including risk factors for infection and heat-related disease, must be thoroughly explored. It is important for healthcare workers and the public to be aware of potentially fatal neonatal heat-related illness.

BACKGROUND

Young children, especially preterm newborns, do not acclimatise physiologically in the same way as adults and are particularly vulnerable to high temperatures. As they have limited ability to thermoregulate, they are at increased risk of becoming dangerously hot.[1] They depend on caregivers' awareness and help to be protected from environmental heat and consequently heat-related illness with dehydration and electrolyte disturbances, even if regular breastfeeding or formula milk is provided.[2 3] In the setting of environmental heatwaves, newborns, especially preterm infants, experience greater risk of renal dehydration, respiratory disease, electrolyte imbalance and elevated body temperature.[4–10] Even a small change in environmental temperature due to climate change can impact health,[11] and hospital admissions for renal dysfunction increase during periods of high ambient temperature.[10 12 13]

Many African contexts are among the hottest in the world, are resource limited and have poor access to appropriate healthcare. These factors put African newborn infants at particular risk of climate-induced heat stress. However, little is known about the impacts of climate change on neonatal heat-related illness and death in any region of the world.[3 10 14 15] Further, it is often difficult, if not impossible, to distinguish illness caused by extreme heat from other pathologies in the neonatal period, especially infections and neurological conditions.

During periods of heatwaves, a newborn, whether preterm or term, in this case, a late preterm infant, may develop signs of severe hypovolaemia, abnormal weight loss, hypernatremia, haem concentration, anuria and acute kidney injury, non-responsiveness and poor appetite. All these signs and symptoms are also present in newborn sepsis that is a frequent neonatal diagnosis in resource-limited African settings. Meteorological data and feeding information should alert clinicians about potential heat-related illness. Other risk factors include firstborn infants that are a part of dyads with breastfeeding difficulties during extreme heat periods.[2 16 17]

The aim of this case report is to alert healthcare workers, the public and policy makers to heat-related effects on neonatal health. Preventive measures to avoid adverse effects of heat in neonates should be implemented in health systems guidelines and health political awareness campaigns. Educational programmes for healthcare workers must include neonatal heat illness as a differential diagnosis to septic shock with multi-organ failure. Heat-induced illness requires alternative awareness and treatment approaches, other than what is normally done for sepsis or other diagnoses for the ill lethargic infant.

CASE PRESENTATION

Patient information

The patient was a 36-week late preterm infant born during a heatwave. The mother was a healthy gravida 1 with no complications during pregnancy or delivery. The late preterm infant was born with a birth weight of 2300 g (appropriate for gestational age) and discharged home on day of life (DOL) 2. The mother described the high ambient temperature (39°C-40°C) as a challenge. The mother/family contacted health services on DOL 5, and the preterm infant was admitted to a primary hospital (figure 1). On DOL 12, the preterm infant was admitted to our tertiary hospital for intensive care and respiratory support after a brief stay in an inadequately resourced secondary hospital.

Clinical findings

Appearance, pulse, grimace, activity and respiration scores were 8/9/10 at 1, 5 and 10 min, respectively. At discharge from the delivery unit, the preterm infant had normal vital signs and tonicity, without dysmorphic features, jaundice or hepatosplenomegaly. According to the mother, during the preterm infant's days at home, the preterm infant cried most of the time and both the mother and preterm struggled with breastfeeding in the

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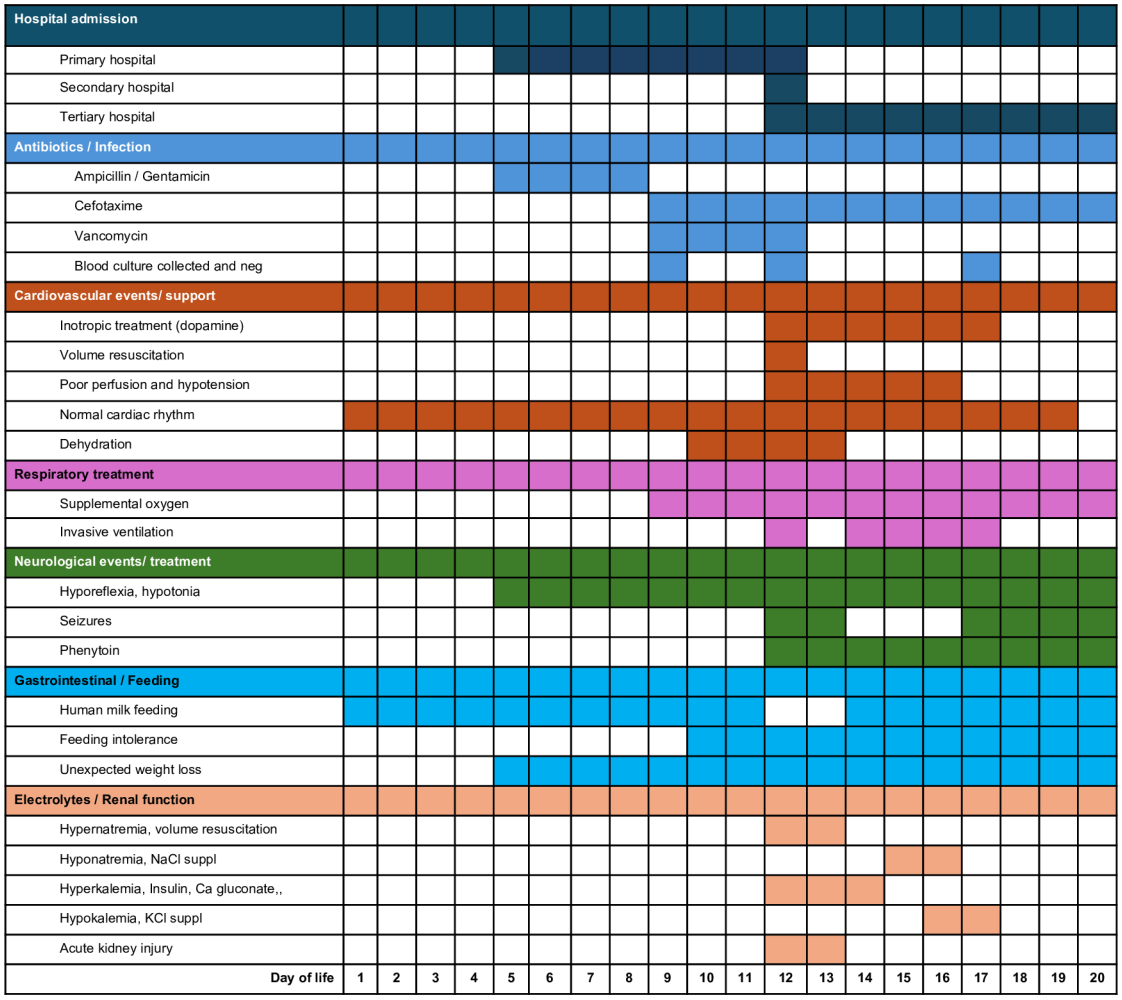


Figure 1 Time sequence of the diagnostic assessment and therapies.

extreme heat. The preterm infant became lethargic and sleepy with poor appetite and was therefore admitted to hospital. The preterm infant's condition worsened during hospitalisation in the primary hospital from DOL 5 to 12, with a need for intensive care with respiratory support.

Infection: The preterm infant's mother was healthy without any signs of infection during pregnancy or at delivery. Antenatal testing for HIV and syphilis was negative. No premature rupture of membranes was reported. Throughout all hospital admissions, the tentative diagnosis was sepsis, developing into septic shock and apnoeas. Clinical signs and symptoms included a combination of fast breathing and heart rate, irritability, lethargy, poor feeding, delayed capillary refill time, weak peripheral pulses and hypotension. There was no cyanosis. The preterm infant was normothermic. Available laboratory measures were not indicative of systemic infection (table 1). C-reactive protein or other biomarkers of infection are not available in our setting, only full blood cell counts. Blood cultures were repeatedly negative. The umbilical stump was dry and clean. The abdomen was not distended, making necrotising enterocolitis less likely, although an intra-abdominal infection is difficult to diagnose. Antibiotic treatment initiated at the primary hospitals was ampicillin and gentamicin, per hospital guidelines. This was changed to vancomycin and cefotaxime, due to failure of clinical improvement at the first hospital and continued as cefotaxime (monotherapy) on DOL 12, after transfer to our hospital, due to renal failure (table 1).

Neurologically: Upon admission to our hospital, the preterm infant was poorly responsive to stimuli and hypotonic but moving all limbs. Newborn reflexes were weak, and the fontanelle was full and slightly tense. The preterm infant deteriorated with more and longer apnoeic episodes, shallow and less spontaneous breathing. Clinical seizures were evident from DOL 12, and the preterm infant was initiated on anticonvulsive medication phenytoin. Clinical seizures reappeared on DOL 17, in spite of ongoing anticonvulsants. Cranial ultrasound at a delayed scan on DOL 16 due to radiological understaffing showed intraventricular haemorrhage grade 2 with normal cerebral structures for a late preterm infant. A lumbar puncture was never done due to the critical condition of the infant.

Weight development, nutrition and metabolism: Breastfeeding was initiated from the preterm infant's first DOLs, but the mother struggled with breastfeeding and the preterm infant's crying was interpreted as a sign of hunger. During all hospitalisations, a minimal volume of expressed breast milk was continued, on top of continuous dextrose intravenous infusion. Parenteral nutrition (PN) was not available in the primary hospital and was never provided in the other hospitals. On transfer to our tertiary hospital, PN was omitted due to deranged kidney and electrolyte values in the first days of admission (table 1). PN was later unavailable in our hospital. When admitted to our hospital, the preterm infant had a weight loss of 11% from birth weight. No enteral feeds were provided the first 2 days (DOL 12 and 13), but minimal enteral feeding was restarted when perfusion had

Table 1 Laboratory analyses from admission to our tertiary public hospital (DOL12) to demise (DOL 20)

	DOL 12, admission	DOL 12, 5 hours later	DOL 15	DOL 16	DOL 17	DOL 20, demised
Leukocytes, 10^9/L	19.9					
Neutrophils, %	64.7					
Lymphocytes, %	30.3					
Monocytes, %	14.3					
Platelets 10^9/L	580					
Haemoglobin (g/dl)	16.9					
MCV	98.5					
Na mmol/L	146	151	115	106	124	133
K mmol/L	7.9	5.9	4.5	3.7	2.8	4.0
Cl mmol/L	118	121.3	76	72	90	98
Ca mmol/L		2.5	2.8			
Mg mmol/L		0.92				
P_i mmol/L		0.69	0.94			
Urea mmol/L	19	13	4.3	5.7	4.8	3.6
Creatinine micromol/L	116	60	21	58	49	25
AST U/L	77	47			35	
ALT U/L	33	20			12.6	
GGT U/L	375					
ALP U/L	409	298			236	
Direct bilirubin	112	58			93	
Albumin g/L	41	36			30	
Total protein g/L	50	49			44.5	
*Arterial blood gas**						
pH	7.49	7.37				
BE	-0.3	-1.5				
HCO_3 mmol/L	24.2	23.8				
pCO_2 mmHg (kPa)	28.3 (3.76)	41.9 (5.51)				

*Arterial blood gas analysis was only available at DOL 12.
ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BE, base excess; GGT, gamma-glutamyl transferase; MCV, mean corpuscular volume.

almost normalised. At admission to our neonatal unit, we noted mild hyperglycaemia (7.9 mmol/L). Random blood glucose measures were normal or slightly supranormal on subsequent checks. The newborn's daily weight continued to fall, and 1865 g was the latest weight prior to death (19% less than birth weight, table 2).

Cardiovascular and respiratory condition: A mixed acidosis was noted at discharge from the secondary hospital where the preterm infant was intubated for respiratory support before transfer to us (table 1). On admission to our tertiary unit, the preterm infant showed respiratory alkalosis likely due to manual ventilation through the endotracheal tube. On admission to our tertiary hospital, the preterm infant was then severely ill-looking, hypoperfused and in need of haemodynamic and invasive respiratory support. At the tertiary hospital, the preterm infant was immediately put on a ventilator. Later that day, the on-call team extubated the preterm infant after not noting any respiratory distress. After extubation, there were frequent apnoeic episodes. When the preterm was noted to have poor respiratory efforts on supplemental oxygen (SpO_2 90–95%), the infant was put back on the ventilator from DOL 14 to 17. The chest was clinically clear and chest X-ray negative. Inotropic medication was started immediately after admission (DOL 12, dopamine), due to persistent poor perfusion and low blood pressure. Frequent blood pressure and perfusion checks (capillary refill time) showed moderate pallor and weak peripheral pulses with only intermittent improvement. Cardiac echocardiography was not available. No blood gas analysis was done after DOL 12, due to the hospital's lack of reagents. Blood haemoglobin (Hb) on admission was supranormal for a DOL 12 preterm infant, but declined after volume resuscitation, indicating initial dehydration (table 1).

Table 2 Weight development from birth to demise indicating dehydration and malnutrition

DOL	At birth	12	15	16	17	18	19–20
Weight	2300	2065	2118	1990	1885	1865	ND

DOL, Day of life; ND, no data.

Acute kidney injury and electrolyte imbalance: On admission to our tertiary hospital admittance (DOL 12), the preterm infant showed elevated urea and creatinine levels and electrolyte disturbances. Glomerular filtration rate measurements could not be calculated. Initially, the patient had no urine output. However, urine output improved after volume resuscitation. Kidney function measures normalised by DOL 15 (table 1). At admission, the preterm infant was hypernatremic, hyperkalaemic and hyperchloraemic and was therefore continued on dextrose infusions. Hyperkalaemia was treated with salbutamol inhalations, insulin infusion and calcium gluconate, which temporarily normalised the potassium levels. Later, the preterm infant developed hypokalaemia, hyponatraemia and hypochloraemia, possibly due to a failed rehydration strategy with continued dextrose infusions containing too little electrolytes, now corrected with electrolyte supplementation (figure 1 and table 1 show available electrolyte measures).

Indications of multiorgan dysfunction: At arrival at our hospital, the preterm infant demonstrated elevated gamma-glutamyl transferase (GGT) and alkaline phosphatase (ALP) (table 1). This was presumed to be due to the preterm infant's systemic illness, possibly metabolic bone disease, hypoxic neonatal cholestasis, malnutrition and/or organ hypoperfusion. The preterm infant showed moderate clinical jaundice, and the infant never received phototherapy. Liver and renal function later normalised, whereas direct bilirubin declined but remained elevated. Total bilirubin and indirect bilirubin measures were not available due to lack of reagents in the laboratory. Other diagnostic tests to exclude possible pathologies were never available or completed in our public tertiary unit.

Counselling of the mother: Counselling was initiated shortly after admission to our tertiary hospital and repeated several times. She was informed that her baby was critically ill with severe infection, multiorgan dysfunction and a poor prognosis. When we met her again to inform her of her infant's death on DOL 20, she exclaimed, 'Ohh – The heat killed my baby'. Before this counselling, no doctors or other healthcare workers had considered heat stroke and dehydration as a potential explanation to the neonate's symptoms and signs.

GLOBAL HEALTH PROBLEM LIST

Impacts of extreme heat on newborn health should be penetrated well and heat-related illness evaluated as a possible diagnosis.

Heat-related illness may be misdiagnosed, leading to inappropriate preventive and treatment measures.

During extreme heat periods, adequate hydration and nutrition are essential for newborn health, especially for preterm infants and in cases of breastfeeding challenges.

GLOBAL HEALTH PROBLEM ANALYSIS

Extreme heat may cause clinical complications,[18–20] also in newborns and infants,[3 6 8 10 21 22] especially if preventive measures are not in place. However, there is little scientific literature on direct clinical consequences of extreme heat exposure in newborns and infants, at least in sub-Saharan Africa. In an intervention study from Kenya, we reported on clinical effects of heat on newborn health, including the importance of staying hydrated, breastfeeding frequently and avoiding heavy clothing for newborns.[21]

Clinical adverse outcomes of extreme heat could be prevented. Vulnerable groups like preterm infants are at risk of heat-related illness and death.[23 24] First-time mothers, breastfeeding difficulties and extreme heat periods represent risks for adverse heat-related disease.[2 16 17] Climate hazards due to high ambient temperature, poor maternal and neonatal hydration, poor shading and cooling opportunities, delayed seeking of healthcare, lack of awareness of adequate hydration and breastfeeding problems may impact on acute neonatal and later health.[3 10]

Information about extreme heat exposure and breastfeeding challenges must be explored well in the history taking, and if positive, alert clinicians to think of heat-related illness as a differential diagnosis. The impact of extreme heat was even speculated by the mother as a cause of her preterm infant's poor condition and death. The preterm infant reported here was born healthy during a heatwave, without risk factors for sepsis and with negative blood cultures throughout. During the first DOLs, the late preterm infant was crying intensely with normal vital signs, indicating a normal neurological condition.

One of the most common neonatal diagnoses in our setting is presumed sepsis,[25] which is in accordance with worldwide data.[26 27] Presumed neonatal sepsis leads to initiation of antibiotic treatment, even despite negative or unavailable microbiology testing. In addition, we have limited access to infection parameters, like CRP and biomarkers for infection, which makes expanding the diagnosis more difficult. Further, we could not rule out the possibility of hypoxic events or hypoperfusion leading to multiorgan failure. This may have occurred during manual bag ventilation from the secondary hospital as indicated by the blood gas results.

The early exposure to extreme heat, according to the mother and in retrospect, may have impacted later neurological disease and the fatal outcome. Inadequate hydration and suboptimal early postnatal nutrition may have contributed to poor brain health and negative growth and development.[28] On admission to our hospital, the relatively high haemoglobin level for a DOL 12 preterm infant, abnormal weight loss, elevated urea and creatinine and electrolyte imbalances were in accordance with severe dehydration. Further, we cannot rule out if in-hospital inadequate nutrition and the rehydration and electrolyte supplementation treatment may have impacted on the outcomes including neurological complications, persistent seizures and finally death. Electrolyte imbalances with progressing hyponatraemia could be suggestive of syndrome of inappropriate antidiuretic hormone secretion that is associated with cerebral oedema and severe hyponatraemia. However, cerebral oedema due to rehydration with solutions containing too few electrolyte substitutes may be a more likely explanation for the worsening condition. After rehydration, we observed sharp electrolyte changes from high to low electrolyte levels. We speculate that the initially elevated liver function tests, which later normalised, may be due to the preterm infant's progressive dehydration and poor nutrition. The persistently elevated, but declining ALP, GGT and direct bilirubin measures could have been caused by hepatic cholestasis secondary to dehydration. However, we could not rule out other pathologies as a variety of tests and examinations were not available.

In retrospect, providing more nutrient supply to avoid severe weight loss might have been appropriate. However, after repeat consultations with dieticians and team members with strong nutritional backgrounds, the risk of worsening the preterm infant's acute kidney failure and electrolyte disturbances prompted us to take a more cautious approach. Insufficient nutrient supply may cause severe malnutrition associated with increased mortality risks,[29] due to suboptimal nutrition and presumably failure to efficiently absorb water and electrolytes that potentially lead to dehydration and electrolyte imbalances.[30] The primary hospital could not offer parenteral nutrition with protein and lipids when enteral nutrition intake was minimal. Also, after admission to our neonatal intensive care unit, the late preterm infant only received small amounts of breastmilk and never parenteral nutrition. The absence of adequate nutrition likely contributed to subsequent borderline protein and albumin levels, worrying growth pattern and continuous weight loss.

In our previous publications, we pointed out that climate change and global warming are some of the most complex and important contemporary public health threats for newborns. We also emphasised the importance of early weather warning information and forecasting, including preventive measures.[3 10 21 31] This should be in place for caregivers of newborn infants. Studies from rural Bangladesh and India demonstrated that those born during periods of extreme heat have increased risks of illness and death.[32 33] Other studies have shown a link between infant mortality and high daily temperatures.[34] In addition, newborns are more likely to be admitted to hospital if there is a high air temperature.[35] Further, the scientific literature shows that high temperatures increase the risk of preterm birth, low birth weight and stillbirth.[36 37]

Professionally planned and high-quality education for healthcare workers caring for newborns is needed. Training should include knowledge and practical sessions related to health hazards in days of extreme heat. Preventive measures to avoid adverse effects of heat should be implemented in health systems education, guiding recommendations and health political awareness systems. This is especially important in regions with poor housing, clean water scarcity and few cooling spaces indoors and outdoors. In communities, preventive measures should include early warning systems and public awareness with information about the importance of cooling, sufficient hydration and adequate nutrition,[21 31 38] also special public education campaigns directed at families with newborns and infants. The aim of this case report is therefore to alert health personnel and health authorities to the possible impact of extreme heat on newborn health, especially in preterm infants, and the potential differential diagnosis of heat-induced illness.

Learning points

- Climate change is one of the most complex and important contemporary public health threats for newborns, especially preterm infants' health.
- Early weather warning information and forecasting, including preventive measures, should be in place for caregivers of newborn infants.
- Newborns, especially preterms, are vulnerable to extreme heat. Early postnatal nutrition as well as adequate hydration is important to prevent heat-related neonatal morbidity and mortality.
- It may be difficult to distinguish the clinical presentation of neonatal septic shock and its neurological complications from heat-related illness in newborns, especially the preterms.
- Professionally planned and good education for healthcare personnel, including knowledge and training related to health hazards in extreme heat, is urgently needed, not least in poorly resourced regions in the global south.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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