Basiliximab/steroids/tacrolimus

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Pneumonia with *Scedosporium apiospermum* and *Lomentospora prolificans*: case report

A 13-year-old boy developed pneumonia with *Scedosporium apiospermum* and *Lomentospora prolificans* and died due to respiratory failure during treatment with basiliximab, tacrolimus and unspecified steroids as immunosuppressive therapy. Additionally, he exhibited lack of efficacy while receiving micafungin, itraconazole and voriconazole as antifungal [*dosages and duration of treatments to reactions onsets not stated; not all routes stated*].

The boy, who had pulmonary hypertension due to peripheral pulmonary artery stenosis, underwent bilateral lung transplantation. He was receiving tacrolimus. Twelve months following surgery, he was diagnosed with acute rejection because he had been affected with persistent gastroenteritis. At this time, his blood concentration of tacrolimus trough level was between 3 and 5 ng/mL for a month, which is far lower than the expected target level. He was treated with unspecified steroid pulse therapy and recovered. However, acute rejection recurred, and a second unspecified steroid pulse treatment was given, along with basiliximab. His blood tacrolimus trough was adjusted to an appropriate level of 9-12 ng/mL. Subsequently, he was discharged. At the time of discharge, pulmonary function test demonstrated that his total lung capacity (TLC) of 1740mL, and forced expiratory volume in 1 second (FEV1) of 990mL. Both TLC and FEV1 were much worse compared to the baseline value, which were 2280 and 1320 mL, respectively. After 1 week, he complained of shortness of breath, and was admitted. The FEV1 decreased further to 830mL. Chest CT and laboratory data were unremarkable. He met the diagnostic criteria for restrictive allograft syndrome, which was a phenotype of chronic lung allograft dysfunction (CLAD).

The boy was treated with azithromycin and another unspecified steroid pulse therapy. However, TLC and FEV1 continued to decline. Anti-human leukocyte antigen antibodies against DQ7, 8, 9 were identified in his serum. Considering clinical diagnosis of antibody-mediated rejection, plasmapheresis was performed and IV immune-globulin (IVIG) therapy was administered. On day 1 following the completion of immune-globulin treatment, the b-D-glucan serum level increased to 23 pg/mL. Chest x-ray and chest CT scan showed no significant changes and the sputum culture was also negative for bacteria or fungus. IV micafungin injection was empirically administered for 2 weeks. Afterwards, the b-D-glucan serum level reduced to 10 pg/mL. Micafungin was stopped, and he was discharged on oral itraconazole. After 12 days, he developed shortness of breath, and was again admitted to hospital. A fungal infection was suspected, and micafungin treatment was restarted. His shortness of breath did not improve, he was transferred to other hospital. At this time his b-D-glucan serum level was 5 pg/mL. CT scan revealed ground-glass opacities in the right upper lobe and left lower lobe that deteriorated during the next 2 weeks. A transbronchial lung biopsy (TBLB) and bronchoalveolar lavage (BAL) were performed. Microscopical examination of BAL revealed yeast-like fungi. Therefore, IV voriconazole was started in place of micafungin. Pathological examination showed filamentous fungi and yeast-like fungi with neutrophil and lymphocyte infiltration in the alveolar wall. Eventually, he developed ventricular tachycardia on the basis of hypoxia and required ventilator support. At this point, his culture result of BAL fluid was found positive for *Scedosporium apiospermum* and *Lomentospora prolificans*. Subsequently, he was diagnosed with pneumonia secondary to these infections. Around 17 months after transplantation, he died of respiratory failure by pneumonia with *Scedosporium apiospermum* and *Lomentospora prolificans*, despite the intensive care.

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