# A Case of Complication of Rheumatoid Arthritis and Interstitial Pneumonitis both of which Healed Completely after Repeated Intradermal Injections with a Non-Specific Antigen Preparation

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#### Abstract

**Introduction:** Mutual exchanges between specific and non-specific antibody molecules on receptors on the surface of cytolytic T lymphocytes should take place after an accumulation of non-specific antibodies in the patients' bodies. These exchanges will bring about decompositions of combinations of cytolytic T lymphocytes and specific antibodies. These decompositions equal eliminations of causes of autoimmune diseases.

**Case report:** A 33-year-old man had been suffering from morning stiffness of all hand-fingers for a year and had diagnosis of rheumatoid arthritis and interstitial pneumonitis. He received intradermal injections with 0.1ml of 10 to the 30-fold with saline diluted Neurotropin. U/ml). At the end of the treatment, the patient said that he had nothing to claim.

Conclusion: His complication has been completely cured with a non-conventional treatment.

Keywords: Rheumatoid Arthritis; Interstitial Pneumonitis; Intradermal Injections

## Introduction

According to the traditional concept of the contemporary Immunology, neither autoimmune diseases nor allergic diseases can be cured completely. Nevertheless, a fortunate coincidence led the author to discover a novel concept that eliminations of the causes of these diseases are possible. In other words, combinations of pathogenic antibodies with responsible cells, namely, cytolytic T lymphocytes in cases of autoimmune diseases and mast cells in cases of allergic diseases, can be decomposed by replacing the pathogenic antibodies with non-specific antibodies [1].

#### **Case Report**

**Case 1:** A 33-year-old man (T.K.) visited the author's clinic on February 6, 2013. He said that he had been suffering from morning stiffness of all hand-fingers for a year. He also said that a Clinical Immunologist in Kyoto University Hospital had diagnosed him as rheumatoid arthritis and had prescribed him daily 20 mg of Prednisolon in the beginning of September, 2012. In the author's clinic, he started receiving intradermal injections with 0.1 ml of 10 to the 30-fold with saline diluted Neurotropin; a product of Nippon Pharmaceutical Company (Osaka), consisting of an extract of rabbit skin inflamed by inoculation of Vaccinia virus, on his upper arms at 2~12 day intervals. The Clinical Immunologist reduced the amount of Prednisolon to 18 mg per day in the end of February, 2013. The Immunologist reduced the amount of Prednisolon to 16 mg per day on March 20, 2013.

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He reduced the prescription to 15mg per day on May 7, 2013. The prescription was reduced to 12 mg per day on September 12, 2013. It was reduced to 10mg per day on October 18, 2013. As of November 16, the daily dose became 9 mg. The patient claimed a low degree of dyspnea upon ascending stairways since the summer of 2013. Results of his blood KL-6 measured in Kyoto University Hospital on January 15, 2016, February 26, 2016, April 8, 2016, June 17, 2016, July 22, 2016, October 7, 2016, December 9, 2016, March 17, 2017, June 16, 2017, September 15, 2017, December 15, 2017 were 942 U/mL(Normal Limit < 500 U/mL), 804 U/mL, 739 U/mL, 751 U/mL, 651 U/mL, 564 U/mL, 560 U/mL, 490 U/mL, 526 U/mL, 356 U/mL, 369 U/mL, and 294 U/mL, respectively. As a matter of course, he was diagnosed as complication of interstitial pneumonitis in addition to rheumatoid arthritis. He kept receiving the same intradermal injections through December 21, 2016. On December 24, 2016, the injected preparation was changed to 0.1 ml of 10 to the 33-fold with saline diluted Neurotropin. It was again changed to 0.1 ml of 10 to the 36-fold with saline diluted Neurotropin on December 26, 2016. It was again changed to 0.1 ml of 10 to the 39-fold with saline diluted Neurotropin was further raised as follows: (1) Ten to the 49-fold on March 24, 2017. (2) Ten to the 50-fold on May 24, 2017. His blood rheumatoid factor estimated on December 15, 2017 was < 8.0 IU/ml (Normal limit: 15.0 IU/ml) The total number of injection was 452. On December 16, 2017, the patient told the author that he had nothing to claim.

#### Discussion

The patient completely healed with a non-conventional treatment. It is well established that the etiology of allergic diseases is that combinations of mast cells and allergen-specific antibodies cause allergic symptoms when the patients meet allergens. Similarly, the etiology of auto-immune diseases is that combinations of cytolytic T lymphocytes and organ-specific antibodies cause injury of the organ. A most plain idea would be that break down of the above-mentioned combinations must bring about disappearance of causes of the diseases. To work out the above mentioned concept, it is necessary to have the patients make non-specific antibodies for themselves. In order for the patients to do so, they need to receive intradermal injections with non-specific antigen preparations. Consequently, non-specific antibodies accumulate in the patients' bodies, which may replace specific antibodies from respective cells bringing about elimination of causes of the diseases. Needless to mention, where there is no cause, there is no disease. Details are demonstrated elsewhere [1]. The conceptual basis of antibodies' mutual exchange is existence of equilibrium state among antibody molecules in the vicinity of receptors, which was first proposed by Porter [2]. One of the contemporary trends concerning treatments of allergic diseases is an intravenous infusion of solution of non-specific antibody preparation. However, a large number of these infusions are dangerous because anti-antibody antibody might be produced in the recipient's body, which may cause an anaphylactic reaction.

Intradermal injections with a non-specific antigen preparation induce productions of non-specific antibodies in the body of the patient. Repetitions of the injections bring about an accumulation of them. The basis of increments of antigen dilution is the following: As accumulation of non-specific antibody proceeds, the patient's synthetic capacity of non-specific antibodies decreases.

Consequently, excessively injected non-specific antigen would attract some of the previously produced non-specific antibodies, which are attached to receptors. The attraction mentioned above comes from the affinity between antigen and its specific antibody. The same is true between non-specific antigen and its antibody. Accumulated non-specific antibodies will occupy most of the receptors on the surface of responsible cells. When the accumulation reaches the sufficient level, virtually no pathogenic antibodies would remain on the receptors. That is, no causes of the diseases remain.

#### **Bibliography**

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