

## Influenza-B Associated Rhabdomyolysis and Acute Renal Failure

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We present a 15-year-old boy who developed severe rhabdomyolysis and acute renal failure following influenza B infection. His renal function was restored after appropriate therapy for rhabdomyolysis. Although rapidly progressive pneumonia, respiratory failure, and acute respiratory distress syndrome are the most common severe complications of influenza B infection, clinicians should be aware that influenza B may be complicated with rhabdomyolysis and acute renal failure in children.

**Key words:** *Rhabdomyolysis, Renal failure, Influenza B.*

Influenza B often present with myalgia, rhabdomyolysis is rarely seen. The clinical presentation of rhabdomyolysis varies from an asymptomatic increase in creatine kinase (CK) to severe ARF and hypovolaemic shock. Few reports are available on association of rhabdomyolysis and acute renal failure (ARF) with influenza virus type B infection in children [1-5]. We present the case of a child with cerebral palsy whose renal dysfunction, caused by influenza-associated rhabdomyolysis, was restored after adequate treatment.

### CASE REPORT

A 15-year-old boy, diagnosed with cerebral palsy, was in bedridden status. He had been admitted to the hospital many times for airway infection. Four days prior to presentation, he developed sudden onset of fever, decreased activity, productive cough and yellow sputum. On examination, his weight was 31 kg. The vital signs were stable. Coarse crackles were heard over bilateral lower lung fields. Laboratory investigations revealed TLC  $7,600/\text{mm}^3$  ( $P_{61}L_{31}M_6E_2$ ); serum urea nitrogen (BUN), 16 mg/dL, serum creatinine, 0.9 mg/dL; aspartate aminotransferase (AST), 33 U/L; (normal, 5-35 U/L); and alanine transaminase (ALT) 16 IU/L (normal, <40 U/L). The rapid screen test for acute influenza infection was positive for influenza B, but negative for influenza A. Chest radiography showed no active lung lesion. The electrocardiogram was normal, and blood cultures, sputum cultures, urine cultures and viral throat cultures were all negative for microbial growth. After admission, oseltamivir 60 mg bid and ampicillin and sulbactam 1.5g every 6 hours were prescribed. On day 3, chest radiography revealed an

increased pneumonitis patch on right upper lung.

Shortness of breath, and dark urine with oliguria were noted on the 5<sup>th</sup> day. Laboratory test results revealed serum CK 407,421 IU/L (normal, 27-168 U/L), CK-MB 827 IU/L (normal, <16 U/L); creatinine:1.47 mg/dL; AST/ALT 3060/744 IU/L, LDH 23,880 IU/L (normal, 135-147 U/L); sodium 157 meq/L, potassium 5.1 meq/L, calcium 6.6 meq/L; uric acid 13.8 mg/dL (normal, 2.5-7.2 mg/dL). Clinical presentation and laboratory findings were suggestive of rhabdomyolysis with acute renal failure, most probably caused by influenza B infection. The patient was transferred to intensive care unit. Due to altered consciousness, he received endotracheal intubation and mechanical ventilation. The systolic blood pressure decreased to 50-60 mmHg and poor cardiac contractility was detected. Standard management was instituted for shock including inotropic agents. Metabolic acidosis was treated by administration of sodium bicarbonate.

On hospital day 6, the blood pressure and cardiac contractility recovered to normal. However, the child developed disseminated intravascular coagulation, and had hematuria and bleeding from the gastrointestinal tract. Renal functions deteriorated and the child was started on hemodialysis. The oseltamivir treatment was shifted to peramivir because of poor GI absorption. Peramivir was discontinued 2 days later due to increasing liver enzymes, and subsequently fever subsided. After hemodialysis therapy, his renal function was improved gradually. The serum levels of the muscle enzymes CK and AST decreased rapidly to 2711 IU/L and 168 IU/L, respectively, at discharge 27 days after admission.

One week after discharge, the patient visited our outpatient department for follow-up; the serum CK level, renal and liver function had returned to normal.

## DISCUSSION

Rhabdomyolysis is defined as a clinical and laboratory syndrome resulting from skeletal muscle breakdown with leakage of muscle cell contents into the systemic circulation. It is characterized by an elevated serum creatine kinase level and myoglobinuria, and may lead to renal dysfunction [2]. Rhabdomyolysis can cause life-threatening complications, including hypovolemia, hyperkalemia, metabolic acidosis, acute renal failure (ARF) and DIC [3]. ARF often results from the nephrotoxic effects of lytic myocyte components and usually presents as oligouric pigment-induced intrinsic renal failure [4]. The early and aggressive fluid repletion and bicarbonate therapy are the standard treatment to prevent ARF in such cases.

Influenza B-associated rhabdomyolysis is an infrequent and little-known complication of influenza B virus infection in children. In 2010, Wu, *et al.* [5] reviewed hospitalized children with influenza B virus infection at a university children's hospital in North Taiwan during 2000–2007 and found that 24 had presented with rhabdomyolysis; none had renal involvement. A recent review suggests that the risk of acute kidney injury in rhabdomyolysis is usually low when CK level at admission is <15,000 to 20,000 IU/L [3]. Our patient had high level CK 407,421 IU/L. Because limited data indicate that administering

oseltamivir via a gastric tube can provide systemic absorption in critically ill patients [6], our patient was treated with intravenous peramivir. It is a neuraminidase inhibitor, authorized for emergency use for the treatment of hospitalized patients with known or suspected 2009 H1N1 influenza. Clinicians should be alert to patients with flu-like symptoms with severe muscle pain and dark brown urine (to rule out rhabdomyolysis). The high level of CK could be an indicator for the fatal complication of ARF. The early diagnosis and appropriate therapy should decrease mortality and restore renal function.

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## Naxos Disease and Carvajal Variant

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An 11-yr-old girl, born out of a consanguineous marriage presented with recurrent exertional syncope due to ventricular tachycardia. She had woolly hair, palmoplantar hyperkeratosis and mild cardiomegaly. Echocardiogram revealed mild left ventricular dysfunction. Features were consistent with Carvajal variant of Naxos disease, an arrhythmogenic cardiomyopathy with autosomal recessive inheritance.

**Key words:** *Cardiomyopathy, Palmoplantar keratoderma, Ventricular tachycardia, Woolly Hair.*

**N**axos disease is a recessive form of arrhythmogenic right ventricular dysplasia/cardiomyopathy associated with a cutaneous phenotype characterized by

palmoplantar keratosis and woolly hair. It is caused by mutations of the genes encoding desmosomal proteins [1]. Cardiac disease has 100% penetrance by adolescence, manifested as symptomatic arrhythmias,