so far described and not in pediatric patients as per available English literature.

A large number of adverse reactions to IVIG have been reported [4-7], including hypersensitivity reaction like urticaria, angioedema and bronchospasm. Respiratory distress following any infusion can also occur because of circulatory overload or anaphylactic transfusion reactions. Transfusion-associated circulatory overload develops within minutes to hours of transfusion as congestive cardiac failure, cyanosis, hypertension and it rapidly responds to aggressive diuresis and ventilatory support. Anaphylactic transfusion reactions results in laryngeal and bronchial spasm and manifest as tachypnea, wheezing, cyanosis, erythma, edema and severe hypotension, during the transfusion of any type of protein-containing blood component [8].

Akin to post blood component infusion TRALI, that following IVIG infusion could have been due to immune mediated mechanism [9] or neutrophil priming mechanism [2]. In our patient, the possible mechanism could not be ascertained since the estimation of antibody against neutrophils was not available.

It is suggested that clinician using IVIG should closely monitor the patients to pick up this potential fatal complication at the earliest and institute appropriate supportive care timely.

Contributors: VG and TPY were involved in active case management and review of literature. VG and PG revised the paper for critically important intellectual content. All authors approved the final version to be published.

Funding: None. *Competing interest:* None stated.

REFERENCES

1. Rizk A, Gorson KC, Kenney L, Weinstein R. Transfusion-related acute lung injury after the infusion of IVIG. Transfusion. 2001;41:264-8.
2. Kleinman S, Caulfield T, Chan P, Davenport R, McFarland J, McPhedran S, *et al*. Toward an understanding of transfusion-related acute lung injury: statement of a consensus panel. Transfusion. 2004;44:1774-89.
3. Suassuna JHR, da Costa MADL, Faria RA, Melichar AC. Noncardiogenic pulmonary edema triggered by intravenous immunoglobulin in cancer – associated thrombotic thrombocytopenic purpura- hemolytic uremic syndrome. Nephron. 1997;77:368-70.
4. Orbach H, Katz U, Sherer Y, Shoenfeld Y. Intravenous immunoglobulin: adverse effects and safe administration. Clin Rev Allergy Immunol. 2005;29:173-84.
5. Berkovitch M, Dolinski G, Tauber T, Aladjem M, Kaplinsky C. Neutropenia as a complication of intravenous immunoglobulin (IVIG) therapy in children with immune thrombocytopenic purpura: common and non-alarming. Int J Immunopharmacol. 1999;21:411-15.
6. Daphnis E, Stylianou K, Alexandrakis M, Xylouri I, Vardaki E, Stratigis S, *et al.* Acute renal failure, translocational hyponatremia and hyperkalemia following intravenous immunoglobulin therapy. Nephron Clin Pract. 2007;106:c143-8.
7. Shorr AF, Kester KE. Meningitis and hepatitis complicating intravenous immunoglobulin therapy. Ann Pharmacother. 1996;30:1115-6.
8. Silliman CC, Ambruso DR and Boshkov LK. Transfusion – related acute lung injury. Blood. 2005:105:2266-73.
9. Moalic V, Vaillant C, Ferec C . Transfusion related acute lung injury (TRALI): an unrecognised pathology. Pathol Biol. 2005;53:111-5.

Kawasaki Disease in Association with Urinary Tract Infection

ENTESAR H HUSAIN[†‡] AND [#]MARYAM AL-RASHID

From the [†]Department of Pediatrics, Faculty of Medicine; [‡]Department of Pediatrics, Mubarak Al-Kabeer Hospital; [#]Department of Pediatrics, Amiri Hospital; Kuwait.

Correspondence to:
Dr Entesar H Husain,
Department of Pediatrics, Faculty of Medicine, P.O. Box 24923, Safat, Kuwait 13110. entesar@qualitynet.net
Received: March 03, 2010;
Initial review: April 06, 2010;
Accepted: July 26, 2010.

We report a 2-month-old infant with *E. coli* urinary tract infection, who did not respond to antibiotic therapy. She later developed clinical features fulfilling criteria of Kawasaki disease (KD), and was treated with intravenous immunolglobulin and aspirin. KD should be considered in the differential diagnosis in patients who present with infection and do not respond to antibiotic therapy.

Key words: *Kawasaki disease, Urinary tract infection*.

Kawasaki disease (KD) is an acute febrile vasculitis of childhood characterized by the following clinical features; bilateral non-exudative conjunctivitis, erythema of the lips and oral mucosa, changes in the extremities, rash and cervical lymphadenopathy. Sterile pyuria has been seen in 10-50% of patients with KD in the acute phase [1]. Recently, some reports have shown the association of KD with urinary tract infection (UTI) [1,2].

We report an infant with KD with UTI in whom the clinical manifestations of KD evolved later.

CASE REPORT

A previously healthy 2 ½ -month old infant, presented with a 2-day-history of a febrile illness associated with maculopapular rash and nasal congestion. She also had a history of irritability and decreased feeding for one day prior to admission. On examination, she was unwell and irritable. Her temperature was 39.5ºC, heart rate 145/minute and respiratory rate 35 per minute. The cardiovascular, respiratory and abdominal examination was within normal. She had severe nasal block and tearing of the right eye but no conjunctivitis. A generalized maculopapular rash was noted over the face, extremities and trunk. The Initial investigations showed hemoglobin 84 g/L, platelets 388×10^9 /L, and white blood cell count 10.3×10^9 /L (60% neutrophils, 32% lymphocytes, 7% monocytes, 1% eosinophils). The C-reactive protein was 57 mg/L and liver function tests were normal. Urinalysis showed WBC 6-8 cells /HPF and urine nitrite positive; urine culture grew 10^5 CFU/mL of *E.coli*. She received treatment with intravenous cefotaxime (150 mg/kg/day). On the fourth day of admission, the child was still irritable, with decreased oral feeding and febrile at 39ºC; swelling of both hands and feet was noted with accentuated erythema. She had also developed cracked lips with bleeding and bilateral conjunctivitis. The baby was diagnosed to have KD based on the presence of the clinical features and was started on intravenous immunoglobulin 2 g/kg, and aspirin 100 mg/kg/day. The fever subsided after 48 hours and there was a marked improvement of the irritability and feeding pattern. Cefotaxime was continued for treatment of the UTI. Subsequently on the 9th day of the illness, she developed peeling of the skin overlying the tips of her fingers and toes. Echocardiography, done during hospitalization and follow up, was normal. Follow up blood counts on the 10th day of admission showed increasing platelets counts to 722×10^9/L. An ultrasound scan revealed normal kidneys and a voiding cyctourethrogram four weeks after discharge excluded vesicoureteric reflux.

DISCUSSION

KD is a systemic vasculitis, which may be associated with abnormal urinary findings such as sterile pyuria, mild proteinuria and microscopic hematuria. Sterile pyuria has been reported to occur in 10-50% of children during the acute phase [1]. It was well known that pyuria in KD originate from the urethra [3], but a recent study showed that the white blood cells in the urine in patients with KD might originate from the urethra or the kidney or both [4]. In most of the described series, sterile pyuria is found more commonly in infants than in older children [5].

In addition to the current case, there are two cases in the literature of children diagnosed with a documented UTI who subsequently had KD [1,2]. It is unclear if the vasculitis is provoking the infection or the gram negative organisms precipitating UTI are able to produce superantigens similar to staphylococcal and streptococcal antigens that have been suggested to be responsible for the development of KD. From the cases described, UTI seems to be more common in infants with incomplete features of KD. It is difficult to conclude from the limited number of cases if infants with underlying vesicoureteric reflux are more likely to present with UTI when presenting with KD. The likelihood of developing coronary aneurysms increases in children who had UTI due to misdiagnosis and the delay in administering IVIG [2].

In conclusion, it is unclear if UTI is a cause of KD in some infants or this observation is only coincidental. Laboratory evidence of infection in a patient does not always rule out KD. KD should be one of the differential diagnoses in patients who are suspected of having UTI and do not respond to antibiotic therapy.

Contributors: Both authors were involved in all aspects of patient management and manuscript preparation.
Funding: None.
Competing interests: None stated.

REFERENCES

1. Shiono N, Koga Y, Ito H, Egawa K, Ono S, Itami N. Really sterile pyuria with Kawasaki disease. Pediatr Nephrol. 2004;19:124.
2. Wu CY, Hsieh KS, Chiou YH, Wang RS, Huang IF, Lee WY, *et al*. Prolonged fever and pyuria: a urinary tract infection presentation of incomplete Kawasaki disease. Acta Paediatr. 2005;3:375-7.
3. Melish ME, Hicks RM, Larson EJ. Mucocutaneous lymph node syndrome in the United States. Am J Dis Child. 1976;130:599-607.
4. Watanabe T, Abe Y, Sato S, Uehara Y, Ikeno K, Abe T. Sterile pyuria in patients with Kawasaki diseases originates from both the urethra and kidney. Pediatr Nephrol. 2007;7:987-91.
5. Wirojanan J, Sopontammarak S, Vachvanichsanong P. Sterile pyuria in Kawasaki disease. Pediatr Nephrol. 2004;19:363.