Case Report

Microscopic Polyangiitis following Silicone Exposure from Breast Implantation

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We describe a case of a patient who developed microscopic polyangiitis (MPA) in the setting of exposure to silicone after breast implantation. A 57-year-old Hispanic woman was admitted to our hospital with complaints of fever, cough, and hemoptysis. She had undergone silicone breast implantation two years prior to presentation. She was diagnosed as having microscopic polyangiitis (MPA) based on acute progressive renal failure, hematuria, pulmonary hemorrhage, and positivity for myeloperoxidase-anti-neutrophil cytoplasmic antibody (ANCA). A renal biopsy performed showed focal segmental necrotizing and crescentic glomerulonephritis. The patient received high dose steroids, cyclophosphamide, and plasmapheresis with remarkable clinical response. This case report raises the possibility of the development of MPA after silicone exposure from breast implantation.

1. Introduction

There have been a number of reported cases in which autoimmune syndromes have occurred following exposure to various chemicals. In 1964, Miyoshi et al. [1] first coined the term human adjuvant disease in their report of two patients who developed connective tissue-like disease after exposure to silicone-related substances during augmentation mammoplasty. Since this first observation, there have been numerous published cases relating silicone exposure to autoimmune rheumatic diseases. We, herein, report a patient who developed anti-neutrophil cytoplasmic antibody- (ANCA-) associated vasculitis following exposure to silicone from breast implantation.

2. Case Report

A 57-year-old Hispanic woman presented to our institution with fever, cough, and hemoptysis. She had a history of type II diabetes mellitus, hypothyroidism, intermittent asthma, and nephrolithiasis. She also had left breast cancer for which she had a curative left mastectomy and a prophylactic right mastectomy with subsequent bilateral breast implantation with isotonic saline-filled silicone elastomer shell two years prior to presentation. Her physical examination was significant for bibasilar lung crackles. The rest of the examination was unremarkable. Radiologic imaging of her chest revealed left mid lung, basilar, and perihilar opacities. She was initially managed as a case of healthcare associated multifocal pneumonia with broad spectrum intravenous antibiotics. Because of her poor response to therapy and clinical deterioration, a bronchoscopy was pursued which revealed diffuse alveolar hemorrhage. Additional testing revealed microscopic hematuria (RBC: 44 per high-power field, elevated ESR and CRP, and antineutrophil cytoplasmic antibody >100 U/mL (normal: <6 U/mL)) (Table 1). A preliminary diagnosis of microscopic polyangiitis (MPA) was made. A renal biopsy done revealed focal segmental necrotizing and crescentic glomerulonephritis, pauci-immune type (antineutrophil cytoplasmic antibody associated) with moderate activity and minimal chronicity, minimal tubular atrophy, and interstitial fibrosis (Figure 1). Immunofluorescence microscopy was negative for any significant immunoglobulins and complement deposition and no electron-dense deposition was detected by electron microscopy. She was treated with a combination of pulse dose of methylprednisolone, cyclophosphamide, and plasmapheresis with remarkable clinical response.
susceptibility. Indeed, in a recent publication by Tsuchiya et al., an association of HLA-DRB1*0901 with MPA and MPO-ANCA-positive vasculitis in Japanese patients has been reported [11].

The mechanism of silica exposure in the development of small vessel vasculitis is not well understood but several potential mechanisms have been proposed [12, 13]. One theory suggests that silica particles stimulate production of lymphocytes, including T cells and B cells, and that in certain clinical and genetic settings causes autoimmune disease as well as the production of autoantibodies, including ANCA [12]. A second theory suggests that silica particles activate monocytes and macrophages, resulting in the release of IL-1 or tumour necrosis factor-α, oxygen-derived free radicals, and lysosomal enzymes such as PR3 and MPO [13].

To our knowledge, this is the second report of MPA after exposure to silicone from breast implantation. An accumulation of such cases and further studies are necessary to clarify whether exposure to silicone or silicone-containing compounds or implants is related to the development of autoimmune disease.

**Conflict of Interests**

The authors declare that there is no conflict of interests regarding the publication of this paper.

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