CASE REPORT

This 35-year-old man was in good health until 6 days prior to admission, when he experienced malaise, high-grade fever, and symptoms of upper respiratory infection. He developed skin rashes 6 days before admission after contacted with his own child with chickenpox. On arrival, a chest x-ray showed fluffy opacities over both lungs (Fig. 1). He was admitted to our dermatological ward under the impression of varicella pneumonia. After admission, he received acyclovir and Ampicillin/Sulbactam (Unasyn®) administration. Two days later, persistent fever up to 38.6°C, and tachypnea with a respiratory rate of 50 breaths per minute were noted. A bilevel positive airway pressure (BiPAP) ventilator was given. Chest x-ray followed up revealed nodular fluffy opacities over both lungs (Fig. 2). Then he received intravenous Hydrocortisone (Solu-cortef®) 200 mg every 6 hours. The patient’s pulmonary gas exchange had substantial improvement. Five hours later, he was successfully weaned from the ventilator. He received intravenous Hydrocortisone (Solu-cortef®) 200 mg every 6 hours and acyclovir for a course of 2 and 7 days respectively. A chest x-ray followed up 2 days after administration of parental Hydrocortisone (Solu-cortef®) revealed some resolution of fluffy opacities (Fig. 3).

Fig. 1
Chest x-ray film of admission.

Fig. 2
Chest x-ray film prior to intravenous administration of solu-cortef.

Fig. 3
Chest x-ray film 2 days after intravenous administration of solu-cortef.
DISCUSSION

Most reported cases of varicella pneumonia have occurred in cigarette smokers. Smokers are at increased risk because of underlying damage to alveolar macrophages. Varicella pneumonia causes an interstitial pneumonitis with subsequent impairment of pulmonary gas exchange, specifically oxygenation capacity. Pathologic change manifests as a florid immune reaction characterized by an interstitial pneumonitis with mononuclear cell infiltrates, capillary endothelial cell destruction, intra-alveolar exudates and hemorrhage, septal wall infiltration by mononuclear cells, and inflammatory changes in the bronchioles.

Varicella pneumonia is deemed to be due to the host response rather than to specific virally mediated tissue injury. Corticosteroids may potentially modify the inflammatory response when administered early. At a molecular level, corticosteroids inhibit transcription of genes coding for cytokines, resulting in a decrease in the release of macrophage-derived proinflammatory cytokines such as interleukin-1-β and tumor necrosis factor-α. T-cell function is directly inhibited, as is neutrophil adherence to epithelial cells and complement-induced granulocyte aggregation. Production of membrane-derived products such as leukotrienes and prostaglandins by inflammatory cells is also inhibited, with a consequent decreases in edema and vascular permeability.

A literature review for patients with varicella pneumonia that were treated by steroids and acyclovir suggested the following advantages: shorter ICU stay, shorter hospital length of stay, no or low mortality, rapid improvement in oxygenation or radiological improvement. Those patients who received corticosteroids in addition to antiviral therapy and broad-spectrum antibiotics made striking recoveries, particularly in terms of gas exchange. In our case, the initiation of acyclovir therapy was late. The tachypnea of the patient deteriorated gradually despite of acyclovir and antibiotics administration. The cause of tachypnea probably was due to the host response rather than to specific virally mediated tissue injury. After the corticosteroid was given, the patient made amazing recoveries especially in oxygenation, and tachypnea subsided gradually in 5 hours, then the BiPAP was successfully weaned.

We proposed oral acyclovir appears to be a reasonable therapeutic option for those immunocompetent patients presenting within 24 hours of the onset of rash. Intravenous therapy will continue to be the standard therapy for those with pneumonia and immunocompromised hosts. As varicella induced pneumonia may potentially be complicated by chronic sequelae, cases in which the clinical course is highly suggestive of this diagnosis should warrant consideration for steroid therapy in addition to antiviral therapy and appropriate supportive care.

REFERENCES