CASE REPORT

A case of H3N2 complicated by acute kidney injury secondary to rhabdomyolysis

Sriram Gonakoti, Janak Bahirwani, Raja Naga Mahesh Maddala, Sudha Vidyasagar

Department of Medicine,
Kasturba Medical College,
Manipal University, Manipal,
Karnataka, India

Correspondence to
Sriram Gonakoti,
gonakoti@gmail.com

Accepted 7 April 2018

SUMMARY

H3N2 was first detected in July 2011 in the USA. It is responsible for sporadic cases of influenza and localised outbreaks and has not yet taken over on an epidemic or pandemic scale. An 84-year-old man presented with a dry cough, fever and myalgia for 3 days. On examination, he had a pulse of 98 bpm and blood pressure of 124/88 mm Hg. The patient was tachypnoeic, SpO₂ was 90%. Auscultation revealed bilateral diffuse wheeze and crackles. He had generalised muscle tenderness on examination. On admission, creatinine was 1.9 mg/dL and CK(creatine kinase) was 44 000 U/L. Chest X-ray was suggestive of ARDS (acute respiratory distress syndrome). Throat swab was positive for H3N2. The patient was given intravenous fluids, oral sodium bicarbonate, oxygen and oseltamivir tablet. In view of ARDS, he was given intravenous methylprednisolone and bronchodilators for bronchospasm. The patient improved symptomatically; vitals and lab reports were normal at the time of discharge.

BACKGROUND

This is an atypical presentation of an uncommon disease and this case report highlights the basic outline of management.

CASE PRESENTATION

An 84-year-old man presented to the emergency triage with a dry cough, fever and myalgia for 3 days. There was no history of expectoration or breathlessness or wheeze and no history of recent seizure-like activity, physical exertion or prolonged immobilisation. He was known to have type 2 diabetes and was taking mixtard and glargine injections, known case of seizure disorder on levetiracetam tablet 500 mg twice daily. On examination, he had a regular pulse of 98 bpm, blood pressure of 124/88 mm Hg and was afebrile. He was tachypnoeic and SpO₂ (peripheral oxygen saturation) was 90% on 2 L of oxygen. JVP (Jugular Venous Pressure) was not elevated. Respiratory system examination revealed normal inspection and palpation findings. On auscultation, the patient was found to have bilateral diffuse wheeze and crackles. He had generalised muscle tenderness on examination. The remainder of the systemic examination was normal. A clinical diagnosis of acute bronchitis with myositis was made, and the patient was admitted to the inpatient service.

INVESTIGATIONS

Investigations on admission revealed elevated leucocyte counts ($11.1 \times 10^9/\mu\text{L}$) with a mild left shift. The renal function tests revealed a high serum creatinine (1.9 mg/dL) and high blood urea (57 mg/dL) suggestive of acute kidney injury. The liver function tests revealed elevated liver enzymes (AST [aspartate transaminase] 666 IU/L, ALT [alanine transaminase] 150 IU/L and ALP [alkaline phosphatase] 56 IU/L). Serum uric acid was raised (9.9 mg/dL). Creatine kinase was elevated (44 000 U/L). A urine dipstick was positive for blood. Urine microscopy revealed granular casts and was negative for blood. Urine myoglobin was negative. Blood and urine cultures were sent and were negative. A throat swab was positive for H3N2. A 12-lead ECG (electrocardiogram) on hospital admission showed normal sinus rhythm. ABG (arterial blood gas) done on room air (FiO₂ of 0.21 [fraction of inspired oxygen]), revealed a PaO₂ of 53 mm Hg, and consequently, a PaO₂/FiO₂ ratio of 250 mmHg. A chest X-ray PA (posteroanterior) view revealed bilateral fluffy shadows suggestive of ARDS (acute respiratory distress syndrome) (figure 1). A final diagnosis of H3N2 influenza with AKI (acute kidney injury) secondary to rhabdomyolysis and ARDS, was made.

TREATMENT

The patient was given tablet oseltamivir 75 mg twice daily for 5 days with tablet azithromycin 500 mg twice daily for 5 days. Intravenous methylprednisolone was started in view of ARDS. Nebulisation was with salbutamol and ipratropium bromide every sixth hour for severe bronchospasm along with O₂ via nasal prongs 2 L/min. Acute kidney injury secondary to rhabdomyolysis was treated with intravenous fluids and oral sodium bicarbonate. He was switched over from intravenous methylprednisolone¹ after 5 days to a tapering regimen of oral prednisolone. He was gradually weaned off oxygen.

OUTCOME AND FOLLOW-UP

The patient improved symptomatically; vitals and lab reports were normal at the time of discharge. However, the patient was lost to follow-up.

DISCUSSION

Rhabdomyolysis, as the name suggests, is characterised by the breakdown of striated muscle fibres in the body and release of myoglobin into the blood. Trauma is the most common cause of

To cite: Gonakoti S, Bahirwani J, Maddala RNM, et al. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2018-224334

Figure 1 Chest X-ray PA view on admission showing bilateral hazy lung fields suggestive of acute respiratory distress syndrome.

rhabdomyolysis. Though, other conditions² such as epilepsy, exertion, prolonged immobilisation, hyperthermia, drugs, metabolic syndromes like carnitine deficiency, infection and autoimmune conditions are established causes as well. Bacteria, virus and parasites have been documented to trigger the breakdown of muscle. The pathogenesis behind rhabdomyolysis in influenza is not fully understood. Two proposed theories include direct viral invasion of muscle tissue and the generation of cytokines, which are toxic to the muscle fibres in response to the virus.³ However, there have been reports of exertional rhabdomyolysis occurring post-treatment and recovery from H3N2 infection.⁴

H3N2 influenza virus is a variant of the H1N1 influenza virus. It was first detected in July 2011 in the USA. It is responsible for sporadic cases of influenza and localised outbreaks and has not yet taken over on an epidemic or pandemic scale. USA has reported only 423 such cases which were responsible for 25 hospital admissions and 1 death⁵

Complications of influenza include ARDS, secondary bacterial pneumonia, acute kidney injury, rhabdomyolysis, encephalopathy, aseptic meningitis, myocarditis. Our patient was in the high-risk group for influenza complications. Risk factors in our patient included older age (>65 years) and diabetes.⁶

Patients develop muscle weakness and myalgia involving the lower limb and back muscles or generalised myalgia. Patients may also present with a darkening of the colour of urine, decreased urine output, fever, malaise. Rarely, complications such as compartment syndrome⁷ and disseminated intravascular coagulation, can be the presenting symptoms of rhabdomyolysis.⁸

A clinical diagnosis of rhabdomyolysis can be supplemented by laboratory confirmation. A urine analysis will reveal dark acidic urine, with brown granular casts suggestive of acute tubular necrosis and elevated urine sodium concentration. A positive urine myoglobin and absence of red blood cells despite a positive urine blood dipstick is highly suggestive of rhabdomyolysis. Serum myoglobin will be elevated. Muscle enzymes such as creatine kinase and aldolase B will be elevated. Potassium will be elevated (>5.5 mg/dL) and can lead to life-threatening arrhythmias. Creatinine and BUN (blood urea nitrogen) will be elevated with a BUN: creatinine ratio less than 20, suggestive of acute tubular necrosis. However, in rare instances, such as in the case of rhabdomyolysis associated with metabolic diseases, BUN and creatinine can remain normal without evidence of acute kidney injury.⁹

The basic outline of management¹⁰ includes intravenous fluid resuscitation, urinary alkalinisation with sodium bicarbonate and prevention and management of complications.

Intravenous fluid infusion should be titrated to maintain a urine output of 200–300 mL/kg body weight. Sodium bicarbonate reduces the cast formation within the renal tubules and also neutralises the toxic effects of the muscle pigment, thereby preventing the development of acute kidney injury.¹¹ Monitoring of CK levels, urine output, serum creatinine and electrolyte are vital during treatment. Dialysis is necessary if the patient develops life-threatening hyperkalaemia, metabolic acidosis, anuria, pulmonary oedema and uraemia, that is refractory to treatment.

Learning points

- ▶ Rhabdomyolysis can be diagnosed by the presence of blood on a urine dipstick and the absence of red blood corpuscles on microscopic examination of urine, elevated CK levels, serum creatinine levels, and urine myoglobin. A muscle biopsy is not required for a diagnosis.
- ▶ One must treat rhabdomyolysis at the earliest while simultaneously trying to uncover its aetiology.
- ▶ Patients with H3N2, especially those in the at-risk group, must be monitored closely for complications.
- ▶ In view of the presenting case, a possibility of rhabdomyolysis must be considered in any patient presenting with myalgia and underlying evidence of infection.

Contributors Preliminary manuscript: SG and JB; revision of the manuscript. SV, SG, RNMM.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2018. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

REFERENCES

- 1 Marik PE, Pastores SM, Annane D, et al. Recommendations for the diagnosis and management of corticosteroid insufficiency in critically ill adult patients: consensus statements from an international task force by the American College of Critical Care Medicine. *Crit Care Med* 2008;36:1937–49.
- 2 Huerta-Alardin AL, Varon J, Marik PE. Bench-to-bedside review: Rhabdomyolysis -- an overview for clinicians. *Crit Care* 2005;9:158–69.
- 3 Fodili F, van Bommel EF. Severe rhabdomyolysis and acute renal failure following recent Coxsackie B virus infection. *Neth J Med* 2003;61:177–9.
- 4 Sevketoglu E, Kural B, Beskardes AE, et al. Exertional rhabdomyolysis after influenza A (H3N2) infection in a basketball player boy. *Ann Trop Paediatr* 2011;31:93–6.
- 5 Centre for Disease Control and Prevention. Case Count: Detected U.S. Human Infections with H3N2v by State since August 2011. 2011 <http://www.cdc.gov/flu/swineflu/h3n2v-case-count.htm> (accessed 6 Nov 2017).
- 6 Centre for Disease Control and Prevention, MMWR Recomm Rep. *Prevention and control of seasonal influenza with vaccines. Recommendations of the Advisory Committee on Immunization Practices-United States, 2013-2014*. 62, 2013:906.
- 7 Bhalla MC, Dick-Perez R. Exercise induced rhabdomyolysis with compartment syndrome and renal failure. *Case Rep Emerg Med* 2014;2014:1–3.
- 8 Hollenstein U, Thalhammer F, Burgmann H. Disseminated intravascular coagulation (DIC) and rhabdomyolysis in fulminant varicella infection--case report and review of the literature. *Infection* 1998;26:306–8.
- 9 Elmer P, Berry AC, Lenchus JD, et al. Exertional Rhabdomyolysis: A Case of Markedly Elevated Creatine Kinase Without Acute Kidney Injury. *Journal of Medical Cases* 2014;5:483–5.

10 Shapiro M, Sommer C. Rhabdomyolysis. BMJ Best Practice. 2017 <http://bestpractice.bmj.com/topics/en-gb/167> (accessed Feb 2018).

11 Malik G R. Rhabdomyolysis and Myoglobin-induced Acute Renal Failure, Saudi J. Kidney Dis. Transplant. 9. *Kidney Dis. Transplant* 1998;9:273–84.

Copyright 2018 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow