Chronic inflammatory conditions are often associated with an expansion of the lymphatic network, or lymphangiogenesis. While typically considered only as altering fluid, solute, and cell transport functions, recent evidence has highlighted new roles for lymphatics in modulating immunity beyond drainage; however, the immunomodulatory roles of lymphangiogenesis in airway inflammation – whose understanding is crucial to developing new molecular targets – remain poorly understood. We have explored the role of lymphangiogenesis in allergic airway inflammation characteristic of asthma by blocking pro-lymphangiogenic signaling using antibodies, and found that lymphangiogenesis has both protective and pathological effects. In acute allergic inflammation, blocking lymphangiogenesis temporarily ameliorates the inflammatory response, decreasing regulatory and innate immune cells in the lungs. In both chronic and acute inflammation, blocking lymphangiogenesis also leads to less inflamed lymph nodes as indicated by the decreased type 2 T cell to regulatory T cell ratio. In striking contrast, blocking lymphangiogenesis enhances the pathogenic allergic memory response, resulting in significantly more type 2 inflammatory cells in the lungs of mice where inflammatory lymphangiogenesis was prevented from occurring. Similarly, transgenic mice lacking pro-lymphangiogenic growth factors displayed an exacerbated memory response to allergens. Our data thus indicate that blocking lymphangiogenesis has contradictory effects – initially decreasing immune cell recruitment to the lungs making them appear less inflamed, but these changes also decrease regulatory T cells, ultimately resulting in an exacerbated memory response. Altogether, our data indicate a new role for lymphangiogenesis in allergic inflammation and suggest that lymphangiogenesis may be a novel target for ameliorating allergic responses.