RHABDOMYOLYSIS AND ACUTE RENAL FAILURE IN A CASE OF CHILD ABUSE

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The most common manifestations of child abuse are skin lesions, such as ecchymoses, abrasions, scars and burns.[1] Bone fractures, failure to thrive, subdural and retinal hemorrhage, genital injuries, subgaleal hematoma, unkempt appearance, frozen stares, human bite marks, visceral injuries, etc., are additional manifestations.[2] Munchausen syndrome by proxy is a rare presentation which has recently been well described in the literature.[3-5] Various unusual manifestations of child abuse, such as fetal pepper aspiration, thirsting and hypernatremic dehydration, intentional microwave burns, transection and pseudocyst of the pancreas, tin ear syndrome, subcutaneous fat necrosis, etc., have also been recognized recently.[6-8] These unusual symptoms deceive clinicians and prevent early diagnosis, thus endangering the life of the victim. There are very few reports in the world literature on the association of child abuse and acute renal failure induced by rhabdomyolysis.[6,9-11] To our knowledge, this is the first confirmed case of child abuse with acute renal failure due to rhabdomyolysis in Saudi Arabia.

Case Report

A five-year-old Saudi girl was admitted to the hospital with a history of skin rashes for 15 days, swelling of the buttocks and thighs for three days, and fever for two days. The child had twice been seen by a primary care physician who had prescribed antibiotics and antipyretics without any improvement. She was referred to the hospital from the primary care center as a case of idiopathic thrombocytopenic purpura.

The patient's past medical history was uneventful. Her mother died when she was two months old, and her father, a healthy 45-year-old office clerk, had remarried. Her maternal grandmother took care of her, while her two older full brothers lived with the father and stepmother. Her father had recently taken the child away from her grandmother to live with him. The child's stepmother was a 30-year-old housewife with a ten-month-old daughter, and the family was apparently living in harmony.

On presentation at the hospital, physical examination revealed a sick, pale-looking child with multiple ecchymoses over the back, buttocks, thighs and forehead. There were some scars on the dorsum of the fingers. Her growth parameters were, however, normal, and vital signs were stable. Her thighs and buttocks were swollen and tender. There was no jaundice, lymphadenopathy or hepatosplenomegaly. The joints and oral mucus membranes were normal. Other examinations, including funduscopy, were normal.

The initial laboratory results included the following: hemoglobin 74 g/L; white blood count 15.5×10^9/L with normal differential; platelets 161×10^9/L; serum sodium 129 mmol/L; potassium 6 mmol/L; chloride 96 mmol/L; calcium 1.7 mmol/L; phosphorus 1.05 mmol/L; magnesium 1.1 mmol/L; BUN 13.1 mmol/L; creatinine 150 μmol/L; alanine aminotransferase (ALT) 440 U/L; aspartate aminotransferase (AST) 1570 U/L; alkaline phosphatase (ALP) 130 U/L; lactate dehydrogenase (LDH) 4219 U/L; creatine kinase 2000 U/L; total serum bilirubin 28 μmol/L; total protein 61.4 g/L; albumin 32 g/L; serum ammonia 42 mmol/L; and uric acid 550 μmol/L. The color of the serum was clear. Prothrombin time, activated partial thromboplastin time, fibrinogen and fibrin degradation products were normal. Cerebrospinal fluid analysis was normal. Initial cultures of blood and cerebrospinal fluid showed no growth. Skeletal surveys did not show any recent or old fractures.

Upon admission, the patient received blood transfusion, fresh frozen plasma and antibiotics. She was found to be oliguric, and urinary catheterization was done. The first specimen of urine was dark brown in color. Dipstick test was positive for blood (4+), protein ++, specific gravity (l020), and microscopy did not show any RBC. Urinary electrolytes showed sodium of 67 mmol/L with fractional excretion of sodium more than 1%. Both kidneys were hyperechoic on ultrasound examination, with normal appearance of other organs. Over the next three days, the patient became more oliguric even though the urine color had become clearer. Urine output decreased to as low as 0.05 mL/kg/hr on the 4th day of admission. Urea and creatinine were rising and she became acidotic. Since the

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patient did not respond to conservative management of acute renal failure, peritoneal dialysis was started.

From the time of the patient's hospitalization, the hospital nurses noticed a lack of interest by the stepmother in the child's illness. The child showed fear of the stepmother and avoided eye contact with her. In a confidential interview, the stepmother admitted to battering the child with a broomstick in order to discipline her. She also admitted to accidentally inflicting hot iron burns to the child's fingers a few weeks previously. After five days of hospitalization, the child's maternal grandmother came to visit her and indicated that the child's father had forcibly taken the girl away from her two months earlier. She also indicated that the child's stepmother had been battering the child since her father brought her to stay with them. Her two elder brothers apparently seemed to have adjusted to the stepmother's behavior. During her stay at the hospital, the child developed *Pseudomonas* sepsis, peritonitis and pulmonary edema. She needed ionotropic support, specific antimicrobial therapy and ventilation. She soon recovered and was weaned off the ventilator. Her renal function improved, and dialysis was stopped after two weeks.

In the diuretic phase, the child had hypercalcemia and hypomagnesemia. The grandmother was attending the child during her remaining hospital days, and the child seemed very attached to her emotionally. She did not develop any new skin lesions, and the old ones healed. Her thigh and buttock swellings subsided, but a residual fluctuant swelling over the left buttock was drained of some 100 cc thin reddish serous fluid.

CT and isotope scans of kidneys, hemoglobin electrophoresis, hepatitis markers, and sickling tests were negative. Urine specimen was tested later on by spectrometry and was positive for myoglobin. Her renal profile, muscle enzymes like CPK, LDH, AST, and blood counts all became normal. Blood-free carnitines and acylcarnitines by tandem mass spectrometry and ischemic exercise tests were also normal.

Social workers were involved in further management of the case. After several sittings with the father, stepmother, maternal uncle and grandmother, it was agreed that the child should be put under the grandmother's care. A care order was obtained from the Riyadh Governor's Office and the child was discharged from the hospital to the care of the grandmother. On follow-up, the child was well, with no further clinical problems.

Discussion

Child abuse in our patient was suspected when initial laboratory results ruled out the possibility of bleeding disorders. The presence of extensive ecchymoses over the back and buttocks, skin lesions of different ages, different statements by the stepmother at different times, lack of interaction between the child and the stepmother, and the presence of burn scars over the dorsum of the fingers, were further evidence of child abuse. The stepmother also initially fabricated her relation with the child. Ultimately, her confession of maltreatment of the child confirmed the diagnosis of child abuse in our patient. Rhabdomyolysis and myoglobinuria as causes of acute renal failure in our patient were suspected by urine color, and positive dipstick test for blood which lacked red blood cells in urine microscopy. Our patient had low hemoglobin on admission. The possibility of hemoglobinuria was ruled out in the absence of other signs of hemolysis, such as reticulocytosis and clear color of serum. We believe the low hemoglobin in our patient on presentation was due to extensive subcutaneous and deep tissue bleeding without significant hemolysis as to cause hemoglobinuria and renal failure. Anemia was corrected with a single blood transfusion. Other supportive evidence of myoglobinuria in our patient included high levels of CPK, LDH, AST, uric acid, serum potassium and low serum calcium at presentation, and hypercalcemia at the diuretic phase. Sequestered fluid from muscle and necrosis of the muscle cells of the buttock resulted in a fluctuant swelling, which left a visible area of depression after aspiration and became normal on follow-up. However, confirmation of myoglobinuria came from spectrometric examination of urine later on. Rhabdomyolysis and myoglobinuria are rare causes of acute renal failure in children, and may be caused by severe muscle injury, as in crush syndrome, unaccustomed physical exercise, grand mal seizure and ischemia. They may also result from metabolic diseases, such as deficiency of muscle phosphorylase, phosphofructokinase, lactate dehydro-genase, carnitine palmityl transferase or myoglobino-pathy.[12,13] From a clinical perspective, all these possible causes seemed unlikely.

In recent years, several reports of child abuse have been published in the Saudi medical literature.[4,5,7,14-17] Most of these reports have been published from two tertiary care centers. The importance of epidemiological survey and early detection has been emphasized. Kattan and Al-Eissa have recently addressed this issue.[2,18] Unusual manifestations of child abuse demand reporting in order to increase the awareness of clinicians.[19] Rhabdomyolysis and myoglobinuria in child abuse have previously been reported in the German and American literature.[6,9-11] In all previous cases, diagnosis of myoglobinuria as a cause of renal failure had been delayed because of lack of experience with such a manifestation in child abuse. Some of the previous patients were diagnosed by contrast urography and by the finding of prolonged dense nephrogram.[9,10] Such investigations might aggravate the incidence of renal failure in cases of myoglobinuria. Diagnosis of myoglobinuria may be made under such circumstances by the findings of positive dipstick for blood in urine, absence or relatively few RBCs under microscopy, pigmented granular casts, and clear color of serum. Other

biomechanical evidence of rhabdomyolysis, such as high CPK, LDH, AST, uric acid, potassium, phosphorus with low calcium in early phase, and hypercalcemia in diuretic phase, are widely available. Myoglobin may be detected in urine by spectrometry or by radioimmunoassay.[12,13] Early diagnosis and aggressive management of myoglobinuria by volume replacement and forced alkaline diuresis may reverse the renal failure.[12,13]

The clinical implication of this report is that dark urine in a battered child should raise the suspicion of myoglobinuria. In such a case, laboratory and simple bedside tests for diagnosis of myoglobinuria should be done immediately, and appropriate supportive medical therapy should be instituted.

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