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Vrsalović, R., Tešović, G., Miše, B. (2007) *Rhabdomyolysis and acute renal failure in a child with parainfluenza type 1 infection.* Pediatric Nephrology, 22 (9). pp. 1369-1371.

The original publication is available at www.springelink.com

http://www.springerlink.com/content/x3ju2486250tpk62/

http://dx.doi.org/10.1007/s00467-007-0486-2

Rhabdomyolysis and acute renal failure in a child with parainfluenza type 1 infection

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ABSTRACT

We present a rare case of parainfluenza type 1 virus induced rhabdomyolysis, complicated by acute renal failure (ARF). The child underwent continuous venovenous haemofiltration and has shown full clinical and biochemical recovery. ARF in child due to rhabdomyolysis in parainfluenza type 1 infection has to the best of our knowledge not been previously reported.

Keywords: Rhabdomyolysis, Parainfluenza type 1 virus, Acute renal failure, Children

INTRODUCTION

Rhabdomyolysis is a potentially life-threatening syndrome resulting from the breakdown of skeletal muscle fibers with leakage of muscle contents into the circulation.[1] The most common causes of rhabdomyolysis in children are viral myositis, trauma, and connective tissue disease.[2] ARF in child due to rhabdomyolysis in parainfluenza type 1 infection has to the best of our knowledge not been reported previously. We report a case of ARF following rhabdomyolysis due to evident parainfluenza type 1 virus infection in a 5-year old boy.

CASE REPORT

Our patient is 5-year-old boy with psychomotor retardation and spastic quadriplegia who 2 days before admission developed rhinorrhea, cough and vomiting. One day before admission he developed chills and fever up to 40.5 °C. There was no family history of renal or musculoskeletal disease nor had he experienced any trauma, drug abuse or a recent increase in his exercise level. His past medical history was remarkable for cerebral palsy due to perinatal asphyxia.

At the time of admission the patient was semicomatose, dehydrated and dyspneic. He was febrile 40.0 °C, with a heart rate of 180 beats/minute, a respiratory rate of 40 breaths/minute and a blood pressure of 135/80 mmHg. Crackles were audible throughout both lung fields, but heart sounds were normal. Meningeal signs were negative.

The initial laboratory studies revealed: erythrocyte sedimentation rate 33 mm/1st h; C-reactive protein 73 mg/l; white blood cell count 17.9 × 10^9/l with 83% neutrophils, 11% lymphocytes and 6% monocytes; hemoglobin 75 g/l; hematocrit 0.24; platelets 511× 10^9/l; blood urea nitrogen (BUN) 6.1 mmol/l; creatinine 39 μmol/l and normal electrolytes. D-dimer was 0.2 mg/L with prothrombin time of 16.4 s and partial thromboplastin time of 28.8 s. Arterial blood pH at admission was 7.342, base excess –6.9 mmol/L, $PaCO_2$ 34.7 mmHg, PaO_2 57.3 mmHg, O_2 saturation 86.9%. Serum bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, lactate dehydrogenase, γ-glutamyl transpeptidase were normal. Urine analysis revealed protein 2+, ketone 1+, microscopic examination of the sediment revealed 5 white blood cells per high-power field but no red blood cells.

The patient's cerebrospinal fluid (CSF) was clear, with white blood cells 11/3 mm^3, protein 164 g/l and glucose 3.4 mmol/l. No organisms were isolated from blood and CSF cultures. Chest radiography revealed bilateral diffuse infiltration.

A nasopharyngeal swab specimen obtained on the first hospital day indicates sensitive *Streptococcus pneumoniae*. Parainfluenza type 1 virus was identified from nasopharyngeal aspirate sample taken on 1st hospital day by direct immunofluorescent antibody assay (DFA).

During the first 12 hours of hospital stay the patient condition was stable. He was febrile around 40 °C and tachydyspnoeic but well oxygenated following oxygenotherapy.

The next day the patient condition deteriorated; he became extremely tachydyspnoeic (80-90 breaths/minute) with a heart rate of 200–216 beats/minute. Despite the oxygenotherapy, hypoxia developed and mechanical ventilation was initiated because of respiratory failure. On the third hospital day arterial hypotension unresponsive to crystalloid infusions occurred and the continuous dopamine infusion (10 μg/kg/minute) was started. During the next day, he had progressive decline of renal function with a peak creatinine of 214 μmol/l, blood urea nitrogen of 33 mmol/l, and hyperkalaemia of 6.8 mmol/l. Relevant laboratory values included a creatine phosphokinase (CPK) level of 22,242 IU/l, aspartate aminotransferase 1,040 IU/l, alanine aminotransferase 664 IU/l, and lactate dehydrogenase 2,995 IU/l, consistent with skeletal muscle necrosis. Metabolic acidosis was registered for the first time with arterial

blood pH 7,169, base excess -16,6 mmol/L and bicarbonates concentration of 12,2 mmol/L. Urinary pH was 5,5.
Despite adequate hydration with normal saline and urine alkalization with sodium bicarbonate the patient developed oliguric acute renal failure requiring temporary continuous venovenous hemodiafiltration (CVVHD) three days after admission. The patient was oliguric until day 17 of hospitalization. Hemodialysis was stopped on day 18. His condition and renal function continuously improved and he required no further dialysis. CPK levels decreased until normalization on day 20. The patient's respiratory status gradually improved, and 31 days after admission he was successfully weaned and extubated. The hospitalization course was further complicated by acquisition of posttransfuse CMV infection confirmed by CMV DNA PCR. The patient was discharged on day 50 with a serum creatinine of 38 μmol/l and BUN of 4.0 mmol/l.
A wide variety of infections which could have caused the illness similar to that in our patient were considered at the outset and sought diagnostically, including bacterial sepsis, mycoplasma infection, legionellosis, influenza A or B, respiratory syncytial virus, adenovirus and parainfluenza type 1, 2 or 3 virus, but an exhaustive workup excluded all with exception of parainfluenza type 1 virus.
Three months after the discharge from the hospital, the child was readmitted because of bilateral bronchopneumonia (nontypable *Haemophilus influenzae* was isolated from the nasopharyngeal aspirate). Although he was hyperpiretic for the three days, his CPK level as well as urine output remained within normal ranges.
There has been no recurrence of rhabdomyolysis during the 11 months follow up.

DISCUSSION

The term rhabdomyolysis refers to disintegration of striated muscle, which results in the release of muscular cell constituents into the extracellular fluid and the circulation.[3] Rhabdomyolysis can be induced by numerous factors including crush injury, skeletal muscle overuse, heat, alcohol abuse, myopathies, drugs, toxins and metabolic derangements (such as hypokalemia, hyponatremia or hypernatremia, and hypophosphatemia), as well as several types of viral and bacterial infections.[4] Viral myositis cause more than a third of all rhabdomyolysis cases among pediatric patients, especially in the first decade of life.2 Viral associated rhabdomyolysis follows infections caused by influenza virus (forty-two percent of cases of viral mediated rhabdomyolysis),[5] Coxsackie virus, enterovirus, human immunodeficiency virus, Epstein-Barr virus,[6] varicella-zoster virus[7] and cytomegalovirus.[8] To date, three cases of parainfluenza virus induced rhabdomyolysis infection have been described in the literature, none with parainfluenza type 1.[9, 10, 11] In children rhabdomyolysis is usually a mild event causing only elevation of muscle enzymes in asymptomatic patients.2 However, in some patients, complications occur. Complications of rhabdomyolysis are classified as early or late. Early complications include severe hyperkalemia that causes cardiac arrhythmia and arrest.4 The most serious late complication is ARF, which occurs in approximately 5 percent of pediatric patients with this syndrome.2 The precise mechanism by which rhabdomyolysis causes renal failure remain unclear. The potential mechanisms may be renal vasoconstriction/hypoperfusion, renal tubular obstruction due to cast formation and myoglobin mediated tubular cytotoxicity.[1, 2, 4] The contributing factors for the development of ARF in pediatric patients with rhabdomyolysis beside the severity of muscle damage are presence of dehydration, metabolic acidosis or presence of multiple organ dysfunction syndrome (MODS).1 In patient that we present here, the development of ARF coincides with MODS occurring during the early hospital stay. According to Proulx criteria, our patient had primary MODS defined by dysfunction of cardiovascular and respiratory system as well as

ARF.[12] Due to our opinion the main, or maybe the only cause of MODS in our patient was severe rhabdomyolysis caused by parainfluenza type 1 virus. Although, hyperpyrexia on the beginning of symptoms of viral disease could not be fully excluded as a contributor, we conclude that its influence was not crucial. Namely, three months after present disease the child experienced bilateral bronchopneumonia with hyperpyrexia as a symptom, but without signs of rhabdomyolysis and with normal urine output. Immobilization during actual disease is also less possible cause of rhabdomyolysis in our patient because of fact that he is due to his chronic condition for the majority of his life bedridden.

Finally we can conclude that this is the first young child to be reported with parainfluenza type 1 virus induced rhabdomyolysis and development of ARF requiring continuous venovenous haemofiltration. Presence of MODS caused by rhabdomyolysis is probable the main (or only) factor that contributes to the development of ARF.

In conclusion, parainfluenza type 1 virus infection must be considered as a possible cause of viral rhabdomyolysis and ARF in pediatric patient.

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