Transfusion Related Acute Lung Injury with Intravenous Immunoglobulin

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This case report describes transfusion related acute lung injury with the use of intravenous immunoglobulin in a child with Guillain barre syndrome.

Key words: Acute lung injury, Intravenous immunoglobulin, Transfusion.

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Irtravenous immunoglobulin (IVIG), a pooled plasma derivative, has long been in clinical use and is considered a safe drug. Serious complications of IVIG infusion are extremely rare. We report a case of life threatening pulmonary edema in a two-year old child following IVIG infusion.

CASE REPORT

A 2-year (10 kg) female child was admitted with complaints of progressively increasing weakness of both lower limbs and trunk for last 10 days, and inability to speak and difficulty in feeding for last 5 days. There was no prior medical history of urinary retention, seizures, loss of consciousness, headache, vomiting, rash, loose motion, cough, coryza, paresthesias, hyperaesthesias or drug intake. The child did not suffer from any medical illness or allergy in the past. There was decreased power in lower limbs (3/5), trunk (3/5) and upper limbs(4/5), generalized hypotonia with head lag, hyporeflexia with loss of gag reflex with no pooling of secretions. All other cranial nerves were normal and rest of the systemic examination was also unremarkable. CSF showed albumino-cytological dissociation and nerve conduction velocity revealed demyelinating pattern consistent with Guillain Barré syndrome. Her serum levels of IgM, IgG and IgA were within normal limits. She was started on IVIG at the dose of 400 mg/kg (2g/kg over 5 days) with infusion rate of 0.05 mL/kg/ min.

She was put on nasal oxygen. Her respiratory distress worsened in next few minutes and blood pressure started falling. She was intubated and kept on mechanical ventilation in the intensive care unit. She was given one fluid bolus of normal saline at 20 mL/kg over 1 hour. Chest X-ray done after intubation revealed a normal cardiac silhouette and bilateral interstitial and alveolar infiltrates consistent with pulmonary edema. PaO$_2$ / FiO$_2$ was 237.5mm Hg (95/0.4). CVP was 7 cm. Echocardiography revealed normal left ventricular function with ejection fraction of 60 %. In view of falling BP with CVP of 7 cm, no more fluids were administered and child was started on inotropic support along with mechanical ventilation. She maintained normal acid base balance with normoxia (95% saturation) during the ventilation. After 72 hrs, she was successfully extubated and maintained normoxia on minimal oxygen support (FiO$_2$ 35%). Repeat chest X-ray after 7 days of extubation was normal. She did not receive any further dosages of IVIG. During the next 10 days of hospitalization, her gag reflex and power in the trunk and lower limbs improved.

DISCUSSION

Our patient developed pulmonary edema and hypotension within 2 hours of completion of first dose of IVIG. This life threatening complication of acute lung injury is well known after the administration of blood components but not after IVIG. This usually presents within 1-6 h after transfusion and manifest as clinical syndrome characterized by severe respiratory distress, pulmonary edema, hypoxemia (PaO$_2$/FiO$_2$ <300 mmHg), normal left ventricular function and fever [1]. Our patient fulfills the clinical criteria for transfusion related acute lung injury (TRALI) as described by Kleinman, et al. [2]. TRALI after IVIG infusion has been reported only in two adult patients.
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, erythema, 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.

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REFERENCES


Kawasaki Disease in Association with Urinary Tract Infection

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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 immunoglobulin 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.