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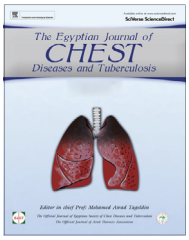

ORIGINAL ARTICLE

Dexamethasone as adjunctive therapy for treatment of varicella pneumonia

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KEYWORDS

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Abstract *Background:* The most common and most serious complication of varicella (chickenpox) in adults is pneumonia, which can lead to severe respiratory failure. Whether addition of corticosteroids to antiviral treatment benefits patients with varicella pneumonia is unclear.

Objectives: To assess the effect of dexamethasone as adjunctive therapy for treatment of varicella pneumonia on the length of hospital stay, which might cause earlier resolution of varicella pneumonia.

Patients and methods: Forty patients were diagnosed as varicella pneumonia and divided into two groups, the first one involved 20 patients who received dexamethasone and acyclovir, and the second one involved also 20 patients but they received placebo and acyclovir. We measured liver function test, kidney profile, complete blood count, blood glucose, C-reactive protein and the levels of interleukin-6 on the day of presentation, after 4 days of admission and on the day of discharge from the hospital.

Result: The mean length of hospital stay in the dexamethasone group was 6.5 days compared with 7.1 days in the placebo group and was significantly different between two groups. The mean time of switching to oral administration of acyclovir was 3.4 days in the dexamethasone group and 4.2 days in the placebo group. The mean time of switching to oral was significantly lower in dexamethasone group than in placebo group.

Conclusion: Adding of dexamethasone to acyclovir in patients with varicella pneumonia can reduce the length of hospital stay.

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Introduction

Varicella (chickenpox) is a common contagious infection caused by varicella-zoster virus with a benign outcome in children. However, pneumonia is the major and most frequent complication of varicella in healthy adults, with incidences

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being reported as high as 50% [1] and carries an overall mortality rate of between 10% and 30% [2].

It has been reported that the susceptibility of varicella pneumonia in healthy adults is 25-fold greater than in children. Mortality rates approach 50% in patients who experience respiratory failure which requires mechanical ventilation, despite appropriate supportive and aggressive therapy [2]. Smoking [1] and pregnancy [3] are well-known as risk factors linked to the development of varicella pneumonia in adults. Patients with chronic lung disease are also at risk of manifesting varicella pneumonia [2].

Varicella pneumonia typically develops within 1–6 days after the onset of rash with symptoms of cough, dyspnea, chest tightness, fever and occasionally with chest pain and hemoptysis. In a limited number of cases, pulmonary symptoms may be present before the appearance of the skin rash [1]. Patients demonstrate impaired gas exchange with progressive hypoxemia. Chest radiographs typically reveal diffuse nodular opacities, progressing to extensive air-space consolidation [4]. The pulmonary lesions caused by acute varicella infection consist of endothelial damage in small blood vessels, with focal hemorrhagic necrosis, mononuclear infiltration of alveolar walls and fibrinous exudates with macrophages in the alveoli, which contain eosinophilic intranuclear inclusions [5].

The main stays of treatment for varicella pneumonia are early diagnosis and initiation of intravenous acyclovir [6]. Adjunctive therapy for varicella pneumonia might help to reduce disease severity. Varicella pneumonia causes an interstitial pneumonitis and this pneumonitis is deemed to be due to the host response rather than to specific virally mediated tissue injury [7]. Corticosteroids may potentially modify the inflammatory response when administered early [8]. Corticosteroids are very potent inhibitors of inflammation [9]. They switch off genes that encode pro-inflammatory cytokines and switch on genes that encode anti-inflammatory cytokines. Treatment with low dose corticosteroids down regulates pro-inflammatory cytokine transcription, which prevents an extended cytokine response and might accelerate the resolution of systemic and pulmonary inflammation in the early phase of varicella pneumonia [10]. Corticosteroids inhibit also the production of membrane-derived products such as leukotrienes and prostaglandins by inflammatory cells, with a consequent decreases in edema and vascular permeability allowing improvement of pulmonary gas exchange [11].

We postulated that adjunctive treatment of varicella pneumonia with intravenous dexamethasone might change the immune response and thereby reduce morbidity and length of stay of patient in hospital. Dexamethasone has potent anti-inflammatory effects and weak mineralocorticoid effects compared with other corticosteroid, thus avoiding interference with sodium re-absorption and water balance. Moreover, dexamethasone has a long lasting effect, allowing for a once-a-day regimen [12].

Aim of the work

We aimed in this study to assess the effect of intravenous dexamethasone compared with placebo on length of hospital stay in non-immunocompromised patients who were admitted to hospital with chickenpox complicated by varicella pneumonia.

Patients and methods

We undertook 40 patients were diagnosed as varicella pneumonia at Infectious Disease Hospital (IDH) which is the only tertiary infectious hospital in Kuwait. Diagnosis of varicella pneumonia was based on the presence of a typical rash of chickenpox associated with development of respiratory symptoms and radiological findings of diffuse interstitial or nodular infiltrates within 10 days following the onset of clinically evident varicella infection.

Serological confirmation was not performed. Hypoxemia was defined as low level of oxygen in the blood ($PaO_2 \rightarrow$ 80 mmhg). Levels of hypoxemia were defined as mild hypoxemia ($PaO_2 \rightarrow$ 60–79 mmhg), moderate hypoxemia ($PaO_2 \rightarrow$ 40–59mmhg), sever hypoxemia ($PaO_2 \rightarrow$ 40 mmhg) [20].

Patients were excluded if they had a known congenital or acquired immunodeficiency or receipt of chemotherapy, any dose of corticosteroid, or immunosuppressive medications in the previous 6 weeks or malignant diseases. Patients who needed immediate admission to the intensive care unit at presentation and pregnant or breastfeeding women were also excluded. Eligible patients who were included in this study provided written informed consent.

The patients were divided into two groups, the first one involved 20 patients who received dexamethasone and acyclovir, and the second one involved also 20 patients but they received placebo and acyclovir. All patients were subjected to history taking and thorough clinical examination. We measured liver function test (LFT), kidney profile (KP), complete blood count (CBC), blood glucose, C-reactive protein (CRP) and levels of the pro-inflammatory cytokine interleukin-6 (IL-6) (Biomedix medical group, Synlab, German) on the day of presentation, after 4 days of admission and on the day of discharge from the hospital. We also measured arterial blood gases (ABG) on the day of presentation and on the day of discharge from the hospital. Some of the patients in both groups needed O_2 therapy in the form of 2–3 L/min of O_2 by nasal prongs and any patients needed mechanical ventilation were excluded from the study.

Patients in the dexamethasone group were given a bolus of 5 mg (1 ml) of dexamethasone intravenously and the patients in the placebo group were given 1 ml of sterile water for injection intravenously once a day at time of admission and for the subsequent 3 days. All patients received acyclovir intravenously in proper dose according to body weight for 7–10 day [13].

Statistical analysis

The data was analyzed using the statistical package for social sciences (spss) version 8.0 software. The significance of differences between mean values of the study variables was evaluated by using *t*-test. The significance of differences between proportions was performed using the Chi-square test. The *P* value less than 0.05 is considered significant.

Results

From September, 2010, to November 2011, we enrolled 40 patients in this study and were divided into two groups, dexamethasone group and placebo group; each of them involved