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Extreme nutritional neglect leading to cardiac arrest in a child: a rare presentation of severe acute malnutrition in England

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Abstract

This case report from England describes an 8-year-old girl who suffered severe health complications culminating in cardiac arrest due to profound neglect and malnutrition.

The patient presented with cardiac arrest, which was attributed to her severe wasting. Upon admission, her body mass index measured 12.8 kg/m^2 (z-score ranging between -2 and -3), indicative of severe malnutrition. The patient presented with respiratory, renal, and hepatic failure, in addition to disseminated intravascular coagulation. Resuscitative measures were administered, and she underwent 162 hours of ventilation in the paediatric intensive care unit. Despite the gravity of her initial presentation, the patient exhibited a notable recovery.

Paediatric cardiac arrest stemming from extreme nutritional neglect is a rare occurrence in England. This case underscores the paramount importance of prompt intervention and comprehensive medical oversight. The patient's relatively favourable recovery underscores the potential for positive outcomes when timely and appropriate medical interventions are employed.

Introduction

This case study follows an 8-year-old girl of white descent who suffered extreme neglect, leading to a cardiac arrest as a complication of her severe malnutrition. At her presentation, she had a low weight-for-height z-score of -2.37 and an approximate body mass index of 12.8 kg/m^2 (z-score between -2 and -3). This defines severe undernutrition as per the Royal College of Paediatrics and Child Health charts and thinness as per the World Health Organisation charts.[1,2] In 2022, in England and Wales, malnutrition was noted to contribute to the death of 436 adults and children. Of which 77 deaths had this recorded as the underlying cause.[3] In our report, we will focus on the medical management of this rare presentation secondary to severe nutritional neglect.

Case Report

Resuscitation and acute management

The patient was carried into the Emergency Department with no palpable pulses and no respiratory effort. Accordingly, cardiopulmonary resuscitation (CPR) was performed. When agonal breathing was identified after 30 seconds, CPR was stopped. Subsequently, she had generalised tonic-clonic seizures for approximately 60 seconds, which spontaneously terminated without rescue medications. She was pale and emaciated with a weight of 16.4kg (<0.4th centile). Signs of severe neglect included a significant head lice infestation, necessitating a cap to prevent the lice from crawling onto the bed and infesting healthcare professionals. Fatal neglect, stemming from inadequate hydration, lack of adequate nourishment, and failure to administer medical care to children, continues to be a relatively infrequent and difficult-to-detect cause of death in developed nations.[4] Her body temperature was initially low, and after one hour of rewarming with a warm touch blanket and warmed fluids, her initial recordable temperature was 32.5°C. The initial venous blood gas is shown in Table 1.

As her capillary refill time was 5 seconds and her blood pressure (BP) was unrecordable, intraosseous access was gained, and two fluid boluses were given. The first BP was 80 systolic after the two fluid boluses. The initial Glasgow Coma Scale (GCS) was 3/15, which increased to 6/15 after fluid resuscitation. A subsequent external jugular vein and arterial line sited in her left femoral artery was gained. Such a presentation portrayed the deleterious consequences of severe malnutrition. The initial haemoglobin was 34g/L; this was difficult to quantify given how low it was using a blood gas analyser. Given this severe anaemia, two units of group O negative packed red blood cells were administered. Severe anaemia is among the additional health conditions

contributing to heightened mortality rates in severely malnourished children.[5] Additionally, given the severe lactic metabolic acidosis, two boluses of sodium bicarbonate 8.4% were administered. A computed tomography scan of her brain did not show any abnormalities. A urinary catheter was inserted, and the urinalysis showed ketone 1+, blood 3+, and protein 3+ with rose-coloured urine. The chest x-ray demonstrated a globular heart with hilar shadowing on the right side. Other medications that were given were 10% glucose at 2mL/kg (initial blood glucose level of 3.9mmol/L), vitamin K 5mg as the INR was 2.7, ceftriaxone (80mg/kg) and gentamycin (7mg/kg), paracetamol 360mg and full maintenance fluids. Given the persistent GCS<8, she was intubated with a size 5.0 cuffed endotracheal tube. She was ventilated with a tidal volume of 250 mL and FiO_2 of 80% to 100%.

A comprehensive assessment of severe acute malnutrition (SAM) in this patient included a thorough examination of her nutritional history, exposing a long history of explicit nutritional neglect.

The initial blood test results are shown in Table 2. The full blood count was not technically processable (the lab suspected erroneous sampling versus a diluted sample).

Whole body ischemia and reperfusion injury after cardiac arrest led to the massive inflammation clinically manifested in the post-cardiac arrest syndrome.[6] This would explain metabolic acidosis, high serum lactate, high LDH, high ALT, and acute kidney injury. Moreover, tissue damage in severe malnutrition contributes to increased serum LDH levels, liver cell damage, acute kidney injury, disseminated intravascular coagulation (DIC), possible haemolysis, and rhabdomyolysis. Children and adults with malnutrition have been shown to have a decreased glomerular filtration rate and renal plasma flow, as well as a lowered capacity to concentrate the urine and excrete an acid load.[7] In severe malnutrition, compromised renal function leads to decreased excretion of

magnesium and phosphorus contributing to their increased serum levels. There may be decreased production of active vitamin D, which impairs calcium absorption from the gut necessary for the activation of alkaline phosphatase. Low levels of the latter can contribute to impaired bone mineralisation and neuromuscular dysfunction. Hypoproteinaemia and hypoalbuminemia were noted. In children under the age of 5, hypoalbuminemia presents with greater severity among those diagnosed with kwashiorkor, characterised by symptoms such as oedema, fatty liver, skin lesions, and alterations in hair pigmentation, in comparison to those with marasmus, where oedema is less pronounced, and children tend to exhibit higher levels of activity and attentiveness. Chronic parasitic infections or other prolonged infectious diseases contribute to the development of hypoalbuminemia and subsequent growth impairment.[8] There were low ferritin levels reflecting iron deficiency anaemia in severe malnutrition. Children with SAM are known to have lower mean haemoglobin, haematocrit, and red cell indices.[9]

Following her initial stabilisation, ongoing care was discussed with the regional paediatric transfer team, subsequently leading to her transfer to a tertiary paediatric intensive care unit (PICU) for further management and monitoring.

Tertiary centre

The tertiary PICU continued with ventilation for a total of 162 hours and six days of inotropic support (noradrenaline). After extubation, she was eating and drinking with no issues. Additional blood tests in the PICU illustrated multiorgan failure that was hepatic, renal, and respiratory and was in disseminated intravascular coagulation (DIC). Therefore, she required two pools of platelets (initial platelets $48x10^9$/L), one of fresh frozen plasma, one of cryoprecipitate, and a further unit of red blood cells. The magnetic resonance imaging of her brain showed extensive microbleeds due to several conditions: thrombotic microangiopathy, disseminated intravascular coagulation,

and/or hypoxic brain injury, with a prognosis of doubtful reversibility. On further follow up, given the absence of cortical and grey matter involvement, the long-term progress is thought to be optimistic.

Refeeding syndrome (RFS) is a potentially fatal condition commonly characterised by rapid changes in fluid and electrolyte balance leading to problems of cardiac arrhythmias, and cardiac and respiratory failure.[10] She was treated with seven days of vitamin B substances and ascorbic acid (Pabrinex®), given that thiamine deficiency is common when there is a shift from fatty acid metabolism to carbohydrate metabolism, which increases the thiamine requirements.[11] She also received maintenance fluids, nasogastric Paedia-sure, acyclovir, ceftriaxone, clarithromycin, metronidazole, Movicol®, a proton pump inhibitor, one dose of dexamethasone post-extubation, ivermectin and dimethicone for the head lice infestation. Due to the RFS risks, she required several phosphate and potassium corrections. Given the widespread multiorgan involvement and poor health, an extensive battery of investigations involving all body systems was performed. Despite this, no unifying diagnosis could be detected; hence, her condition was explained by her chronic malnutrition, poor living conditions, inadequate care, and lack of social interaction. At discharge, the patient was in good general condition, withdrawn, not verbally communicating but engaging with staff, eating and drinking normally with improvement of her biochemical markers.

Discharge and follow-up

The patient was discharged to foster care after her hospital admission and has had regular follow-ups regarding various health issues. Given how underweight she was, her weight has been monitored closely (Table 3). Despite a varied diet and high-calorie content, she still struggles to put on weight, which is likely the result of chronic malnutrition.

At the last follow-up, the patient's weight increased by 6.7kg, which equates to roughly 3kg/year, and height growth of 10.0cm, which is roughly 5.2cm annually. She had a delayed bone age that was 8 years and 10 months when she was 10 years and 2 months old. She has ongoing learning difficulties and cognition difficulties (poor memory), for which she is receiving speech and art therapy. This is likely due to a constellation of reasons, such as malnutrition, emotional trauma, and neglect from early childhood. With good psychological support, there is evidence that development can occur when compared to no psychological support.[12]

Discussion

Extreme nutritional neglect can result in severe and potentially life-threatening complications. This case study highlights an imminent risk of death due to such a life-threatening form of child abuse, specifically extreme nutritional neglect. Cardiac arrest is a notable cause of death among severely wasted children, as evidenced by studies on children with eating disorders. The cardiac effects of severe malnutrition include bradycardia due to heightened vagal tone, hypotension resulting from peripheral muscle atrophy, prolonged corrected QT interval, reduced left ventricular mass, pericardial effusion, and complications associated with RFS. These cardiac complications can be reversible with timely and appropriate management.[13] The presence of a concomitant multiorgan failure in the studied girl is another nearly fatal consequence of severe malnutrition.

While elevated liver enzymes can be attributed to cardiac arrest, severe malnutrition may also contribute. Severe anaemia in children with severe acute malnutrition is typically microcytic hypochromic, although megaloblastic anaemia can occur due to vitamin B12 and folic acid deficiencies.[5] These children are also susceptible to clinical rickets, vitamin D deficiency,

hypocalcaemia, and bone disease.[14] Malnutrition impairs immune priming by dendritic cells and monocytes and reduces effector memory T cell function, increasing infection susceptibility.[15] Hypothermia is a significant mortality risk factor in children with SAM.[16]

Rapid refeeding of malnourished children can result in RFS, characterised by potentially life-threatening electrolyte imbalances such as hypophosphatemia, hypokalaemia, hypomagnesemia, and thiamine deficiency. Management involves initiating feeding with low-calorie fluids, gradually increasing caloric intake over several days, and closely monitoring electrolyte levels with prophylactic thiamine supplementation.[17] Although the girl in this case study demonstrated catch-up growth during follow-up, her neurodevelopmental prognosis remains guarded. SAM in childhood is associated with reduced intellectual capabilities, poor academic performance, attentional deficits, and impaired executive function.[18] Long-term educational support and special education may help bridge the resulting intellectual gap. Undernourished children and adolescents often experience delayed onset of secondary sexual characteristics and menarche compared to their well-nourished peers, potentially necessitating fertility support in adulthood.[19] Ophthalmologic complications such as infective conjunctivitis, xerophthalmia, and night blindness are also possible, with micronutrients and vitamin supplementation being essential for their reversal.[20]

Conclusions and Recommendations

Cardiac arrest resulting from extreme nutritional neglect in children is a rare occurrence in England. The case under study illustrates the devastating consequences of this severe form of abuse. The acute presentation underscores the ultimate clinical sequelae of severe acute malnutrition, a near miss, and inevitable physical and mental health repercussions.

Effective management of such critical cases necessitates a robust care plan to prevent recurrent severe acute presentations and align the child's growth and development with age-appropriate milestones. Integral to this approach is the collaboration between social services and a multidisciplinary team, including immediate family members, carers, schoolteachers, primary care providers, paediatric services, community nurses, dietitians, and child and adolescent mental health services. Such collaboration is crucial to ensure consistent and holistic growth and development.

Furthermore, consideration must be given to the long-term implications of early exposure to severe malnutrition as the child transitions through adolescence into adulthood. Proactive monitoring and support during these developmental stages are essential to mitigate the long-term physical and cognitive deficits associated with early severe malnutrition. This holistic and forward-thinking approach is vital to optimise outcomes and support the child's full potential for recovery and development.

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Table 1. Initial blood gas.

Variable	Result	Normal Parameters
pH	6.6	7.35-7.45
pCO_2	4.0	4.7-6.0 kPa
pO_2	10.3 (FiO_2 0.5)	10.5-14.0 kPa
HCO_3^-	3.1	22-26 mmol/L
BE	-34.9	-2 to +2 mmol/L
K^+	5.8	3.5-5.3 mmol/L
Lactate	20.9	0.5-2.2 mmol/L
Hb	Too low to quantify	112-145 g/L

Table 2. Initial blood results.

Variable	Result	Normal Parameters
LDH	7260	240-280 U/L
Magnesium	1.55	0.7-1.0 mmol/L
Phosphate	3.37	0.9-1.8 mmol/L
Sodium	131	133-146 mmol/L
Potassium	5.8	3.5-5.3 mmol/L
Urea	15.1	2.5-6.5 mmol/L
Creatinine	87	26-55 μmol/L

Bicarbonate	4	19-28 mmol/L
CRP	4	<5 mg/L
Total protein	44	60-80 g/L
Albumin	27	30-50 g/L
Globulin	17	20-35 g/L
Alkaline Phosphatase	60	60-425 U/L
Total bilirubin	17	0-21 μmol/L
ALT	2345	0.6-2.5 mmol/L
Plasma lactate	20.01	0.6-2.5 mmol/L
Amylase	24	28-100 U/L
Calcium	1.92	2.2-2.7 mmol/L
Adjusted calcium	2.22	2.2-2.7 mmol/L
Vitamin D	32	>50 nmol/L
Full Blood Count	Not available	
Ferritin	7	15-300 ng/ml
Haptoglobin	0.67	0.3-2.0 g/L
PT	35.3	10.3-13.3 seconds
INR	2.7	0.8-1.2
Derived Fibrinogen	1.98	2.0-5.3 g/L
APTT	27.6	25.7-35.3 seconds
APTT ratio	0.90	0.8-1.2

Table 3. Anthropometric measurements.

Age	Height (cm)	Centile	Weight (kg)	Centile
8 years	-	-	16.4	<0.4th
8 years 1 month	113	<0.4th	19.2	2nd
8 years 3 months	115	0.4th	19.9	2nd
8 years 7 months	116	0.4th	20.9	2nd
9 years 3 months	120	0.4th	21.6	0.4th
9 years 9 months	122	0.4th	22.4	0.4th
10 years 1 month	125	2nd	23.1	0.4th