Pediatr Nephrol (2011) 26:153–154
DOI 10.1007/s00467-010-1633-8

LETTER TO THE EDITORS

Acute glomerulonephritis and acute kidney injury associated with 2009 influenza A:H1N1 in an infant

Juan C. Kupferman · Howard Trachtman · Eric D. Spitzer

Received: 20 July 2010 / Revised: 22 July 2010 / Accepted: 23 July 2010 / Published online: 14 August 2010

Sirs,

We read with great interest the letter by Jain et al. regarding a 14-year-old boy with postinfectious glomerulonephritis following infection with influenza A:H1N1 [1]. We present a case of acute glomerulonephritis (AGN) and acute kidney injury (AKI) associated with 2009 A:H1N1 infection in an infant.

A 5-month-old girl presented to the Emergency Department (ED) with a 2-day history of vomiting, diarrhea, and swelling of the face. There was no cough, fever, respiratory distress, or gross hematuria. She was voiding well. Blood pressure (BP) was 117/91 mmHg. Her lungs were clear. Laboratory evaluation revealed hemoglobin 8.9, hematocrit 26.7, white blood cells (WBC) 23.3, platelet count 388, serum sodium 131 mmol/L, potassium 7.0 mmol/L, carbon dioxide 9 mmol/L, glucose 87 mg/dl, blood urea nitrogen (BUN) 133 mg/dl, serum creatinine 1.4 mg/dl, calcium 6.6 mg/dl, serum albumin 2.3 g/dl, complement C3 49 mg/dl, complement C4 11 mg/dl, antinuclear antibody (ANA) negative, and antistreptolysin O (ASO) titer undetectable. Urinalysis showed proteinuria, hematuria, and two to five fine granular casts. A renal ultrasound showed increased parenchymal echogenicity, and a chest X-ray showed no acute cardiopulmonary disease. The patient was treated with calcium, Kayexalate®, insulin, glucose, bicarbonate, and albuterol and transferred to another institution. On admission, the infant had generalized edema, with BP 119/78 mmHg. Laboratory evaluation confirmed anemia, azotemia, and hypocomplementemia. The child was placed on furosemide and propranolol, and within 24–48 h, BP was normal and BUN and creatinine levels decreased to 49/0.7. She was discharged 4 days later. Genetic testing for infantile nephrotic syndrome was negative for *NPHS2*, *NPHS1*, *LAMB2*, and *WT1* sequence variants.

One day after discharge, the patient returned to the original ED with dry cough, tachypnea, tachycardia, fever of 103°F, and BP 130/89 mmHg. She had persistent diarrhea but no vomiting. There was nasal congestion and periorbital and pedal edema. Laboratory evaluation revealed anemia, hypocomplementemia, and improved renal function, although proteinuria and hematuria were still present. The infant was rehospitalized. A nasopharyngeal swab specimen was positive for influenza A (by immunoassay), confirmed as 2009 influenza A:H1N1 by real-time polymerase chain reaction (RT-PCR), and negative for respiratory syncytial virus (RSV). By the time of discharge, BUN had decreased to 8 mg/dl and creatinine to 0.6 mg/dl, and the infant had no proteinuria. Laboratory studies obtained 4 weeks later revealed normalization of complement levels.

A presumptive diagnosis of infantile nephrotic syndrome (INS) was initially considered. However, genetic studies were negative for several mutations associated with INS. Although a confirmatory biopsy was not obtained, the clinical and laboratory findings, namely, hematuria, reversible hypocomplementemia, and reversal of AKI and

J. C. Kupferman (✉)
Division of Pediatric Nephrology and Hypertension,
Department of Pediatrics, Maimonides Medical Center,
977 48th Street,
Brooklyn, NY 11219, USA
e-mail: jkupferman@maimonidesmed.org

H. Trachtman
Division of Nephrology, Department of Pediatrics,
Cohen Children's Medical Center of New York,
New Hyde Park, NY, USA

E. D. Spitzer
Department of Pathology, SUNY,
Stony Brook, NY, USA

nephrotic-range proteinuria, are strongly suggestive of AGN. This case occurred during the fall peak of 2009 influenza A:H1N1 activity in New York City.

Recently, Ghiggeri et al. [2] reported two children with influenza A:H1N1 infection who presented with hematuria 48 h before the start of classic pulmonary signs of influenza. These patients did not develop AGN, but it was suggested that macroscopic hematuria may be the first sign of an influenza A:H1N1 infection. Before the influenza A:H1N1 pandemic, direct kidney involvement in children with influenza A virus infection had been rarely reported [3].

In summary, a severe but reversible case of AGN and AKI associated with 2009 influenza A:H1N1 infection was presented. Although a causal effect cannot be confirmed, this case suggests that influenza A:H1N1 should be considered in the differential diagnosis of AGN and AKI in children. It also illustrates the potential value of measuring complement levels in infants presenting with renal disease.

References

1. Jain T, Hemington L, Etuwewe B (2010) A case of post-infectious glomerulonephritis following infection with influenza A subtype H1N1. Pediatr Nephrol. doi:10.1007/s00467-010-1559-1
2. Ghiggeri GM, Losurdo G, Ansaldi F, Canepa A, Magnasco A (2010) Two cases of swine H1N1 influenza presenting with hematuria as prodrome. Pediatr Nephrol 25:779–780
3. Watanabe T, Yoshikawa H, Abe Y, Yamazaki S, Uehara Y, Abe T (2003) Renal involvement in children with influenza A virus infection. Pediatr Nephrol 18:541–544