

Case Report

Severe Rhabdomyolysis Secondary to Fulminant Meningococemia: Case Report

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Abbreviations

CK: Creatine Kinase

Introduction

Rhabdomyolysis is a disorder characterized by acute damage of the sarcolemma of the skeletal muscle leading to release of potentially toxic muscle cell components into the circulation, which can then result in hypovolemia, acidosis, hyperkalemia, acute renal failure and disseminated intravascular coagulation. The most common causes of rhabdomyolysis in paediatric patients are viral myositis, trauma, connective tissue disorders, exercise, and drug overdose [1]. An increased plasma concentration of Creatine Kinase (CK) or myoglobin and the presence of myoglobinuria are useful parameters in the diagnosis of this disease. Serum CK at a level 5 times higher than the normal value usually confirms rhabdomyolysis. It ranges in severity from an asymptomatic elevation of CK levels in the blood, to severe life-threatening cases of myoglobinuria and acute renal failure, which are associated with very high CK levels. Fulminant meningococemia is a relatively rare life-threatening disease caused by *Neisseria meningitidis*, and is perhaps the most rapidly lethal form of septic shock in humans. Rhabdomyolysis secondary to meningococcal infection has rarely been investigated, and there are very few case reports [2-4]. The case is here reported of fulminant meningococemia in a 2-year old child due to a non-groupable strain of *Neisseria meningitidis*-associated severe rhabdomyolysis.

Case Presentation

A previously healthy girl aged 2 years and 8 months was brought to the Emergency Department with complaints of fever and vomiting which had been ongoing for 1 day, and a rash that had been evolving on her face and limbs for 4 hours. On presentation, temperature was 38.4 °C, heart rate 188/minute, and blood pressure 70/50 mmHg. There were numerous ecchymotic lesions on the face, chest, back, abdomen, arms, and legs. Meningeal signs were absent. There were no other findings of significance. The child had not been immunised against any meningococcal infection. Investigations on admission revealed a platelet count of 27,000/µL and an international

Abstract

Fulminant meningococemia is a relatively rare life-threatening disease caused by *Neisseria meningitidis*. Rhabdomyolysis secondary to meningococcal infection has only rarely been investigated. We report a case of severe rhabdomyolysis secondary to fulminant meningococemia in a 2 years and 8 months old girl. She had a serum creatinine kinase of 84000 U/L. The isolated strain of *N. Meningitidis* in our patient was not belonged to one of the most frequent five serotype (A,B,C,Y and W-135).

Keywords: Nongrupable; *Neisseria meningitidis*; Rhabdomyolysis; Child

normalized ratio of 3.1, creatinine 1.2 mg/dL (0.4-1.4), alanine transaminase 109 U/L (0-35), aspartate transaminase 267 (8-46), CK 7429 U/L (35-195) and C-reactive protein 96.9 mg/L (0-5). No lumbar puncture was applied to the patient who had thrombocytopenia and hypotension. On deterioration of the breathing pattern, the patient was transferred to the pediatric intensive care unit and intubated. The initial treatment included many units of blood and blood products, and intravenous ceftriaxone in consideration of the local sensitivity patterns. The patient became severely hypotensive and was initially stabilized with volume resuscitation and dopamine infusion. Adrenaline infusion was started (up to 0.8 mcg/kg/min) when the blood pressure continued to be low despite the dopamine infusion. Hydrocortisone was administered for a possible adrenal insufficiency. Therapeutic plasma exchange was performed daily for 6 days. The creatine kinase reached a peak value of 84000 U/L on day 2 of admission, with a gradual decline over the next few days, reaching a normal level by day 7 of admission. Urine dipstick showed presence of blood. Urine myoglobin was not performed. Fluid and bicarbonate intravenous administration was applied. Throughout the period of hospitalisation, daily urine output and renal function test were within the normal range from slightly raised creatinin (maximum 1.25 mg/dL) which settled with adequate intravenous hydration. Adrenaline and dopamine infusions were tapered and stopped by day 3 of admission. Although the patient was hemodynamically stable, on examination she was comatose with absent brain stem reflexes and no motor responses to painful stimuli. The patient was declared brain dead after completion of testing on day 10 of admission. *N. meningitidis* was detected in the serum and although polymerase chain reaction specific for 5 serogroups: (Men) A,B,C,Y and W-135 was used to genogroup this strain, negative results were obtained.

Discussion

The pathogenesis of rhabdomyolysis secondary to meningococemia is not yet fully understood. However, direct bacterial invasion into muscle, bacterial toxin, metabolic derangements, rigors, fever and non-specific sepsis-related mechanisms may be precipitating factors that explain the muscle injury [5]. In the current case, there was no other contributing history to suggest

other causes of rhabdomyolysis; there was no history of trauma or recent seizures and the patient was not on any known medication that could potentially cause rhabdomyolysis. It has been reported that about 33% of patients with rhabdomyolysis develop acute renal failure [6]. However, it is also recognized that myoglobinuria itself does not cause acute renal failure, and the renal dysfunction seems to result from a combination of hypovolemia, hypoxia, endotoxin- or endotoxin-like substances with other events. Once the diagnosis of rhabdomyolysis is made, adequate hydration and alkalization of the urine are necessary in order to prevent renal failure. In the current patient, adequate hydration, alkalization and therapeutic plasma exchange proved effective in slowing the process of rhabdomyolysis.

There are 12 recognized serogroups of *N. Meningitidis*, but the vast majority of invasive disease is related to six meningococcal serogroups: (Men) A, B, C, W-135, X and Y [7]. The most prevalent serotypes of the causative agent *Neisseria meningitidis* in Turkey are serotype W-135 and B [8]. The isolated strain of *N. Meningitidis* in the current patient did not belong to any of the 5 most frequent serotypes: (Men) A,B,C,Y and W-135.

Conclusion

To the best of our knowledge, previous case reports of rhabdomyolysis associated with meningococemia have been of 1 child and 2 adult patients [2-4]. The above case highlights the presence of severe rhabdomyolysis in the case of fulminant meningococemia secondary to non-groupable *Neisseria meningitidis* infection in a previously healthy child.

References

1. Khan FY. Rhabdomyolysis: a review of the literature. *Neth J Med.* 2009; 67: 272-283.
2. Chamberlain MC. Rhabdomyolysis in children: a 3-year retrospective study. *Pediatr Neurol.* 1991; 7: 226-228.
3. Morano Amado LE, Vazquez Alvarez OM, Brapas Fernandez F, Sesma Sanchez P. Rhabdomyolysis associated with *Neisseria meningitidis* infection. *An Med Interna.* 1990; 7: 546-547.
4. Vishal S, Mukul AP, Gri S. Severe rhabdomyolysis associated in meningococemia. *J Glog Infect Dis.* 2010; 2: 315.
5. Bandi S, Chikermane A. Rhabdomyolysis in a child secondary to *Staphylococcus aureus* endocarditis. *J Global Infect Dis.* 2009; 1: 146-148.
6. Gabow PA, Kaehny WD, Kelleher SP. The spectrum of rhabdomyolysis. *Medicine.* 1982; 61: 141-152.
7. Stephens DS. Conquering the meningococcus. *FEMS Microbiol Rev.* 2007; 31: 3-14.
8. Ceyhan M, Gürler N, Ozsurekci Y, Keser M, Aycan AE, Gurbuz V, et al. Meningitis caused by *Neisseria Meningitidis*, *Hemophilus Influenzae* Type B and *StreptococcusPneumoniae* during 2005-2012 in Turkey. A multicenter prospective surveillance study. *Hum Vaccin Immunother.* 2014; 10: 2706-2712.